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A

PRACTICAL TREATISE

ON

MEDICAL DIAGNOSIS

FOR STUDENTS AND PHYSICIANS.

BY

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THIRD EDITION, REVISED AND ENLARGED.

ILLUSTRATED WITH 253 WOODCUTS AND 48 COLORED PLATES

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TO THE

MEMORY OF MY FATHER

BENJAMIN MUSSER, M.D.,

AND

MY GRANDFATHER

MARTIN MUSSER, M.D.

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PREFACE TO THIRD EDITION.

The appearance of a third edition seems to justify the author's convictions expressed in the preface to previous editions concerning methods of diagnosis.

The present issue has been largely rewritten and rearranged. The recent advances in methods of diagnosis which have proved to be reliable have been added.

I am indebted to many kind friends for valuable assistance. Dr. Joseph Sailer rewrote the chapter on Nervous Diseases, arranging it to conform with the body of the book. Dr. William C. Posey wrote the section on Diseases of the Eye. Dr. Thomas S. Kirkbride, Jr., wrote the pages on the Pictoric Records of Physical Signs, and spared neither time nor labor in the preparation of the new plates. Dr. W. S. Smith, of Boston, most kindly revised the section on Sputum, making valuable additions. Dr. Fred. H. Howard rendered valuable assistance and prepared the index. Finally, I am indebted to my secretary, Miss Fannie V. Coe, for valuable assistance rendered while the work was passing through the press.

1927 Chestnut Street, Philadelphia.

PREFACE TO SECOND EDITION.

Success in treatment requires both accuracy and completeness in diagnosis. Partial knowledge of the nature of the case differs merely in degree from ignorance, and treatment based on either invites chances unjust alike to the patient and to the interests of the physician.

Diagnosis, being a practical art, should be held to include not merely the recognition of a disease or a complication of diseases, but also a determination of the *health-value* of the patient. Thus in a case of pneumonia not only should the presence of the malady be established, but the functional condition of all the organs should also be investigated, in order that rational treatment may be prescribed and a rational prognosis given. In other words, the physician should never forget that a patient is a unit, comprising closely interacting organs, and that the response to treatment will be satisfactory in proportion to its adaptation to the condition of the entire organism. After twenty years of experience as a general practitioner, a hospital physician, and later as a consultant, the writer is confirmed in the conviction that success in treatment follows only upon diagnosis of the most comprehensive character, and, furthermore, that the *status præsens* should be clear to the physician, not only at the outset, but also at every stage of the disease.

The first edition of this work was prepared with these ideas of completeness in view, and its early exhaustion is gratifying as an evidence that practitioners and teachers recognize the vital importance of complete diagnosis, and have given their approval to an earnest effort to present a knowledge of it in available form.

This opportunity for revision has been conscientiously utilized, and the new edition will be found to embody the latest approved advances and the newly established facts and methods in this most

active and practical branch of medicine. The series of illustrations, which was already unusually large for a work of this character, has been enriched with many new engravings and colored plates. The author can claim to have been a most critical student of his own book, and likewise to have profited by the criticisms of other teachers and practitioners.

Although there is no "royal road" to diagnosis, either through compends or more or less elaborate catalogues of diseases which aid the memory at the expense of comprehension and judgment, a serious study of the subject is repaid in the acquisition of a most valuable power. Modern research has placed this fundamental branch upon the plane of an exact science, and has correspondingly elevated the whole superstructure of medicine. Instruments and methods of precision, physical, chemical, microscopical, and biological, are now so readily at the command of every practitioner that he is legally as well as morally bound to exhibit in his diagnosis and treatment a degree of certainty far greater than could formerly have been exacted.

In conclusion, it has been the primary purpose of this book to deal with the whole subject of diagnosis in its present state of development in clear language and with abundant illustration, to afford the practitioner a consultant upon which he might rely, and to present the facts and principles in such a manner as to give the undergraduate and postgraduate student a rational grasp and practical working knowledge of this fundamental science and art.

The author takes this opportunity of acknowledging his renewed indebtedness to his friend, Dr. H. B. Allyn, for valuable assistance while the work was going through the press; to Dr. J. Allison Scott for the care and patience he took in supervising the production of most of the drawings and for suggestions in the chapter on Diseases of the Kidney; to Dr. Joseph Sailer, Dr. J. Dutton Steele, and Dr. James Ely Talley for timely suggestions and great aid in verifying references. To Mrs. Philip Putnam Chase the author is under obligations for the skill and patience required in the execution of many of the drawings.

1927 Chestnut Street, Philadelphia,
October, 1896.

PREFACE TO FIRST EDITION.

Modern methods of medical education demand that the student should be taught the expressions of morbid action, or, in other words, the phenomena of disease. He must be brought into contact with them in the hospital-ward and the outpatient-room, which are the medical laboratories where all the data are collected, analyzed, and used in discriminating the various disorders.

The object of this volume is to aid the student in the pursuit of such laboratory-studies, and at the same time to furnish the practitioner with a reliable practical guide to diagnosis for use in his daily work. It has been thought best to combine in these pages the study of the objective phenomena or *signs of disease*, the subjective phenomena or *symptoms*, and the methods employed for their determination. Special attention has been paid to research for objective phenomena appearing in physical, chemical, and biological changes in the tissues and secretions. The necessity for elaborate descriptions or extended lists of minutiæ as guides to differentiation is being rapidly displaced by the use of instruments of precision. Formerly, for instance, extensive tables were displayed to indicate the differential diagnostic features of anæmia and chlorosis; now a few moments' examination of the blood decides the nature of the affection, and whether iron or arsenic is to be given for its cure.

The following pages bear evidence that the author does not undervalue the direct and collateral data obtained by inquiry. Without them an examination carefully conducted according to all other methods may go for naught in the distinction of disease.

The association of morbid processes with their phenomena is a practice of the utmost importance to students, and a chapter has therefore been inserted upon the Symptomatology of Morbid Processes. Bacteriological Diagnosis has become an established method by which

various disorders are recognized, and it is essential that the procedures in this new means of research should be fully outlined. The chapter on this subject is included not merely as a guide and reference for the trained student, but it is hoped that it will also emphasize the possibilities of bacteriological studies, and inspire those who are themselves without facilities for prosecuting laboratory-work to have examinations made for diagnostic purposes by experts with laboratories at their command.

My best thanks are due to my associate in private and hospital work and teaching, Dr. H. B. Allyn, for assistance without which this book could not have been written; to Dr. H. Toulmin for aid in the collaboration of the sections devoted to the examination of Sputum and Feces; to Dr. Charles Burr, of the Infirmary for Nervous Diseases, for the articles on Cerebral and Spinal Localization and on Electrical Diagnosis; and to Drs. Joseph Sailer, W. H. Fenn, and J. E. Talley, for valuable assistance.

Fortieth and Locust Streets, Philadelphia,
February, 1894.

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ERRATA.

Page 203, third line from top, *omit* "disease" after "malingering."

Page 230, twelfth line from bottom, *for* "moist," *read* "most."

Page 268, seventh line from bottom, *for* "in a week," *read* "in about three weeks."

Page 272, fourth line from bottom, *insert* "actinomycosis" after "glanders."

Page 277, fifth line, *omit* the "comma" after "malignant."

Page 282, fourth paragraph, second line, *for* "four hours," *read* "forty-eight hours."

Page 284, third line from top, *for* "roseate" *read* "rosette."

Page 289, third paragraph, fifth line, *insert* "combined" after "ulceration."

Page 299, third line from bottom, *for* "forms are decreased," *read* "forms are relatively increased."

Page 305, fourth paragraph, fifth line, *for* "1 to 5," *read* "1 to 50."

Page 359, thirteenth line from bottom, *for* "intercellularis," *read* "intracellularis."

Page 369, sixth line from bottom, *for* "identity," *read* "entity."

Page 373, fifteenth line from bottom, *for* "value," *read* "volume."

Page 386, third paragraph, first line, *for* "acidity of blood," *read* "alkalinity of blood" in both places.

Page 489, fifth line from top, *insert* "tissue" after "pulmonary."

Page 491, thirteenth line from top, *for* "implies," *read* "applies."

Page 496, fifth line from top, *for* "tympanite" *read* "tympanitic."

Page 611, second paragraph, third line, *for* "distended" *read* "distending."

Page 821, fourth line from top, *for* "tubules" *read* "intestines."

MEDICAL DIAGNOSIS.

PART I.

GENERAL DIAGNOSIS.

CHAPTER I.

GENERAL OBSERVATIONS.

The data upon which a diagnosis is based: The data obtained by *inquiry*. The data obtained by *observation*. Object of diagnosis—Requisites on the part of the student—Methods of diagnosis: Direct. Indirect (by exclusion). Differential—Diagnosis sometimes impossible. Avoid haste—Diagnosis should not be limited—Modern diagnosis—Case records—Scope of the present volume.

The sufferings of one who comes under the care of a physician are indicated by *symptoms* of which the patient himself is cognizant, and for which usually he applies for relief; or by alterations of the physical or chemical structure of the whole or a part of the body, or of the functional activity of organs—alterations which, although not apparent to him, are evident to the observer, the physician. The symptoms of which the patient complains, and of which he alone has knowledge, are known as the *subjective symptoms* of disease. The symptoms which the physician observes, some of which, as the changes of the exterior, may be apparent to the patient, are known as the *objective symptoms* of disease.

The subjective symptoms of disease, as well as such objective symptoms as the patient is aware of, have a history. It may be the brief one of sudden onset, or a long one of rise and fall, of ebb and flow, of the mingling of complex phenomena from time to time. The story of the evolution of the disease is written as the *history of the present disease.*

The present disease may be due to previous attacks of disease, or be modified by the occurrence of previous disease. We may be consulted for the effects of one link in a chain of morbid disorders which began in infancy or early adult life. We should learn, therefore, of the occurrence of *previous disease.* Certain types of constitution and some few diseases are transmitted by parents to offspring; we should, therefore, inquire into the *family history.* A further insight into the nature of the suffering may be obtained by a knowledge of the age, sex, habits,

occupation, environment, etc.—in short, by a knowledge of the *social history*—for, if the cause of the disease under consideration is determined, a distinction from other affections with allied phenomena can frequently be made.

The *subjective symptoms*, the *history of the present disease*, the *previous history*, the *family history*, and the *social history* are learned by *inquiry* of the patient or the friends of the patient by methods and within limitations hereafter to be described. It is proper that they should be ascertained, if practicable, before the objective symptoms are studied.

After the story of the patient is ascertained in full, the *objective symptoms* are sought for. Examination of the patient by the use of the senses of sight, of touch, of hearing, with the instruments of precision to aid them—the physical examination—and by chemical and bacteriological methods, reveals the presence or absence of the latter class of symptoms.

The phenomena of disease are ascertained, therefore, by INQUIRY and by OBSERVATION. The facts or data thus collected and the discriminate interpretation of them constitute *diagnosis*.

Object of Diagnosis. The object of diagnosis is to determine the condition of the living patient who may be suffering from disease. It implies not only that the phenomena of disease are detected, but also that the effects of the disease on the organism are determined, and that the morbid process which is the cause of the phenomena is ascertained. Even this is too restricted an idea of diagnosis. It should include also the recognition of the cause of the morbid process. The latter is known as the *ætiological diagnosis*. In addition to naming the disease and its cause we should include in the diagnosis a determination of the stage of the disease and the recognition of its complications. Moreover, diagnosis implies such knowledge of the patient's condition as to enable an estimation of the dangers liable to arise and of the outcome of the disease—the *prognosis*.

Diagnosis is not made in order to give the disease a name alone but to treat it, and as it is not disease that we treat but a patient with an ailment, full knowledge of the patient and of his environment, his mode of life, habits, occupation, etc., must be obtained by inquiry.

The practical result of diagnosis is the ability to remove or prevent the occurrence of the morbid processes, or to mitigate their effects by rational therapeutics.

Requisites on the Part of the Student. As data are to be collected by inquiry and by observation, it is obvious that he who would inquire and observe intelligently and successfully must be possessed of knowledge and qualifications of a high order. The phenomena of health must be familiar to him. He must have a full knowledge of *physiology*, to recognize the aberrations of function, and of *pathology*, to understand the production of symptoms by disease. He must know the organic results of pathological processes—*morbid anatomy*. He must have learned, by *reading* and *experience*, the significance of symptoms, or of groups of symptoms, and their relation to morbid processes.

He must have a knowledge of the evolution of disease and the phenomena of each period in its development, to secure an accurate account of the disease under consideration. He must know the influence of morbid processes on the body and their effect in the production of subsequent disease, in order to ascertain correctly the various diseases of the patient and infer rightly their relation to the phenomena under consideration. The significance of the family history can be appreciated and correctly applied only by a knowledge of the diseases which are inherited or which arise in certain physical types of individuals, which type is inherited. The social history is not worth securing unless the inquirer knows the influence of age and sex, of race, of occupation, of habits, of residence, of degree of labor, in the development of disease, or the influence of the environment on the individual—the action and reaction of external forces on forces within.

To ascertain the *objective* symptoms, he who would observe properly must know *anatomy*, to recognize the seat of the disease, and *physiology*, to discern the departures from health. He must be trained at the bedside in the use of the senses, and know how to discriminate and interpret phenomena observed by them. He must know how to use instruments of precision, as the microscope, and must learn its revelations. The laws of chemistry and the methods of chemical examination must be familiar to him. Bacteriology and the data obtained from its methods must be appreciated fully.

It is thus seen that the inquirer must have knowledge gained by *reading* and knowledge gained by *observation* at the bedside and in the post-mortem room. He acquires thus, on the one hand, the recorded experience of others, and learns that certain symptoms under certain circumstances indicate a definite malady. On the other hand, he learns that certain symptoms are associated with definite lesions.

Methods of Diagnosis. But we must not only secure facts, we must also be able to utilize them for analysis and induction—the result of which is the formation of the diagnosis. The diagnosis is obtained by three methods—the *direct*, the *indirect*, and the *differential*. By the direct method the data collected are sufficient to warrant a positive conclusion. An indirect diagnosis is made by *exclusion*. A symptom group may represent several diseases. Each affection is passed in review and excluded until one is found to correspond more closely to the data of the case under consideration. It is not one, because of the absence of certain symptoms ; it is not another, because of the presence of certain essentially different symptoms. A negative is thereby proven. By the *differential* method the diagnosis of one of a few possible diseases must be made, the data for and against which are passed in review. The direct method is scientific, rational, and the most practical. It is a process of pure inductive reasoning.

Diagnosis Sometimes Impossible. Notwithstanding our efforts to collect data by inquiry and by observation, we are often unable to make a diagnosis. This arises when premises are wanting for the process of induction. The subjective symptoms may not tally with the

known processes of disease, or the narrator of the history of the present disease may omit important evidence from lack of memory or knowledge, from design, or for other reasons. The objective phenomena may be developed in an ill-defined way, or they may be obscure, as the state of the abdominal contents in a person who is obese; or they may point to one or more processes the subjective symptoms of which are not present. At the time of observation the disease may not have developed fully, may not have "spelled itself out." Under these circumstances a provisional diagnosis must be made or conclusions held in abeyance. If we are considering a contagious disease, for sanitary reasons, the infectious disease should be given the benefit of the doubt. If, on the other hand, the disease requires prompt remedial action, the symptoms must be taken as the indication for therapy.

Avoid Haste. If prompt action is not required, too great haste should be avoided. It is not necessary to make a diagnosis at once, and it is not a confession of ignorance if time is asked before an opinion is given. Repeated observation and reflection should be employed before a conclusion is arrived at. This particularly applies to the class of cases which represent a condition the resultant of improper environment, for the proper detection of which social data, knowledge of temperament, etc., must be acquired. Then, again, it may be necessary to observe the patient under changed circumstances, or study the effects of diet on renal secretion, or on the function of other organs. Haste leads to faulty diagnosis, and therefore to misdirected therapeusis.

Diagnosis Should Not be Limited. It is not sufficient to give a name to a group of symptoms and be satisfied that the diagnosis is made. Every method must be used to collect data. The exact physical condition of the patient must be ascertained and the functional powers of all the organs correctly determined. We thus learn if the more evident disease is the single expression of a morbid process, or if it is the surface storm, the currents of which are underneath. A pleurisy or pneumonia may be the outcome of or complicate a latent nephritis. A peritonitis may be the sequela of an appendicitis or pyosalpinx. Or diseases in two or more organs, due to the same process, may exist at the same time, as suppurative pleuritis and pericarditis. It would not be sufficient to recognize one of the affections alone.

For purposes of treatment it is not sufficient to recognize a neuralgia or a spasm. The state of the patient on account of which the neuralgia developed must be ascertained. Attention must be called to the importance of not being lulled into a false security by the belief that the diagnosis of the first day is sufficient. Complications may arise or the morbid process invade new territory. Thus, in the course of pneumonia, in a few days a meningitis may arise or an ulcerative endocarditis ensue.

Modern Diagnosis. Anyone who takes the trouble to recall the methods of diagnosis that were in use twenty years ago will be struck by the wonderful expansion of the means now at hand to unravel the mysteries of disease. Then a few instruments of precision and a few

chemical reagents were required. The microscope was employed to examine only a few of the excretions and the blood. Now the instruments of precision are multiplied and the scope of their explorations is increased.[1] Chemistry, among other things, helps to fathom the mysteries of gastric disease. The microscope has extended its domain, and, with the new methods of staining fluids and tissues, has become the key that unlocks many of Nature's secrets. The new science of bacteriology has come to our aid, and now instead of waiting to establish a diagnosis until an epidemic counts its victims by hundreds it is obtained at once.

Certainty in diagnosis, for these reasons, has made a decided advance. The number of diseases which can be positively diagnosticated has increased. Methods of investigation and new instruments of precision are increasing daily. May we not hope that in the future the horizon of absolute knowledge will be extended far beyond its present limits? New instruments and new methods will surely avail.

The use of the large number of instruments that are essential, and the chemical and bacteriological examinations that are made, require a great deal of time. Often the diagnosis is a question of hours or even of days. The patient profits thereby. The tax on the physician is far greater than it was a few years ago. The bedside labor is great, and, in addition, he must have a laboratory at his command for microscopical, chemical, and bacteriological work. The outcome is that the scientific physician must have a *clientèle* limited in number, or else have one or more assistants to aid him in his investigations. Without doubt the latter will soon occur. Not as in days of old will we find in the practitioner's office the apprentice, compounding drugs and rolling bandages, assisting in the operations of bleeding and dressing ulcers, but the highly trained, scientific assistant, who by labors in the laboratory and at the bedside is competent to collect data suitable for scientific methods of reasoning.

Case Records. Records of cases should be kept for obvious reasons. The habit compels a general survey of the case, and tends to prevent oversight in the examination. It naturally aids in the training of the powers of observation. It teaches precision in the narration of cases. The memory is aided by repetition and by lack of haste in ascertaining phenomena. The data are on record for more mature reflection, and to aid in the study of the literature of similar cases. The record is of value in case the patient returns for advice after a lapse of time. It may be of medico-legal value. The mental effect on the patient is good, for the taking of notes requires time and accurate studied observation. In case it is desired to study a large number of cases, records are scientific data. The records may be kept on loose sheets and filed for future use. When a sufficient number are secured they may be classified and bound in volumes devoted to the various diseases, or they may be noted in a blank-book. At the end of the year the book

[1] As a most simple illustration, witness the knee-jerk and reflexes, learned by percussion, an old method, in extended use.

is indexed according to the diseases and the names of the patients. A better method is by a system of cards. The cardboard should be six by eight inches. One card is devoted to each case, although more can be used. They are arranged and catalogued according to the library system of card catalogues.

Method of Record. A systematic plan must be pursued in noting the cases. It need not correspond to the lines of inquiry in the examination of the patient, which are modified by the circumstances of the case.

The following outline explains itself. The various data should be recorded in sequence, and in such manner that the facts of each line of investigation can be readily culled for review and analysis. (See Chapters II. and III.).

RECORD OF CASE NO. —.

Diagnosis. *Result.*

Name and residence, place of birth, and former residence.

I. Social history: Age, sex, race, married or single, children, the number and health; miscarriages.

Occupation: Present and previous home surroundings, sanitary conditions, etc.

Habits: Tobacco, alcohol, tea, narcotics; sexual habits; regularity of meals, character of food, and method of eating; number of hours of sleep, degree of fatigue; brain-use, exercise.

II. Family history: Hereditary tendency; health of parents, brothers, sisters, etc. Cause of death and age at which it occurred.

III. History of previous diseases: Character of convalescence from; syphilis and gonorrhœa; injuries.

IV. History of the present disease: Date, mode of onset, and probable exciting cause of present trouble; evolution of the disease to date of examination.

V. The present condition:

A. *Inquiry:* The *subjective symptoms.*

B. *Observation:* The *objective symptoms.*

External appearance, development, color, figure, height and weight, attitude, expression of face.

Temperature, perspiration, eruption, swelling. Condition of limbs and joints.

Examination of the *digestive apparatus:* Mouth, tongue, gums, and pharynx; abdominal organs; contents of stomach, fæces.

Examination of *respiratory apparatus:* Nose, mouth, and larynx. The lungs: inspection, palpation, percussion, auscultation, mensuration. Cough and expectoration.

Examination of *circulatory apparatus:* Inspection and palpation of cardiac area percussion, auscultation of heart; similar examination of arteries and veins; the pulse; examination of the blood.

Examination of the *urinary apparatus:* Kidneys, ureters, and bladder; examination of urine.

Examination of the *nervous system:* Intelligence, subjective nervous phenomena, sleep, gait, station, reflexes, paralysis, tremor, pain, convulsions, headaches, disturbances of sensation, disturbance of speech. The organs of special sense.

Examination of fluids obtained by puncture.

Bacteriological examination of blood, sputum, secretions, exudations, etc.

Diagnosis.

Prognosis.

Treatment.

How to Use the Book for Diagnosis. We must anticipate a little. The student can by ready reference make practical use of the work as the hand-book is used in the laboratory. It is supposed that the case has been thoroughly investigated, according to the directions indicated in the book, and the data arranged in accordance with the case record. An analysis of the data is then made. The value of that obtained by *inquiry* and that obtained by *observation* is carefully considered. The diagnostic significance of the respective data may be found by consulting the index or by a review of the chapters devoted to the special subject. An estimate of the value of the data obtained by inquiry, including the subjective symptoms of disease, will be found in the sections devoted to general diagnosis if the data are general (Chapters II. to XXII. inclusive). If the data obtained by inquiry refer to special organs they will be estimated in the sections on special diagnosis that treat of the manifestations of disease in the respective organs. In the same manner data obtained by observation that are of a general character are considered in the sections on general diagnosis. Data pointing to disease of special organs are considered in the chapters treating of the respective organs.

It must be understood by the student that by general data we mean such as may be expressive of the disease of various internal organs. Thus, the student of internal medicine examines the eye not with the view of finding any special disease of that organ, but to note any changes, physiological or anatomical, which may have resulted from primary disease elsewhere. Diseases of the nervous system, of the blood, of the heart, or of the kidneys may be expressed in eye alteration of some kind. Similarly, the skin, the bones, and joints, as well as other structures, are studied. Many internal diseases will have their outward or physical expression in general anatomic change or in the change of one set of tissues. When this is the case the disease will be discussed when considering its most manifest external expression, as myxœdema under "enlargements" of the body and acromegaly under "bones and joints." The book is arranged, therefore, for diagnostic convenience and not for pathological classification.

CHAPTER II.

THE DATA OBTAINED BY INQUIRY.

The Social History: Age, sex, occupation, habits, residence (past and present), family relations, exposure to contagion. *The Family History:* Parents, grandparents, brothers and sisters of each—brothers and sisters of patient—wife and children. *The History of Previous Diseases. The History of the Present Disease:* Duration. Mode of onset. Evolution of the disease.

Mode of Procedure. First the *subjective symptoms* of the disease are elicited, so that, if necessary, measures may be directed for the patient's relief at once, and that we may have the advantage of observation of the patient's intelligence, expression, etc., and at the same time ascertain the direction further inquiry should take, in order that embarrassment may pass off and composure ensue before an objective examination is made. It seems preferable, however, to begin the *record* with the social history of the case, for a scientific and orderly procession in the data acquired, and then proceed to record the facts of family history, previous history, and history of present disease. Certainly it is immaterial how they are considered in the following discussion, and for convenience, therefore, the above order will be followed. It is to be remembered that the patient's complaints and the objective phenomena—or, if the patient is unconscious or otherwise unable to speak intelligently, the latter alone—are the central threads around which the diagnosis is woven.

The Social History.

The aid to diagnosis obtained from inquiry into the social history cannot be considered exhaustively. Works on hygiene must be consulted. General ideas will be given; reference to the influence of various factors will be found under the individual diseases. That such data are of value is illustrated in various forms of colic. For instance, knowledge that the patient labored in lead will often simplify the diagnosis of the nature of this symptom.

The Age. The age is learned, for each period in the evolution and involution of life has its peculiar physiological processes susceptible to variations by external influences.

A large group of affections arise in the first period of *infancy,* from inheritance or congenital malformations, from accidents incident to parturition, and from improper management of the cord. In a later period, in acquiring adaptability to environment, by the feebly resisting organism, *disturbances of digestion* from poorly prepared or improper food arise; *pulmonary disorders* from improper clothing, ventilation, etc., occur. The developing nervous system has more acute suscepti-

bilities, and hence a long array of reflex symptoms or diseases is observed at this period. Another group of diseases, the *exanthemata*, and almost all contagious diseases, are more prevalent in *childhood*, because they arise out of exposure to a specific cause which usually occurs before the child attains many years. The anatomical arrangement of the larynx, disproportionately small, makes the diseases of that organ most frequent in childhood, and a serious factor in mortality.

At *puberty* we see the perversions (from earlier years) liable to arise as adolescence advances. Anæmia and chlorosis are prone to develop at this period. In the *middle period* the diseases that arise from occupation, from exposure to external agencies, from habits, are seen. Moreover, processes beginning in adolescence are reaching their acme, and find expression in later life, as the cysts of hydatid disease, or renal calculi, or manifestations of gout. In *later life* degenerations of the vascular and cerebro-spinal systems occur; affections due to fibrosis, a resultant of wear and tear, as atheroma; cancer; calculous disease, and other diseases prevail.

The Sex. The prevalence of various diseases in the sexes in undue proportion arises because of difference in the anatomical structure and physiological offices of the two, and because of the difference in exposure to varying causal agencies. Diseases more common to the male sex occur on account of occupation, from exposure, from over-activity of mind and body, and, finally, from the formation of bad habits. The diseases of the female sex that are more prevalent, apart from their own peculiar affections arising out of menstruation and childbearing, take place because of the more or less sedentary nature of their lives, and hence, among other things, the opportunities for introspection. Hysteria, neurasthenia, and nerve disorders abound with them. Males are more subject to epilepsy, gout, diabetes, locomotor ataxy, and vesical disease. Females are more subject to exophthalmic goitre, rheumatoid arthritis, chorea, and the above-mentioned nervous disorders.

Occupation. This must be ascertained in the inquiry, for each occupation demands effort in one particular direction, or compels exposure to deleterious influences. Writer's cramp, eye-strain, and a series of disorders thus arise. Knowledge of exposure to particular irritants, coal or fine particles of metal or stone, gases, chemicals, effluvia of all kinds, and to diseases contracted from animals, is valuable in diagnosis.

The manner and degree of employment of the mind must be inquired into.

It is not to be forgotten that the occupation at different periods of life must be found out, the age at which life's battle began, and the circumstances that surrounded the early career. The deleterious influence of a former occupation may be observed after the patient is in an entirely different sphere of labor.

Habits. Habits as to clothing (catarrhal affections and rheumatism), as to hours of rest and sleep (neurasthenia), as to character of food, time, regularity, and manner of eating (the indigestions, gout), as to

exercise, and as to the use of alcoholic stimulants (cirrhosis of the liver, neuritis, brain affections), of tobacco (amblyopia, cardiac palpitation), of tea or coffee, of narcotics, must be inquired into. Methods of work, methods of recreation, domestic joys or sorrows, must be ascertained. A knowledge of the habits, of the life (of the inner life, indeed) of the individual, is essential to a rational diagnosis, and hence a true therapeusis.

Place of Residence and Dwelling. A knowledge of the place of residence is of service. Town residence and country residence, a residence in a damp locality, by the sea and in the mountains, in particular valleys, in different water-sheds, in tropical or frigid clime, each makes an impress on the constitution, even if actual disease is not created. Hence malarial regions, goitre districts, localities in which individuals have to an unusual degree vesical calculi, or in which special epidemic diseases abound, as yellow fever, cholera, or dysentery, must be inquired for. Knowledge of the places of residence at different periods of life and the duration of such is often important information.

The situation and degree of comfort for habitation of the dwelling must be learned. The sanitary arrangements—drainage, ventilation, water-supply, heating—are to be scrutinized.

Family Relations. Marriage and the number of children, with their degree of health, must be recorded. If a woman, the number of children born, the character of the labors, the number of miscarriages.

Is there trouble in the marital relation? Has there been sorrow, or sudden shock, or long nursing, or great care? Are the financial circumstances easy? Has there been recent malfeasance? How many invalid women arise out of such ashes!

Questions so personal can only be put after long acquaintance, or information obtained through judicious inquiry of friends.

Frequently more delicate questions must be put, as to masturbation or excessive venery, but with great caution, and only when conditions demand it. In epileptiform convulsions, profound hysteria, neurasthenia, the development of locomotor ataxy, or spinal paralysis, prompt, clear, manly questions as to these habits are to be put, not reference made to them in prudish or mawkish suggestion.

Exposure to Contagion. If the suspected ailment partakes of the nature of a contagious disease, the probability of exposure to the disease must be looked into and the presence of epidemics ascertained. The period of incubation must be known in such cases. The prodromal symptoms must also be known.

The Family History.

This inquiry is instituted in order to determine the affections which may or may not be hereditary. We learn also the average duration of life in the family and the relation of the mortality to the physiological epochs in life. Data of the latter character are of value in esti-

mating the possible duration of life for purposes of life insurance, and they also throw light on abnormal conditions; thus to learn that most of the members of the family died of apoplexy at a comparatively early age, or of aneurism or of arterial degenerations, is to learn that vascular changes developed earlier than usual. To secure accurate data, the age and state of health of parents, brothers, and sisters, if living, are ascertained; or, if dead, the cause of death and age at which it took place. Similar questions may be applied to several generations of the family and to collateral branches.

Inherited Diseases. Concerning the question of direct inheritance of disease, but few are strictly so. Of these, nervous diseases are the most common, as progressive muscular atrophy, hereditary chorea, Thomsen's disease, Friedreich's ataxia, migraine, epilepsy, and forms of insanity. The writer has seen chronic Bright's disease, or a state of the constitution that predisposes to it, occur in several generations without the usual exciting causes of that affection. Syphilis may be inherited. Hæmophilia is the most striking affection that is transmitted by inheritance. Generally it is not the diseases themselves that are hereditary but types of tissue that predispose to disease, as in tuberculosis or cancer; or conditions of the organism that favor imperfect metabolism, as is seen in gout or rheumatism.

The family physician, who comes in contact with one or more generations, profits most by the knowledge of the family history. He learns the predisposition to various minor ailments—to headaches and attacks of indigestion, "bilious attacks," for instance; he learns the power of resistance to disease in the family, or their capability to undertake large duties in life; he learns their susceptibility to drugs and their tendency to take stimulants. Nerve force is the capital with which the battle of life is kept up. If it is at a minimum in groups of families, diseases or conditions of poor health due to its use—a use not excessive in others—arise.

Contagious Diseases. In the inquiry it may be well to ascertain the probability of disease being transmitted from husband to wife, or the opposite. Syphilis and gonorrhœa and tuberculosis are examples. Not only may this probability apply to the transmission of disease from husband to wife, but to its transmission along lines of families. Then, too, we must inquire of mothers for the manifestations of syphilis in the children.

Caution must be exercised in the pursuit of knowledge of this kind, as strained, or even ruptured, marital relations may result from injudicious intimations.

Malignant Disease. Caution must be employed in order not to arouse family pride if evidence of "scrofula" is sought for, or to provoke undue alarm when inquiry into the family history of cancer is made. Disarm suspicion by inquiring for the symptoms of the disease in various organs in which it may occur, as jaundice, uterine hemorrhage, etc., or ask about growths or tumors. Do not use the specific terms, consumption and cancer.

Obscure Terms. Moreover, care must be exercised to secure definite data, not to over-estimate statements as to the cause of death being "dropsy," or "jaundice," or "cold," or "teething," or "change of life." Control questions must be put by inquiry into the character of the symptoms that attended the fatal illness, and by giving the affections the various popular names that are given them in different countries.

Common Morbid Processes. The data of the family history are of no avail unless it is remembered that many fundamental affections have various modes of expression. Various diseases may be allied to the one suspected to exist in the patient, and be overlooked because of this difference of expression. One member of the family may die of heart disease, another of rheumatism, or some have had chorea, or cutaneous affections, or renal calculi; such ailments are expressions of the same morbid process. Finlayson well puts them into groups and fittingly portrays them as follows: "In regard to scrofulous [tuberculous] diseases, we ask for swollen glands or 'waxy kernels,' or running in the neck, diseases of the spine and other bones, bad joints, white swellings, or 'incomes,' as they are termed in Scotland; disease of the glands, of the bowels, water in the head, consumption of the lungs, or decline, or weakness of the chest, with spitting of blood, and so on.

"Heart disease, rheumatism, chorea, psoriasis, and some other cutaneous affections, and perhaps renal concretions and emphysematous bronchitis, appear to replace each other in different members of the same family.

"The neurotic group includes the various forms of neuralgia, epilepsy, hypochondriasis, hysteria, and insanity; apoplexy and hemiplegia may (perhaps doubtfully) be included in this group; their hereditary character seems rather to be associated with vascular disorders. Gout, disease of the liver, contracted kidney, renal calculus and gravel, and angina pectoris form another allied group; and these have also some affinity with the disorders connected with arterial degenerations. Syphilis, which, of course, has marked hereditary characters, assumes such a multitude of forms as to preclude enumeration; but the tendency is for such syphilitic diseases to fail in the course of time from early death or sterility. Abortions, stillbirths, early deaths in infancy, associated with cutaneous eruptions on the buttocks and with snuffles, are important in many family histories; nervous deafness, opacities of the cornea, notched teeth, epilepsy, and imbecility are occasional manifestations of the same disorder in those children who survive."

Conclusions. It is thus seen that in securing the family history data are acquired which may be (1) complete and of value in estimating family tendencies or (2) vague and of doubtful value. The latter is due to lack of memory on the patient's part or to his ignorance of techincal terms. The difficulties must be overcome by control questions prompted by our knowledge of the nature of the disease and its frequency at different ages, by an inquiry for symptoms, and by investigation into collateral and remote branches of the family.

The fact that diseases skip a generation (atavism) must be remembered. A generation may be small or decimated by accidental disease, and hence the force of the family history be weakened. At times in a family sufficient time has not elapsed for predisposition to arise, as when we inquire into the illness of a child whose parents are in early adult life. Finally, all negative facts must be recorded. Such knowledge must act as a control element in estimating the value of the family history.

The History of Previous Diseases.

The remote effects of disease, and of its sequelæ, as impressed on the organism, make it essential to inquire into the nature of the previous diseases of the patient whom we are studying. The date and character of the disease, the duration, the degree of severity, and the completeness of convalescence must be determined.

Many diseases, as the exanthemata, usually occur but once in the same person, and, therefore, in the diagnosis of obscure cases, if a history of their occurrence has been ascertained, they can be excluded in the count. Others recur from time to time, as croupous pneumonia, chorea, acute rheumatism, and tonsillitis. The history of a previous attack of a certain disease may point to the nature of a second attack which otherwise may be obscure. Some diseases, as rheumatism, syphilis, and gonorrhœa, have pronounced sequelæ. Knowledge of the occurrence of the primary disease may solve doubts as to the nature of the sequelæ.

Infectious diseases lead to forms of neuritis and to brain affections, or to inflammations of organs. The seat of the specific inflammatory process varies in different diseases. After measles we find the mucous membranes impressionable; and after scarlet fever, the serous membranes, the ears, the kidneys liable to inflammation. The history of an attack of hepatic or renal colic may point to the diagnosis of an otherwise obscure process in the liver or kidney.

The history of injury must be sought for in brain and spinal affections. The occurrence of a surgical operation in the past may point to lesions for which it was resorted to, which again may be the source of disease.

The History of the Present Disease.

Scope of Inquiry. The history of the present disease includes an account of the sufferings of the patient, which I have said are the *subjective symptoms*, of the *duration* of the disease, of its mode of *onset*, and of the *evolution* of its symptoms up to the time it was seen by the physician. The patient also gives an account of such objective symptoms as could be noted by him, as swelling of the legs, the date of its commencement, mode of onset, and progress. In the case record the history to the date of examination is first recorded, and then the subjective symptoms are noted. The same order will be followed in the text. Practically, it is better to learn the symptoms on account of which the patient applied for treatment, and, with them as a guide, to inquire into the date of origin and mode of development of the disease.

Method of Inquiry. The history and subjective symptoms are best learned in the language of the patient. If the memory fails or the symptoms are not clearly narrated, judicious questions will suffice to complete the story. Leading questions must not be put until the patient's own account is fully given.

Often the patient will be too voluble and introduce irrelevant matter, or too taciturn from modesty or a desire to conceal facts, as when illegitimately pregnant. While much time is lost in listening to a prolix account of sufferings, the student will do well at first to bear with the patient, for it gives him the opportunity to study character, observe the mental and emotional characteristics of the patient and the expression of the countenance. To suppress the loquacious, free the tongue of the silent, gather scintillations of intelligence out of the dense clouds of ignorance, requires knowledge of human nature of a high degree, acquired only by long practice. (Allied difficulties have been discussed in the paragraphs devoted to the family history.) Indeed, the wonderful faculty of seeking information in this manner has been the capital of many physicians of large practice. It is by this means and by tricks that the charlatan plies his vocation. A favorite method of the quack, after a few words from the patient, is to tell him how he—the patient—feels. They have some knowledge of the march of the disease, and portray its full development to the surprised and credulous victim. Elsewhere (see Subjective Symptoms) the reliability of such data is discussed, and the student must not for one moment consider the data obtained by inquiry as of equal value with those obtained by observation—the former is the mere skeleton of the diagnosis.

It is particularly important to secure a chronological order of events in the disease. They are essential and logical and throw much light on the progress of the affection. The diagnosis is much easier if such sequence is followed. Of course, there are circumstances when only the minimum amount of information of this character can be secured. The patient may be unconscious, or in a convulsion, or unable to speak from dyspnœa. It then becomes necessary to rely on the testimony of friends or to gather the information from the circumstances that surround the patient.

Mode of Onset and Duration of the Disease. It is well to learn if the onset of the disease was *sudden* or *gradual*. If the former, the most striking phenomena are to be ascertained—a chill, convulsion, sudden pain, sudden vomiting, a profuse diarrhœa—each points to lines of further inquiry. If the latter, did it follow upon an acute illness, or did each symptom gradually increase in intensity, and as each week or each month passed by new phenomena creep into the symptom complex. We thus learn if the affection under consideration is acute or chronic—its *duration*. It must not be forgotten that certain affections may be two or three days—or, on the other hand, as many weeks—in developing, as typhoid fever, which, nevertheless, is acute. It must be remembered, also, that diseases may have sudden acute expressions, and that a chronic disease may be in existence a long time without the patient's knowledge. An acute colliquative diarrhœa or a convulsion

is often the first intimation of a chronic nephritis, and an attack of angina pectoris the first symptom of organic heart disease of long standing. To appreciate the relationship of acute to chronic disease, or of acute phenomena to chronic morbid processes, requires a full knowledge of the processes of disease.

Evolution of the Disease. In making inquiry concerning the evolution of the subjective symptoms, the *frequency, duration, character,* and *degree of severity* of each symptom, and its relationship to the function of the organ apparently affected, must be inquired into. Thus in the case of pain in the abdomen, we must learn its character, its frequency, its duration, its intensity, and its location, and whether associated with functional disturbance of any of the viscera in which the pain presumably has its origin. Or, if there is frequency of micturition, the length of time the symptom has been present, the degree of frequency, the time in the twenty-four hours when the micturition is most frequent; its relation to food, exercise, or emotions; the character of the act of micturition, and its association with other evidences of functional disorder or organic disease of the genito-urinary tract.

Having ascertained the full story of the patient, including all data obtained by inquiry, special attention must be paid to the sufferings or complaints of the moment. They must be further inquired into in the manner above indicated. They may have been detailed in the beginning; but information obtained from an account of the evolution of the disease or the previous history will require a repetition, with the putting of fresh questions or control questions. Having obtained the chronological account of the factors of life and of disease, we are prepared to examine into the significance of subjective symptoms.

The steps thus far taken in the diagnosis are four in number. While considerable that is not essential may be gathered, the very gleaning of the facts enables the student to acquire objective information from the speech, the gesture, the expression, etc., of the highest value. Moreover, the facts ascertained are of value in determining a line of treatment to be pursued which will be scientific and rational, for in addition to the diagnosis the causal factors of the disease are often found.

To repeat, preceding the fifth and final step in the diagnosis, the data secured by inquiry—(1) the social history, (2) the family history, (3) the history of previous diseases, (4) the history of the present disease—must be fixed in the mind. Marshalling the facts thus obtained in orderly procession, we are enabled to systematically add the facts of the *present condition.* Consideration of the data thus secured leads, by inductive reasoning, to the desired conclusion—*a diagnosis.*

CHAPTER III.

THE DATA OBTAINED BY INQUIRY—(*Continued*).

The Present Condition: The subjective symptoms—Mode of determination—Their fallacy—Their value. *Feigned disease.* Local subjective symptoms—General subjective symptoms.

We now come to the final step in the investigation—the determination of the present condition, the *status præsens*. To determine the present condition *inquiry* and *observation* are necessary. This chapter and the succeeding one will discuss only the data obtained by *inquiry*. They, therefore, include the *subjective* symptoms other than those that pertain to special organs or systems. Caution, circumspection, adroitness, combined with tact and good judgment, are more essential to secure a true account of the patient's sufferings even than to obtain a correct history of the disease.

The *subjective symptoms* are expressive of the sensations of the patient, and vary in accordance with the sensibilities of the individual affected. Thus acute pain may apparently represent a severe process in one, while in another the same severity of process may be represented by the minimum amount of pain. It is well known that individuals of one nationality bear pain with greater fortitude than individuals of another.

Caution. *The patient.* Individuals vary not only as to pain sense but as to other subjective symptoms. The *morale* is shattered in some more readily than in others—thus, for instance, oppression of the præcordia may strike terror to some, while to others it would be simply a sense of discomfort. Moreover, subjective symptoms are constantly before the patient while in distress, if only in the mind's eye. Because of this perturbed state they grow in magnitude rather than diminish. We must study them from many points of view. The mode of onset, frequency, degree, and character of the symptoms must be inquired into. The competency of the witness under the circumstances, from lack of accurate noting of symptoms, failure of memory, varying degree of susceptibility to impressions, etc., may well be doubted.

The physician. But not only does the varying "personal equation" of the patient render subjective symptoms fallacious, the same factor in the physician contributes to the fallacy. The latter may have unfortunately formed, by hearsay regarding the patient, a preconceived notion of the nature of the disease; or from personal bias in favor of particular diseases, on account of narrow lines of study or lack of breadth of view of pathological processes, he may set out to prove a theory rather than to establish a fact. In either case, by leading questions, by placing emphasis on certain parts of the testimony, the subjective symptoms can be juggled with and made to tell any but the truthful story.

It is to be remembered that it is our province not only to ascertain the cause of suffering in the sick, but also to detect the flaws in the testimony of him who would feign sickness. The *malingerer* utilizes subjective symptoms to hide his deception because they cannot be seen, felt, weighed, measured, or ascertained by hearing.

Feigned Disease. To detect feigned sickness demands much acumen on the part of the physician. He must not only be able to make an accurate and exhaustive objective examination of the patient, but be alert to appreciate surroundings and conditions. Feigning may be suspected if there is a motive, as in the case of prisoners, pension applicants, students at school or college, and persons who hold policies of insurance indemnifying in case of sickness. The hospital "beat" thus plays upon charity.

If sickness recurs frequently without definite cause, while the subjective symptoms are mild and quickly relieved and the objective symptoms negative, the use of instruments of precision will detect the malingerer. With their aid we can usually find out if the subjective and objective phenomena tally. The failure of such tally proves the deception. The thermometer frequently exposes the deception, as fever can rarely be simulated, although tricks with the thermometer may be carried on. A favorite method is to rub it, and thus cause the mercury to rise. Frequently the suspected person must be placed under close surveillance, unknown to him, and tricks of all sorts, suggested by the surroundings and circumstances, played upon him to make him unwittingly testify to his deception.

The student will learn later that there is a mimicry of disease, and that in certain nervous affections the simulation of subjective symptoms is its chief *rôle*. In hysteria, subjective and objective symptoms are masked. Long experience and acumen are required by the physician to unmask the deceptions. The age of the patient, the sex, the state of the emotions, the varying expressions of the symptoms (under varying circumstances)—with attention fixed or removed—the mobility of the symptoms under excitement or emotional disturbance, the lack of harmony between functional disorder and organic change, are the elements to be considered in order to fathom the mysteries. Often anæsthesia must be induced in order to dissipate simulated tumors, relax rigid joints or contracted limbs. Magnetism, electricity, and other tests are likewise employed. In the chapter on Hysteria its manifold expressions will be adverted to, and it will be seen that functional disorder of almost every organ or special sense is simulated in this affection. Organic processes even are imitated, as joint inflammations, peritonitis, and other grave conditions.

Value of Subjective Symptoms. Notwithstanding the fallacy of subjective symptoms in that they may be feigned or mimicked, they are valuable evidences in the hands of the scientific inquirer. If the patient is a good witness their value is much enhanced. He must be intelligent and truthful. His testimony is of value if he can array in logical order the sequence of symptomatic events which culminated in

the condition for which he seeks relief. If he can clearly narrate the events in his past life, or in the lives of his ancestors, which appertain to physiological aberrations, his story is an aid to the searcher for truth.

If, with this, the doctor is possessed with a scientific turn of mind, considering evidence without allowing previous conceptions to influence him, capable of discerning the truth and discarding the false, of analyzing and weighing statements, and of appreciating their relationship to what is known of morbid processes, the patient's statements of subjective symptoms are of value in the discernment of disease.

Local Subjective Symptoms. The symptoms of which the patient complains may be general or local. The former will be briefly considered in this section ; the latter will be discussed in the respective sections devoted to disease of the various organs to which the subjective symptoms refer. They are symptoms due to functional disturbances of the respective system that is the seat of disease, as dyspnœa or cough in diseases of the respiratory system, anorexia or nausea in diseases of the digestive system. An exception will be made in the case of *pain*. While there may be such general suffering as to constitute pain (general soreness, aching, rhachialgia), yet the symptom has its point of origin most frequently in some local disorder. Notwithstanding this fact, however, as it is a symptom common to so many affections, and as general rules apply to the recognition of its multitudinous forms, a brief section will be devoted to its study.

General Subjective Symptoms. The general subjective symptoms —that is, the abnormal and disagreeable sensations which extend more or less over the whole body, or are referable to more than one organ or apparatus—are few in number and are not diagnostic of any particular affection. They are at times the only symptoms complained of by the patient, and require investigation. They include abnormal sensations of strength or weakness, general numbness or tingling, and general paræsthesiæ of all kinds ; general vasomotor disturbance, causing sensations of heat, such as occur in flashes, or sensations of cold, from mild chilliness or " creeps " to the pronounced chill or rigor, sudden perspirations, general throbbings or pulsations, and general discomfort, to which the term *nervousness* is applied. Irritability, disorders of sleep, and the more distinct nervous manifestations above mentioned, will be referred to in sections on nervous diseases, and particularly discussed under Hysteria and Neurasthenia.

A feeling of *strength*, or the idea of an ability to perform great feats of strength or endurance, or a great mental feat, is a subjective symptom that is dwelt upon by the patient who is developing or passing through certain stages of paretic dementia. It is accompanied by other evidences of exhilaration. *Exhilaration* attends chlorosis and forms of hysteria and neurasthenia, the physical or mental exhibition of strength taking place in the after part of the day and evening or upon undue excitement. Corresponding depression usually follows.

A sense of *weakness*, or *exhaustion*, or of *fatigue* is often complained of. If an absolute demand is made upon the bodily strength it can

respond, but otherwise it is not exerted. The patient complains of being more tired in the morning than upon retiring, or of a sense of inability to perform accustomed or special duties. Mental depression usually attends the phenomena. It is due to neurasthenia generally, but is a frequent accompaniment of and dependent upon the forms of toxæmia to which malaria, gout, and rheumatism belong; of the toxæmia of certain varieties of indigestion, of tobacco, alcohol, and other narcotic poisons (tea or coffee), and of mineral poisons. The same sense of fatigue attends the prodromal stage of the specific fevers. It has been a symptom observed frequently of late in the sequential period of influenza.

The sensation of weakness must not be confounded with true weakness or *muscular prostration*. While the patient is aware of its presence, it is well to consider it under the objective phenomena of disease, for it is a readily recognized sign of disease.

Numbness, or *tingling*, or *burnings* may be general or local. It is a common form of *paræsthesia*, to be discussed in the section on nervous diseases. It must be remembered that, while a disorder of sensation, it is due to morbid conditions outside the pale of the nervous system. It may be of reflex origin, from irritation at a distant point, or it may be and usually is due to toxæmia, as lithæmia. Other subjective vasomotor disturbances that are of frequent occurrence are likewise manifestations of nerve disorder from reflex or toxic causes. Flushings, and a constant sensation of *heat*, with or without *perspiration*, which attend the perturbation of the menopause, are common in uterine disorders and in chronic gastritis.

The student will learn that the curious manifestations to which reference has been made are all evidences of ill health, of a depressed vitality, of a condition in which there is malnutrition, poverty of nerve-force, and lack of blood-richness (anæmia). There may be peripheral irritation or a toxæmia, but the under-current of ill health is the fundamental derangement.

Chill and *fever*. Both are subjective as well as objective phenomena, but as one can be accurately estimated by an instrument of precision (thermometer), and as both are generally associated, the discussion of them will be postponed. (See Objective Signs.)

The abnormal sensation of cold or of heat will be discussed in the chapter on Nervous Diseases.

CHAPTER IV.

THE DATA OBTAINED BY INQUIRY—(*Continued*).

PAIN.[1]

Definition. Pain is a general term used in medicine to describe a number of subjective symptoms connected with morbid processes. It may be defined as a sensation which produces on the part of the organism, as a whole, the desire to abolish or escape from it. It is the expression in consciousness of injury to the peripheral or central nervous system by irritation or lesion; at times the central end of the peripheral nerves may be the seat of irritation, causing so-called *referred pains*. This definition, however, fails to include the hyperæsthesias, the hyperalgesias, and all simulated pains. But the latter are to be included in this section, on the ground of clinical convenience, whilst the two former are only of significance as conducing to the production of pain.

Pathology. The pathology of pain is generally believed to be a state of impaired nutrition, and hence of injury, gross or microscopic, either at the periphery or in the afferent nerve tract. The cause may be purely functional, as, for example, when pain is due to the overstimulation of the tract by its normal stimulus and its consequent exhaustion; or to strictly local conditions, as pressure, injury, or inflammation; or to systemic conditions acting locally, as the neuralgias of anæmia. There is also the so-called sympathetic or reflex pain, due to irritation in a part removed from the locality to which the sensation is referred.

Pains in reference to the general nervous system may be classified according to the localization of the lesion into (1) peripheral, (2) central, and (3) general. *Peripheral* pains are those due to some alteration either in the structure or nutrition of the peripheral nerves, and the disturbance may be situated at the sensory terminations, or anywhere in the course of the nerve or in the nerve-roots. Pains due to causes situated in the latter place are usually perceived at the peripheral distribution of the nerve, and are, therefore, spoken of as *referred pains*.

[1] Pain is treated in a suggestive manner, and so much space is given to it because it is too frequently improperly managed. Its cause is never thoroughly investigated. Anodynes are given for its relief, thus too frequently creating victims of the morphine-chloral-, or other habit The following articles are suggestive: Head: On Disturbances of Sensation, with Especial Reference to the Pain of Visceral Disease, Brain, vol. xvi., Part I., 1893; Ross: Brain, 1888: Mackenzie: Medical Chronicle, 1888; Mackenzie: Points Bearing on the Association of Sensory Disorders and Visceral Disease, Brain, vol. xvi., Part III., 1893. Also, papers by Starr. See Section on Nervous Disorders.

The nature of *central* pain is not at present clearly understood. Certain cases have been reported in which pain has been perceived in one part of the body, usually an extremity, and at post-mortem no lesion whatever could be found in any portion of the afferent nervous system coming from this region. Lesions, however, have been found in these cases in the brain itself, and it is supposed that these are responsible for the painful impression. *General* pains are those due to some toxic condition of the blood, or impairment of the nutrition of the nervous system as a whole, and manifested as pain in the regions of least resistance.

Cause. Conditions acting upon the *peripheries* of the sensory nerves are injuries or disease of the surfaces or of the viscera. Conditions acting upon the nerve in its course may be either internal or external. Among the internal causes are the chronic and acute forms of neuritis. Among the external causes are tumors, perineural inflammatory processes, or anything causing mechanical injury to the nerve itself. Nerve roots are usually involved in intraspinal growths, in spinal meningitis, and, occasionally, as a result of disease of the vertebral column. The lesions causing the *central* pain are embolism, hemorrhage, softening, inflammatory processes, tumors, and injuries. General causes are the anæmias, the intoxications, the infectious fevers, and perhaps certain drug habits, as morphia; although it is usual to include the pains complained of by opium eaters among those due to simulation.

Variations in Disease. Pain is, perhaps, the most variable symptom in disease. It ranges from a sensation of mere discomfort, as the dull ache of chronic lumbago, to the stabbing pain of pleurisy, or the intolerable anguish of heart-pang. It is at times compatible with the highest mental endeavor or the severest physical exertion, or the whole energy of the organism is absorbed in resisting it. It may be definitely localized in any part of the body, in any of the tissues, or distributed over an ill-defined area.

The Recognition of Pain.

The Mode of Expression. As a rule, the physician learns of its existence by the complaint of the patient. Thus he learns more or less accurately its location, character, degree, and duration, and usually something concerning its causation. But the value of this source of information is variable. The patient may be voluble, and describe too much; or taciturn, and shrink from admitting his suffering; or ignorant, and unable to give a clear account. Fortunately, there are other ways by which suffering is expressed which may be grouped among the *objective symptoms.* They are: (*a*) *Facial expression,* the most common interpreter of the emotion, is far more reliable. The tense and drawn lineaments, the clinched jaws, the dilated pupils, the livid countenance, make a picture of agony which, with the labored respiration, the general shrinkage of the body, are unmistakable. (See Chapter VII., The Face.) Or, in a less intense form, the shrieks and struggles or the groans of more prolonged suffering are no less impressive in their suggestiveness. (*b*) Not less characteristic are the various *postures* assumed; the sudden fixity of heart-pang; the retracted head of meningitis; the immobile side

of pleurisy; the crouching attitude or restlessness of colic; the flexed thighs and immobile trunk of peritonitis; the shoulder drooping to the affected side in renal colic; or the bent knee of arthritis. (*c*) Further, there are certain *reflex actions* that are associated with local irritations; thus the closing of the eyelid on irritation of the conjunctiva; the sneeze or cough on irritation of the nasal or laryngeal mucous membrane; the erection following irritation of the urethra; or even the limp characteristic of pain on moving or resting the weight of the body on an affected limb. Then there is the sudden shrinking of the whole body, the attempt to defend, or the sudden movement of the hand to the affected part, or the sudden jerking away of the part itself if the act be possible; these are true reflexes, and sufficiently diagnostic of local suffering. It scarcely need be mentioned that in children, in the insane, in persons unable for many reasons to communicate their thoughts, the expression of pain is of the greatest diagnostic value in determining its seat. (*d*) The phenomena of the associate morbid processes may serve to indicate the occurrence of pain and its seat. Thus pain is one of the cardinal symptoms of inflammation; it is commonly associated with nerve-injury; it is frequently accompanied by local flushing or herpetic eruptions in neuralgia.

Sources of Error. In estimating the presence or absence of pain, or its degree, certain control conditions must be borne in mind. Unfortunately *pain* is one of the most *unreliable* of symptoms. It is necessarily a subjective symptom, with, in all probability, qualitative as well as quantitative variations. The particular degree in either respect is of importance in diagnosis, and as only the roughest means, if any, are available to estimate it objectively, the physician is compelled to rely almost wholly upon the statements and appearance of the patient. His statement can err in two directions: the patient can *exaggerate* his sufferings or *depreciate* them. The tendency to exaggeration is most marked in the nervous temperament; in those suffering from chronic disease of long standing; in those accustomed to in-door and mental labor; in women and in the young. The tendency to depreciation is most marked in the phlegmatic temperament; in those accustomed to hardship, especially if of small intellectual development; in men and in the aged. Both tendencies are to be corrected as nearly as possible by observing the associated symptoms and the character of the patient, and by skilful questioning. The appearance can deceive because of undue susceptibility to suffering on the part of the patient, or unusual inhibitory power. There can be no question that painful stimuli, usually easily borne, in some produce almost unbearable misery. Such exaggerated sensibility occurs in the emotional, in the weak and debilitated, and in the delicately nurtured. *Mental association* is a powerful factor; it is well known that soldiers, who in the heat of battle disregard serious and necessarily painful wounds, will suffer intensely under the probably less painful offices of the surgeon; and it is unfortunately a common experience that the surroundings of the operating-room make the most trifling and briefest operations full of serious suffering. *Habitual* use of *opium* seems to increase this susceptibility in a remarkable manner. Patients will even submit to operations for the relief of

a supposed ailment that is found to have no physical basis ; and this occurs in cases in which there is no reason to believe that the pain is simulated as an excuse for the indulgence. Moreover, a pseudo-neuralgia is wont to occur in victims of the morphine habit. It may simulate a gastralgia or an intestinal colic. The writer has seen an innocent victim of morphine suffer from pseudo-hepatic colic, withdrawal of the drug causing subsidence of the periodical attacks of pain and vomiting. *Inhibition* is a much more serious source of error, for while undue attention to one part is only reprehensible when practised to the neglect of others, a patient who disregards pain may fail to direct attention to the real seat of disease. It is sometimes exercised to a most remarkable degree. The stoicism of the American Indian under torture is attested by many observers ; certain religious sects among the Hindus habitually afflict themselves in the most ingenious ways ; the early Christian martyrs rejoiced in misery. It is common to find this disregard of pain among those exposed by occupation to discomforts and injuries, and the Teutonic and Slavic races appear to possess it in a higher degree than the Celtic or Semitic. *Shock* either inhibits pain or diminishes the normal response to it. Lastly, and by no means to be neglected, a most common source of error is undue credulity or skepticism on the part of the physician, for he may be deceived by an eloquent and persuasive complaint, or discredit true suffering.

Simulated Pain (see Feigned Disease) is to be recognized by the existence of a motive for deception. The simulation is common enough in those who seek damages for injuries, or in those who have a morbid craving for sympathy and attention. Its detection depends upon the skill of the physician, who, by distracting the attention from the part complained of, observes that the pain disappears, or, on the other hand, that pain is admitted in a part to which attention is directed ; moreover, the physician observes an absence of adequate physical alteration, and usually inconsistency in the symptoms, for the malingerer is seldom able to simulate a correct clinical representation for any length of time. Especially in the latter case is the observation of the invalid's surroundings of considerable importance. The so-called hysterical mask is of much value, for the bitter complaints and the placid or even smiling features cannot fail to strike the observer by their incongruity. True hysteria is apt to be deceptive, and more than one humiliating failure is recorded of even the most skilful of our craft. The difficulty is increased because actual physical changes occur, as amaurosis with dilatation of the pupil, contracture and induration about the joints, unquestionable anæsthesias, and palsies. True hysteria is often to be detected only after prolonged and painstaking study of the case ; the careful exclusion of organic visceral disease ; by the absence of the characteristic symptoms of the nervous degenerations, such as ankle-clonus, or altered electrical reactions, or changes of the fundus oculi ; and often by the impossibility of associating the sensory lesions with the known anatomical distribution of the nerves.

Objective Investigation of Pain. In order to estimate accurately the diagnostic value of pain, the statement of the patient must be cor-

rected by his expression, posture and manner, and the apparent nature of the disease. Pain is one of the cardinal symptoms of inflammation; vasomotor and muscular disturbances are often associated with neuralgia; any morbid condition exerting pressure on a nerve-trunk, as a neoplasm, callus, etc., commonly causes pain. Hence if the objective phenomena of these disorders are present, they lend color to the complaint of pain, and, if not, they should be inquired for. Attempts have been made to estimate the acuteness of the pain-sense with scientific accuracy, or at least to secure a practical method for measuring its varying intensity in different localities in the same case. Björnström, of Upsala, has contrived a pair of forceps that compress a fold of skin; the amount of pressure required to produce pain, which can be read from a scale, indicates the degree of sensibility or rather resistance to painful impression. Another instrument, Buch's, accomplishes the same thing by direct pressure, and hence can be used over the superficial nerve-trunks. Another method more generally available is the application of an induced faradic current of variable strength—single, naked-wire electrodes being best for this purpose. The common clinical method, by far the most inaccurate and only applicable in cases of marked analgesia, is a pin or needle forced through a fold of skin. No method has yet been suggested for even the approximate estimation of the acuteness of sensibility to internal pain, and it must still be left to the judgment of the patient.

The Clinical Value of Pain. The presence of pain is recognized by the above-mentioned circumstances. Its degree, with the limitations indicated, has been estimated. Its clinical value is then to be considered. From what has been said above, the converse of many of the propositions is true. By pain and the mode of its expression we can judge of the character, temperament, and nervous susceptibility and perturbability of the patient. It aids us in the recognition of hysteria and helps to detect the malingerer. We learn the patient's capability of resistance, and hence, in a measure, his strength. We learn the quickness of receptivity in consciousness of the peripheral irritation, or the degree of intelligence, or the amount of stupor; or, if conditions are present which usually cause pain, its absence may show disease of the conducting paths to the brain. Further, the absence of pain under the above circumstances points to the occurrence in the local process of such change as has destroyed peripheral nerve-endings. Thus, when pain ceases in dysentery gangrene has ensued. In intestinal obstruction its cessation indicates the same process. In profound shock pain is not complained of; the amount of pain, therefore, indicates the degree of shock. Hence, in peritonitis, in which shock frequently occurs, pain may be wanting entirely. The abdominal surgeons welcome its occurrence after an operation, as it indicates the absence of shock.

While the above lessons, from the presence or absence of pain, are not to be under-estimated, the value of pain to the physician is from the stand-point of diagnosis. By this symptom we may be enabled to determine the *location* of disease and the *nature* of the causal morbid process.

(*A*) The location of the disease is determined (*a*) by the seat of the pain and (*b*) in part by the mode of expression. The mode of expression also indicates its point of origin in a general way and its probable cause. They are (1) the facial expression, (2) the posture, (3) the reflex actions, (4) the associate phenomena. They need not be referred to again. (See page 37.) (*B*) The nature of the causal morbid process is judged by the study of pain from various stand-points. Thus in the consideration of the symptoms of pain we must learn (1) the mode of onset, (2) the duration, (3) the time of occurrence, (4) the character or variety, (5) the location, (6) the modifications produced by pressure, temperature, rest, motion, posture, electricity, drugs, and climate.

1. Mode of Onset The *mode of onset* of pain is in the majority of cases an indication of the acuteness of the morbid process. (*A*) The onset may be *sudden*, as (1) in gout or acute inflammations of serous membranes, as pleurisy or peritonitis; (2) in certain headaches, particularly in those of congestive or emotional origin; (3) in acute obstruction of canals; (4) in contraction of muscular structures in their effort to remove a foreign body, as in the intestines, the gall-ducts, the vermiform appendix, the ureters, bladder, or uterus; (5) in rupture of the structure in which it is developed. Here we have the most typical sudden pain. Thus, in rupture of an aneurism or of the heart there is sudden, sharp pain. In rupture or perforation of the stomach or intestines, or any of the hollow viscera, this character of pain arises. (6) Sudden pain also occurs in certain neuralgias or neurosal affections. It is seen in its most striking form in angina pectoris, locomotor ataxia, and in acute brow-ague, or trigeminal neuralgia. (*B*) The onset may be *gradual*, and may be associated with continuous increase in intensity or variation. Such onset indicates that the process is one of slow development and not attended by a "solution of continuity," as from rupture or tear. It usually occurs in various forms of rheumatism, in inflammations of muscles and of mucous membranes, in chronic inflammations of serous structures, in chronic bone disease, and in slowly developing mechanical pressure, as tumors.

2. Duration The *duration* of the pain indicates the acuteness or chronicity of the causal morbid process. (*a*) Pain of *short* duration is seen in the affections in which it develops suddenly (see Mode of Onset), in acute serous inflammations, and in neuralgias. (*b*) Pain of *long* duration, if constant, is usually due to organic lesions; if intermittent, it may be due to neuralgia. Pain that is continued over a *long* period of time excludes the sudden accidents that were previously mentioned, unless change in the character of the pain takes place.

Pain is also divided, as to duration, into temporary and constant pain. *Temporary* pain indicates an abeyance or relief of the morbid process, while the constant pain points to its continuance. *Constant* pains are seen in bone affection, in inflammation of muscles, in reflex pains due to chronic disease elsewhere, as the backache of uterine disease, or the inframammary neuralgias from the same cause. Pain may also be *intermittent* or *remittent*, *paroxysmal* and *periodic*. (*a*) Intermittent and remittent pains are characteristic of neuralgias, or point to a functional origin; they are recurring because the cause

which superinduces them is again operative. Thus recurring headaches due to eye-strain may be intermittent or remittent in the sense that they occur only when the eye is used. Attacks of such pain recur over a long period. (*b*) Paroxysmal pain is the form which occurs when there is obstruction of channels, as the gall-ducts in biliary colic, the intestines, the uterus, and the ureters in the various forms of colic to which they are liable. The paroxysms of pain recur in the course of the attacks. (*c*) The term *periodic* is applied to pains that occur at distinct intervals. Pain that is periodic has frequently for its cause malaria in some form. The toxic headaches and nerve headaches, as migraine, are often periodic. (Consult Headaches.)

3. The Time of Occurrence. The time of the occurrence of pain is important. Pains may occur in the daytime, or during the night exclusively. Nocturnal pains are common in syphilis. They are usually due to periosteal inflammation. Diurnal pains are usually reflex from functional disorders. Some pains, as headache due to cardiac weakness and to forms of anæmia, are present during the day, because the patient is in the upright position. They disappear in the recumbent position, and hence are absent at night.

The time-relation of pain to functional acts is of importance. Thus in gastric pain its relation to the taking of food is to be ascertained. Pain coming on before meals is gastralgic; occurring after meals, it is due to ulcer or cancer, sometimes to indigestion. Chest pains, increased by the act of breathing, are muscular or pleuritic.

4. Character. Pain may be sharp, lancinating, or stabbing; it may be throbbing, or it may be dull. Sharp, lancinating, or stabbing pain is usually due to inflammation of serous membranes, to colic in various forms, and to forms of neuralgia. *Cutting* pain is a sharp form that occurs in flatulent colic. *Throbbing* pain is usually associated with acute inflammation, whether superficial or deep. It may be rhythmical with the pulsations of the heart. *Dull* pain is due to slow chronic inflammation in the bones and in the viscera; it is the pain of myalgia and of fatigue in the muscles. It may be of an *aching* character. But aching pains may also be general; they are found among the prodromata of the acute diseases, attend and follow a chill, and occur in most characteristic form in influenza and dengue. *Pressing* pain is complained of when it attends an attempt to remove material from the viscera, as the passage of water when the bladder is inflamed; the passage of fæces in dysentery. The term *tenesmus* is applied to it, so that we have vesical tenesmus and rectal tenesmus. The passage of clots or other material from the uterus is attended by pain with pressure or "bearing-down," as it is termed.

Nature of the Disease. Finally, the character of pain is often an indication of the nature of the disease as well as of the tissue affected: 1. Thus boring and constant pain is seen in bone and periosteal disease. 2. Soreness or aching in muscular affections. 3. The pain is sharp and stabbing when serous membranes are affected. 4. Smarting and burning, or, perhaps, dull and sore when mucous membranes are inflamed. 5. Burning or itching in affections of the skin. 6. Dull and usually constant in visceral affections, although in malignant disease of various

organs it may be sharp and paroxysmal. 7. Aching, burning, and throbbing in the nerve-trunk and its distribution, with tenderness, commonly indicate neuritis. 8. A sense of swelling, distention, or bursting attends the pain of obstructions of hollow viscera, as in renal or hepatic colic. 9. Rending or tearing pain may be complained of when a hollow viscus or sac is ruptured, as notably in the rupture of the sac of extra-uterine pregnancy. (See "pain crises," page 44.)

5. Location and Distribution. It may be of questionable advantage in some cases that the localization of pain generally indicates the situation of the morbid process. Too often an apparently adequate explanation of the symptoms may thus be found, whilst other pathological changes may be overlooked. But, on the other hand, the condition to which attention has been called by the pain might, on account of its obscurity or unusual location, altogether escape observation.

The *location* is, in general, an indication of the seat of the disease. It may be accepted as an almost universal rule that pain due to a local process is limited to the immediate or associated nerve-supply of the diseased region. This holds true even when the *referred pains*—that is, those felt in the associated nerve-supply—are as far distant from the site of the morbid process as the knee pain of coxitis, the shoulder pain of hepatic disease, pain in the neck from pericarditis or diaphragmatic pleurisy, the ear and temporal pain of lingual carcinoma, the pain in the legs from cancer or ulcer of the rectum, the testicular and thigh pain of renal colic, or the umbilical pain of vertebral disease.

On the other hand, Hilton lays down the rule that pain in any part, in the absence of a local process, is due to exalted sensitiveness of the nerves of that part, and depends upon a cause remote from the painful area. The term *sympathetic* is applied to this group of pains. Further, Hilton remarks that pain on the surface of the body must be expressed by the nerve which resides there, and, hence, the cause of the pain must be situated between the peripheral termination and its central origin. This applies particularly to the pains which arise from disease of the vertebræ and the referred pains described above. As a corollary to this, in the investigation of the cause of pain, the nerve, its anastomoses, and the organs supplied by it should be investigated.

But the pains may be general as well as local.

1. General pains are due either to central or to peripheral disturbance of the nervous system by a poison circulating in the blood. This may be the poison of fevers, or it may be a rheumatic or gouty poison. It is seen in the common affection known as "cold," when the pains are probably myalgic. In syphilis, malaria, lead-poisoning, and toxæmias generally there is general pain, soreness, and fatigue. General pains are not confined to the muscles, but are also seated in the fibrous structures and bones. In their more severe forms such pains occur in dengue, and are known as "break-bone."

2. Local pains may be (*a*) superficial or deep-seated; (*b*) circumscribed or diffused.

(*a*) *Superficial* pains are due to involvement of the superficial nerves distributed to the skin or to the muscles directly underneath, or to the structures in close relation to the skin, as the peritoneum, the pleura,

or the pericardium. *Deep-seated* pains, when in the extremities, are due to bone disease; when in the abdomen, to disease of the viscera, particularly inflammatory affections, to aneurism, or bone disease; when in the chest, to disease of the aorta and mediastinum.

(*b*) *Circumscribed* pain is always due to a small area of disease, or is reflex. Thus, in ulcer of the stomach the pain is usually circumscribed to a small area in the epigastrium; in inflammation of the appendix, to the region of that structure. *Diffused* pain indicates involvement of a large area with less intensity of process than when circumscribed. When the pain is *diffused*, or, as it is sometimes called, radiating, over an area of nerve distribution, its point of origin may be found somewhere in the course of the nerve, either in the trunk or in one of its branches. *Corollary:* Given pain in a locality, study the nerve-supply of that region and the nerve anastomoses connected therewith. We learn much from the study of this distribution. The *referred* pains have been indicated (page 43). Among others, the pain of angina radiates down the arms. The pain of diaphragmatic pleurisy is referred to the front of the abdomen above the umbilicus. Radiating pains, however, are chiefly due to disease in the course of the nerve, the pain being referred to its trunk and terminal distributions, as pain in the foot in sciatica. Pain from pressure upon the nerves at their exit from the spinal canal is at the periphery of the nerves, as in the centre of the abdomen, and not at the point of exit. Pain in this locality is frequently an indication of disease of the vertebræ, propagated by the sixth or seventh dorsal nerve. Pain between the shoulders is often due to aneurism which presses upon the vertebræ. (See Pain in the Heart.)

Bilateral, symmetrical, and *superficial* pains indicate a central or bilateral cause; while, on the other hand, *unilateral* pain implies a seat of origin which is one-sided.

Peripheral Pain of Central Origin. We have referred to pains of the extremities or trunk due to central disease. In meningitis and other general organic affections of the brain and cord peripheral pains are frequent, and may be the earliest and most striking symptoms. Indeed, it is very common to find patients with spinal-cord disease who have been treated for a long time for what was supposed to be rheumatism. The pains in the joints of central origin may be constant, or paroxysmal and lancinating when the disease is chronic. (See Character.) The cardinal rule, that all peripheral pains, without obvious local cause, should lead to an examination of the nervous system, must never be forgotten. The paroxysms of pain may be most excruciating, and sometimes cause collapse. They are known as *painful crises*. Pain may be complained of in various viscera, as well as in the joints. Sudden, intense pain, with functional disturbances of the affected viscera, occurs independently of any lesion of the part or of any apparent exciting cause. One class of the attacks is known as *gastric crises*. The pain is in the epigastrium, and is associated with vomiting. In another class *laryngeal crises* occur, with pain in the larynx and violent spasmodic cough, with dyspnœa. The pain extends over the shoulders. Or we may have *rectal crises*, with sensation of burning in that situation; *urinary crises*, simulating renal colic, and *genital crises*. Pains, in

crises, also occur in the muscles. Crises occur chiefly, if not entirely, in locomotor ataxia. They are distinguished from pain due to other causes by their sudden onset, their extreme severity, the absence of organic disease or local cause in the affected viscera, the sudden termination, the normal condition between the attacks.

6. Pain Modified by Pressure, Movement, Rest, or Mental Diversion. We also study pain under the influence of pressure, movement, temperature, rest, etc. Pain that is modified by *pressure* is generally superficial. It is usually of an inflammatory origin. The variety of the pressure gives some clue to the nature of the pain. If the pain is increased by pressure of the finger-tips, it is due to ulcer or inflammation when internal and to inflammation if external. Although of visceral origin, gastralgia and colicky pains in the intestine, which may be a neurosis, are relieved by pressure, particularly if the whole hand is applied. Pain from the dislocation of an organ, as a movable kidney or displaced uterus, or from dependent viscera, may be relieved by judicious pressure in the proper direction, so as to relieve the displacement.

Pain from affections of the nerve-trunks can be distinctly localized by pressure in the course of the nerve-trunk, and particularly at the points where the cutaneous filaments of the nerves come through the fascia. These points in the thorax are along the vertebral column, in the axillary region, and anteriorly about the parasternal line—the points of Valleix. We distinguish neuralgias from myalgias by the presence of these tender points. Pain due to bone disease can frequently be distinguished in this way. By pressure or weight upon the head or shoulders we may ascertain if pain is due to vertebral disease. The presence of renal calculus or of gall-stones may be determined by the excitation of pain by pressure.

Pain increased by *movement* points to an affection of the bone, muscle, joint, or nerve in the part moved; groups of muscles may be isolated for the tests. Some few pains are relieved by movement of the body, only because the mind is diverted in this act. Pain, when superficial and increased by movement, is due to neuritis, myalgia, or rheumatism.

Almost all pains are modified by *rest*. Its influence has but little diagnostic significance. In some cases of doubt as to the nature of a visceral pain, functional rest of the organ, by which relief is obtained, may aid in determining its locality. Thus, rest to the eye may relieve a headache, the nature of which was obscure until this respite was secured. Pain modified by *temperature* (cold or heat applied to the spine, ice or hot water in a sponge) and by electricity usually gives information as to the seat of the disease in the spinal column, of which the pain is the external expression. Pain modified by *climate* is rheumatic or neuralgic; if modified by weather or season, it is due to neuralgia or neuritis, whether of gouty or traumatic origin.

The patient may describe an excruciating pain in an area, but not exhibit outward evidence or physiological change which should accompany such suffering. Thus the pain may simulate that of peritonitis. Such pain is often modified and mollified by fixing the *attention* of the

patient on some other part or on some extraneous subject, when the previously alleged tender area may be pressed upon without causing any evidence of suffering. Similarly, attention may be called, by a leading question, to pain in some other region. The admission of the occurrence of such pain, and other evidences of hysteria, point to the underlying causal factor in the production of pain. A most important characteristic of pain, and one that serves to distinguish the pain of organic disease from that of hysterical origin, is its variability with excitement, or on fixation of the attention of the sufferer on other parts. Moreover, the subject will fall into the trap of describing it as having characters contrary to the usual attributes of pain or being associated with phenomena not compatible with the pain—if judicious leading questions are put.

Résumé. Notwithstanding clinical investigation we may not be able from the character and locality to determine the real cause of the pain. In general it may be borne in mind that pains are due (1) to disease of the central nervous system or the nerve-trunks; (2) to inflammations; (3) to intoxications, as from malaria, lead, and other forms of toxæmia; (4) to pressure on the nerve-trunks; (5) to reflex influences. If in doubt, therefore, the general symptoms and condition of the patient must be ascertained in order to determine the causal origin, and hence the true nature of the pain. In all cases of pain the controlling motive in diagnosis should be to determine the *general condition* of the patient and find the *cause* of the pain.

Reference must be made to the curious change that takes place in persons with chronic morphine intoxication. Such persons are very apt to have functional pain. This form of pain is usually paroxysmal and severe, and may simulate organic pains. The most common clinical form seen is gastralgia. The subjects of locomotor ataxia suffer from pain, on account of which they have to take enormous doses of morphine. This habit is soon acquired, but notwithstanding the large dose of the drug paroxysmal pain continues; in its severity it simulates the crises of the primary disease. It becomes a very difficult matter, and is often impossible, to decide whether the pain is due to the morphine-habit or to the primary affection. (See Source of Error, p. 38.)

Pain in the Head.

Pains in the head may be classified, according to location, into those due to affections of the *scalp*, those due to affections of the *cranium*, and those due to *intracranial* conditions.

1. Affections of the scalp are to be further classified as those of the *skin*, those of the occipito-frontalis *muscle*, and those of the *nerves*. The occurrence of itching and burning commonly indicates some local condition of the skin; if the itching is slight, seborrhœa should be looked for; if more severe, eczema; burning and itching of a severe type commonly indicate dermatitis venenata; the pediculus capitis should not be forgotten. A feeling of tension, with soreness, accompanies the eruption of erysipelas. Intense local irritations are caused by burns and scalds; the latter, however, are alone likely to give rise to

error, because the hair is not immediately destroyed. A sore feeling, with local tenderness, limited to a sharply defined swelling, with a sensation of less resistance in the centre and some darkening of the skin, is diagnostic of a bruise. Hyperæsthesias of the scalp frequently accompany meningeal and cranial affections, and there are even local changes, such as the so-called puffy tumor of necrosis of the inner table of the skull.

Sharp pains in the occipital or frontal region, increased by wrinkling the scalp, or brief pressure, but generally relieved by firm and constant pressure, occurring with irregular periodicity, and associated with meteorological changes, are suggestive of occipital *myalgia*. The diagnosis is confirmed by the presence of other symptoms of lithæmia.

Neuralgia occurs in paroxysms, accurately located in the course of one or more of the nerve-trunks, and presenting points of special sensitiveness where the nerve emerges from the skull and where it divides for its cutaneous distribution. The pain is usually relieved by firm pressure, but it is to be remembered that sharply localized pressure on the nerve-trunks against the hard skull will cause a traumatic tenderness. The character of the pain is variable; it may be of the most acute or rending form, or, but more rarely, a persistent dull ache; it may be throbbing, or occur in successive paroxysms at brief intervals, or it may be regularly periodic. There are often associated vasomotor, secretory, and motor disturbances; local blushing or sweating may be observed along the course of the nerve, and spasms may occur in the muscles of the eyelids, for instance, or more general spasms, as in the terrible tic douloureux, distinguished by pain from tic convulsif. The commonest seat is the supraorbital, the dental, the auricular, and the occipital nerves. In the great majority of cases it is unilateral.

The sensory *nerves* of the scalp and face are the trigeminus and the branches of the cervical plexus. The distribution is as follows: the *ophthalmic* division of the trigeminus is distributed to the eyeball, lachrymal gland, the mucous membrane of the nose and eyelids, the integument of the nose and upper eyelid, the forehead, and the anterior half of the hairy scalp. The *superior maxillary* division supplies the skin over the malar bone, and that of the lower eyelid, side of the nose, and upper lip; the upper teeth, the upper part of the pharynx, the antrum of Highmore, and the posterior ethmoidal cells; the soft palate, tonsil, and uvula, and the glandular structures of the roof of the mouth. The *inferior maxillary* division is distributed to the side of the head, the upper anterior portion of the external ear, the external auditory canal, the lower lip, and lower part of the face; the tongue, the mouth, the lower teeth and gums, the salivary glands, and the articulation of the jaw. The *great occipital* is distributed to the back of the head, the *small occipital* to a narrow region just in front of it, and the greater *auricular* to the skin of the posterior portion of the pinna and the skin over the mastoid and parotid gland.

Pain simulating neuralgia is frequently due to some *local irritation*; foreign bodies have been known to cause paroxysmal attacks for a number of years, until removed; diseases of the bones are a prolific source, especially in the case of the jaws and the cervical vertebræ.

Enlarged cervical glands occasionally irritate the great auricular or small occipital nerve. Bilateral occipital pain is very characteristic of cancer of the cervical vertebræ. In these cases there is usually pain on movement of the head or pressure upon it, and some stiffness of the neck. Intracranial growths occasionally cause pains, usually paroxysmal, limited to one of the branches of the trigeminus.

Reflex Neuralgia. Certain of the cephalic nerve-pains are symptomatic of disturbance in the associated but distant nervous distribution. Pain in the region supplied by the ophthalmic division is very common in influenza. It is usually dull, aching, and continuous, increased by pressure and anything tending to increase congestion. A severe, acute attack of indigestion will produce ocular and supraorbital pain. Refractive lesions of the eye cause the same kind of pains, which are, however, increased by using the eye and relieved by rest and atropine. The use of the latter is an important diagnostic procedure. Pain in the temporal region and the external auditory meatus is often due to intense irritation of some of the branches of the inferior dental; the usual cause is cancer of the tongue, but irritable lingual ulcer may also produce it, and even severe inflammatory conditions of the lower jaw. The pain is described as sharp, lancinating, and paroxysmal, liable to exacerbations, especially when the primary lesion is irritated, and relieved when it is alleviated. Pain may be caused in the ear alone when there is irritation of the teeth.

Systemic Neuralgia. Perhaps in the majority of cases of cephalic neuralgias the cause is to be found in some systemic disturbance. If the attack is preceded by a desire to sleep, occurs when the dew-point is high, and is associated with increase of urates in the urine, it is probably *lithæmic;* the pure gouty forms are most apt to succeed indulgence in rich food or red meat, and there is ordinarily irritability of temper. *Diabetic* neuralgias are invariably worse as the amount of sugar excreted is increased, and there are usually similar affections of the nerves in other parts of the body. Regularly periodic pains, worse in the spring and fall, occasionally preceded by a slight chill or malaise, suggest chronic *malaria.* The diagnosis can readily be confirmed by examination of the blood and by the detection of enlargement of the spleen. *Syphilitic* neuralgias are usually worse at night; the pain is described as boring, and may be periodical. There is likely to be some thickening of the bones, and perhaps a diminution of elasticity of the tissues, and almost always local tenderness. The pain is almost immediately relieved by iodide of potassium. In *anæmic* neuralgias the pain is not characteristic, but it is temporarily improved by the recumbent posture and stimulants, and is worse during menstruation. The general appearance of the patient and an examination of the blood readily suggest the cause. In *locomotor ataxia* there are occasionally cephalic crises of a neuralgic nature; these come on suddenly and are exceedingly severe, but usually occur only at long intervals; the pain is shooting or stabbing, and does not remain located in one nerve-trunk. Chronic *lead-* and *alcohol-poisoning* also cause neuralgias, but they are not of themselves characteristic, and never occur as isolated symptoms, being frequently associated with peripheral neuritis.

Secondary Neuralgia. Dull, burning pains, commencing perhaps with a chill, and accompanied by febrile symptoms, indicate *inflammations* of the mucous membranes of the head. A dull, persistent headache located just beneath the eyebrows often accompanies coryza, and indicates extension to the frontal sinuses; if the nose alone is involved, there is a feeling of fulness and occasional sharp pains or tickling sensations. A feeling of dryness and some discomfort on swallowing accompanies the various forms of stomatitis and pharyngitis; in the latter there is also a sensation of tickling and fulness in the ear, due to extension along the Eustachian tube. Pain at the angle of the *jaw*, with tenderness, and increased on swallowing, almost invariably unilateral and associated with swelling of the parotid, is unmistakably due to parotitis. The neuralgias and inflammations of the middle *ear* are exceedingly painful; they may consist of a sharp continuous pain, or a series of regular exacerbations and remissions, or a throbbing sensation; pain often radiates to the jaws and side of the face. As suppuration occurs, the feeling becomes one of extreme tension until the membrane is perforated, when there is immediate relief. Tinnitus throughout the whole course of the case is very common. The inflammations of the *eye* produce local pain, usually causing the sensation of a rough foreign body. Usually there is a slight supraorbital tenderness, and, in iritis, sharp pains radiate over the whole area of distribution of the two upper branches of the fifth. Certain ulcers of the *mouth* are comparatively painless, noma often developing insidiously. Syphilitic ulcers are to be distinguished by their painlessness from simple and tubercular ulcers, which are very irritable, and carcinomata, which are liable to paroxysms of pain even when not irritated.

It may not be out of place to mention the value of certain anæsthesias as diagnostic signs; thus in neuritis of branches of the fifth there may be cutaneous anæsthesia while there is tenderness over the nerve-trunk.

2. Affections of the Cranium. *A dull, constant headache*, limited to a small area, later increasing in severity, and the pain assuming, perhaps, a boring character; tenderness, often very severe, over the affected area, and probably slight œdema of the scalp, with some rigidity of the muscles of the neck, and the ordinary signs of the inflammatory process, indicate inflammation of the cranial *bones*. In the simple cases there will usually be some history of injury, the pains will not be especially periodic, and the fever will be irregular. In the syphilitic cases there will be the history and symptoms of infection, the pain will become worse at night, and usually there will be concomitant rise of temperature. The pains will also be controlled by iodide of potassium, but as it often requires enormous doses to accomplish this result, the failure of a moderate dose should not be considered as excluding syphilis.

3. Intracranial Headaches. Intracranial headaches are functional or organic. Both forms may be acute or chronic. The typical acute functional headache is seen in the more or less common type known as *migraine* or *hemicrania*.

Migraine is a periodical neurosis characterized by pain in the trigeminus and other cranial nerves. The headache is usually unilateral,

and, as it is probably due to vasomotor disturbances, is always associated with vasomotor symptoms. It occurs more particularly in women, frequently begins in early childhood, and continues throughout adolescence. It is often hereditary. It occurs most frequently in women who suffer from anæmia or from menstrual difficulties. It sometimes occurs in the early stages of secondary syphilis. The habit which predisposes to the headache may develop after long physical or mental over-exertion. The attacks, however, are excited by over-exertion, mental excitement, or disturbances of digestion. Pain of migraine is possibly situated in the pia and dura mater.

Symptoms. The attack develops with or without premonitions. In each individual different prodromal symptoms are recognized as indicating the approach of an attack. Undue nervousness, a general sense of discomfort, pressure or heat in the head, vertigo, tinnitus, spots before the eyes, excessive yawning, and repeated chilliness are the most common.

Premonitory Symptoms. The pain is most frequently felt on the left side of the head first. It is seated in the anterior frontal, the temporal, or parietal region. The pain is continuous, and increases in intensity to the height of a paroxysm. Painful points are not usually detected, although the whole skin may be hyperæsthetic. The patient is sensitive to light and sound, intolerable nausea intervenes, and vomiting may occur at the height of an attack. The eye-symptoms are very pronounced. Flashes before the eyes, scintillating scotoma, or hemianopia may occur.

The vasomotor symptoms that attend the attack are of two varieties, dividing the disease into the spastic and angioparalytic forms. In spastic migraine the skin on the affected side is cool, the forehead and ear pale, the temporal artery is contracted, the pupil is dilated, and the flow of saliva increased. In the paralytic form there is redness of the face on the affected side. The temporal arteries are dilated and pulsate strongly. The face is hot, the pupils contracted, and there is often unilateral sweating.

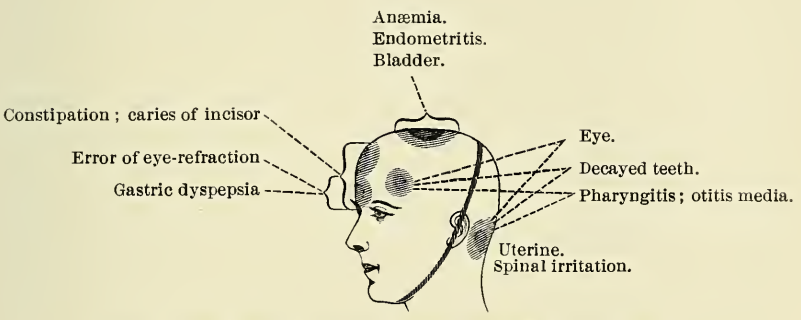
Chronic Headaches. Chronic functional headaches are usually habitual in the sense that the attacks are constant, but there may be longer or shorter intervals of freedom from pain. The nerves affected are the trigeminus, and the four upper cervical and sensory branches of the vagus to the posterior fossa of the skull. Three types of such head-pains are seen: ordinary headache, migraine, and neuralgia. Headaches are caused, as a rule, by diffuse irritations located in or referred to the peripheral ends of the nerve-tracts above referred to. Neuralgias, on the other hand, are caused by irritations of the trunks of these nerves.

Causes. 1. Hæmic. (*a*) Anæmia; (*b*) diathetic states (gout, rheumatism, diabetes); (*c*) infections (malaria, syphilis, specific fevers). 2. Toxic (lead, and other mineral poisons, alcohol, the poison of uræmia, tobacco). 3. Neuropathic states (epilepsy, neurasthenia, chorea, hysteria, neuritis). 4. Reflex causes (ocular, nasopharyngeal, auditory, gastric, sexual, uterine). 5. Organic disease.

Headaches are divided according to their *situation* into frontal, occipital, parietal, vertical, diffuse, and combinations of both. The most

common forms are the frontal, the frontal-occipital, and the diffuse. Ocular headaches are usually frontal when due to errors of refraction. When due to muscular insufficiencies they are occipital and cervical. Nasopharyngeal headaches are dull, frontal, or diffuse. When the pharyngeal tonsil is enlarged the headache may be dull, frequently recurring, and seated in the occipital region. In follicular tonsillitis and in obstruction of the Eustachian tubes the headaches are diffuse. In disease of the middle ear they are temporal and occipital. Gastric or dyspeptic headaches without constipation are often occipital, sometimes frontal. With constipation and intestinal irritation they are diffuse and frontal. Uterine and ovarian headaches are occipital and vertical. *Neuropathic* headaches are seated on the top of the head, as in clavus, or they are associated with spinal irritation. Neurasthenic headaches are usually associated with a sense of pressure or weight, and are seated in the frontal and vertical regions. In spinal irritation the pain is of a boring character in the occipital region. The earliest symptom of the neurasthenic headache is neck-weariness and pain in the neck. The

FIG. 1.



Showing the location of pain in various headaches. (After DANA.)

neurasthenic headaches occur in brain-workers when the brain and eyes are overtaxed. Headaches in epliepsy are severe, and are confined to the vertical or occipital region.

Organic headaches are usually violent, associated with fulness and throbbing. They may be remittent, becoming more intense with each exacerbation. The organic headaches may be due to inflammation, to abscess and softening, to tumor, to congestion of the brain, and to inflammations in the meninges. Anything which increases the blood will increase the pain in organic headaches. In acute inflammation of the brain the pain is agonizing, continuous, associated with vomiting and fever, and sometimes delirium. In abscess of the brain the pain is less violent. It is occasionally paroxysmal and attended by paralysis and disturbed intellection. In tumor of the brain the headache is severe and paroxysmal. In congestion the pain is dull, increased by stooping, by sleep, and by bodily or mental fatigue. Some congestive headaches are due to violent exercise, and are relieved by bleeding at the nose. In all congestive headaches the face is flushed, the bloodvessels are turgid, and the vessels in the eye-ground will be

found to be overfilled. In meningitis the pain is constant, is more or less fixed, and sometimes very sharp. Syphilitic headaches are frontal or temporal, worse at night, and often periodic.

Headaches are divided according to the *character* of the pain: 1. Pulsating and throbbing. 2. Dull and heavy. 3. With constriction, squeezing, or pressing. 4. Hot and burning. 5. Sharp and boring. The headaches of the first class are usually associated with vasomotor disturbances, as in migraine. To the second class belong the toxic and dyspeptic headaches; to the third, the neurotic and neurasthenic; to the fourth, rheumatic and anæmic; to the fifth, hysterical, neurotic, and epileptic. *Vertigo* is a common accompaniment of the dyspeptic type of headache situated in the frontal regions. *Somnolence* is more marked in the syphilitic, anæmic, and malarial headaches. *Nausea* is more common in occipital forms of headache.

Duration. Eye-strain causes occipital pain, which is rarely persistent, but comes on after prolonged use of the eyes. It may be associated with headache in other parts, due to other causes. In chronic meningitis the headache is persistent and located in the vertex or the parietal regions. When thickening of the meninges, with adhesions, takes place from trauma, there is constant pain with frequent exacerbations, sensitiveness of the head, incapacity for study. Uræmic headache is not constant. Persistent headache may be present in the latter stages of Bright's disease and in diabetes. In atheroma pain in a part or the whole of the head is common. It may be persistent, though subject to exacerbations in case of excitement or violent exercise. Headache following study, in children, is due to brain-strain, to eye-strain, or to indigestion. Persistent headache is sometimes due to asthma. In rare instances headache is said to be idiopathic. Neuralgic headaches are usually periodic, and may be associated with throbbings or pulsations. They are associated with vasomotor signs. Hysterical headaches are irregular and shifting; they persist after all causes are removed; they are replaced by pain in other parts of the body. They are usually associated with other manifestations of hysteria.

Neuralgia.

Neuralgia is characterized by pain in the course of distribution of the affected nerve. The pain is of pronounced severity, and occurs in remissions and intermissions. The symptoms of a neuralgic paroxysm may be preceded by hyperæsthesia over the part subsequently affected. The pain is of a burning or shooting character. It is usually limited to the distribution of the affected nerve, but may extend into other regions. It may be excited by external irritants, by mental excitement, and often by movement of the part. On examination the area of distribution of the affected nerve may be found to be anæsthetic, but usually there is a hyperæsthesia of the skin. Wherever the affected nerve is accessible to pressure pain can be elicited. The nerve-trunk may be tender during the attack, as well as during the intervals. In neuralgia there is often some spasm of the muscles supplied by the nerve.

Vasomotor symptoms are common. The skin may be pale or reddened. When the trigeminal nerve is affected the skin and conjunctivæ are both reddened. The secretions, as the tears, may be modified. Eruptions like urticaria or herpes may develop along the course of the nerves. Prolonged neuralgia may cause marked nutritive disturbances.

General Conditions. A patient who is subject to neuralgia may be in apparent good health. The neuralgia may be due to constitutional causes, as rheumatism or gout; to some form of toxæmia, as malaria; to some condition of the blood, as anæmia; and may be due to trauma or to cold.

The following individual forms of neuralgia are seen: 1. Neuralgia of the trigeminus, or *tic douloureux*. The entire fifth nerve or some of its branches are affected. The pain is often severe and may be associated with twitchings, with vasomotor disturbances, with eruptions, and with changes in the secretions. Trophic changes, as the hair turning gray, or ulceration of the cornea may follow. Usually a single branch is affected, either the first branch (ophthalmic), the second branch (supramaxillary), or the third branch (inframaxillary). Points of pressure are, as a rule, readily detected at the foramina for the exit of the nerves. 2. Occipital neuralgia. 3. Neuralgia of the brachial plexus. 4. Intercostal neuralgia. 5. Neuralgia of the lumbar plexus, of which we have lumbo-abdominal, crural, and obturator neuralgia. This form of neuralgia (lumbar plexus) must not be confounded with bone and joint disease, with lumbago, renal colic, appendicitis, and uterine affections. 6. Sciatica. 7. Genital and rectal neuralgia.

Trigeminal neuralgia must be distinguished from headache due to other causes, affections of the bones and periosteum, and affections of the teeth. The distribution and paroxysmal character of the pain and the points of tenderness assist in the diagnosis.

Pain in the Legs and Feet.

Paroxysmal Pain. Pain in one leg may be due (1) to sciatic neuralgia or (2) to neuritis. The former does not exhibit localized tenderness and is not aggravated by movement. The latter, also called *sciatica*, is recognized by tenderness in the course of the sciatic nerve or at its exit from the pelvis, and by increase in the pain when the limb is extended by forced movement. The pain is constant, worse at night, and characterized by agonizing paroxysms. It follows exposure to cold or may be caused by rheumatism. One of the many branches of the sciatic may be affected, exhibiting tenderness in its course. If the sciatica persists, wasting of the muscles, herpetic eruptions, and areas of anæsthesia over the affected leg may be found. Such neuritis is usually traumatic (cold), alcoholic, rheumatic, gouty, or syphilitic; the exact cause in each case must be ascertained by the associate phenomena and by the exclusion of other causes. Pain in the leg may also be due to (3) pressure on the sciatic nerve by a pelvic growth, (4) neuroma, (5) rheumatism, (6) syphilis of bone or a syphilitic gumma of muscle or connective tissue.

Fixed pain in the leg, in contradistinction to the mobile pains of neuritis, is usually situated in the *fasciæ* or *muscles* or in the *bones*. It may be due to rheumatism, when the pain is diffused and the nerve points of tenderness are wanting. It may be the result of strain or injury, a history of which must be carefully inquired for. The latter may be the exciting cause only, in a person of rheumatic diathesis, the fixed pain at the situation of the injury being due to rheumatism. Fixed traumatic pains are usually accompanied by tenderness on pressure, and aggravated by movement both active and passive, the tenderness on pressure not necessarily being in the nerve-trunk. In malignant disease of the long bones, mobile neuralgic-like pains may precede for some time the fixed pain of the permanent process. (See "A Case of Carcinoma of the Bones," J. H. M.)

Bilateral pains in the extremities are often of central origin, and may be due to spinal sclerosis; to malignant disease of the vertebra pressing on the cord; to pelvic growth, or lumbar abscess, causing pressure on both nerve-trunks in the pelvis.

Pains of the *feet* not due to affections of the large nerve-trunks are:

1. Pain in the Articulations due to Flat-foot. This may be in the tarsus or at the metatarsal articulations. It is a common cause of pain in the extremities, and may be unilateral or bilateral. Flat-foot from breaking of the arch can readily be recognized; pressure on the sole of the foot may increase the pain.

2. Pain in the Heel. This is often of gouty origin, and is a persistent source of complaint in many instances.

3. Pain in the Interosseous Spaces Between Distal Ends of the Third and Fourth Metatarsal Bones (Morton's painful affection of the foot). It occurs in people who are on their feet a great deal, is relieved by a night's rest, increases as the day goes on, and is increased by pressure or by wearing a tight shoe. It is worse in wet and cold weather. Localized pressure at the point on the sole indicated above causes extreme pain.

We cannot leave the extremities without a word regarding pains in the extremities of distinctly central origin—the forerunners of hemorrhage into the brain. Mitchell has called attention to these pains. They occur suddenly without evidence of local disease; they are located in one of the extremities, usually the leg, are excruciating, and not influenced by position, local applications, or pressure. In a patient with hard arteries and high pulse-tension they should be looked upon with suspicion.

Pain in the Arms.

Unilateral Pain. It may be due (1) to neuritis associated with tenderness of the nerve-trunk; (2) to neuroma, as, indeed, any peripheral nerve may be affected; (3) to simple neuralgia or neuralgia from the pressure of enlarged axillary lymphatic glands; of a morbid growth of an aneurism on the nerves; (4) to rheumatism or myalgia; (5) to bone disease.

Bilateral pain in the arms is of central origin, due to diseases of the

vertebra or of the spinal cord, or neuralgic, due to anæmia or toxæmia of some form.

Pains of the Thorax.

Painful diseases of the muscles and of the viscera will be considered in the chapters on Diseases of the Heart and Lungs. Pains of reflex origin will be referred to. They are usually seated in the *shoulder* or the *back*, and are due to liver or gastric disease. The pain of liver disease is referred to the right shoulder; of ulcer of the stomach, to the interscapular region and the lumbar region, or to the top of the shoulder, as in a case observed by Wood.

Pain behind the *sternum* is often a reflex neurosis from gastric disorder. It may occur in bronchitis. It may also be due to cancer of the mediastinum, to aneurism, or angina. Pain in the sternum or ribs is syphilitic or due to periostitis or necrosis following typhoid fever, rarely to cancer. Chronic fibrous inflammation of one or more of the attachments of the muscles is of common occurrence. The pain lasts for years. It is persistent, sometimes associated with stiffness; it is increased by movement, and there may be extreme aching pains in the parts. The pain of vertebral caries transmitted along the course of the nerve has been referred to.

Girdle-pain. This is a peculiar pain or sensation in the trunk, due to disease of the spinal cord. It is described as the sensation of a band drawn tightly around the body. It varies from a simple drawing sensation to extreme pain which encircles the trunk. It is situated above the level of the umbilicus. In milder forms it is due to chronic myelitis or spinal sclerosis; in severe forms to inflammation of the nerve-roots, or to cancerous, syphilitic, or tubercular disease of the meninges.

Pain in the Spine.

Pain in the spine is due less frequently to organic disease of the cord than to acute or chronic inflammation of the meninges, to disease of the bones of the vertebral column, or to curvature of various forms from muscle-weakness. Rhachialgia and tenderness in the course of the spine occur after concussion.

I. Disease of the Spinal Cord. In organic disease of the cord pain may be referred to the loins, the sacrum, or to the parts about the spine, but not to the spinal column itself. In the same disease of the cord we may have also the eccentric or radiating pains, of which mention has been previously made, due to irritation of posterior nerve-roots. They may be dull, resembling those of rheumatism. In acute cases the pains are accompanied by febrile symptoms, which may simulate rheumatism, especially when the other spinal symptoms are in abeyance. In chronic cases these peripheral spinal pains are influenced by the weather, and this likewise makes it difficult to distinguish them from rheumatism. Rheumatic pains in the limbs occurring after middle life, with or without joint-changes, should suggest locomotor ataxia.

In this affection sharp and darting pains, "pain crises," and girdle sensations occur.

II. Disease of Vertebræ. Fixed localized pain at some point in the vertebræ points to traumatic, syphilitic, or tubercular caries, or to pressure necrosis, as by an aneurism. Pain due to vertebral disease is both local and radiating. It is increased by pressure directly on the spinal column (on the head), by heat or by cold, or by electricity, applied over the part. It is relieved by removing the pressure of the weight above, as by raising the head or shoulders. It is relieved by the absolutely recumbent posture. With this pain the movements (flexibility) of the spine are interfered with, because of spasm of the muscles or anchylosis; there may be deformity. When the patient is placed upon a flat surface the normal lumbar arch is changed.

III. Disease of Meninges. Pain due to *meningeal disease* is local and radiating. It is associated with muscular spasm and stiffness of the spinal column.

IV. Spinal Curvature. The pain of curvature from muscular weakness extends along the nerves. The patient is afebrile. The signs of organic disease above mentioned are absent, but muscle-weakness and general signs of debility are present. Pain in the spine frequently attends *scurvy* and *rhachitis*. It may be accompanied by paresis of the muscles and closely simulate an organic brain or cord disease.

Pain in the Side.

Pain in the left side—the so-called inframammary pain—is one of the most frequent complaints heard by the practitioner. By discussion of it we can show how the symptom pain, wherever situated, must be investigated in order to determine the tissue affected and the nature of the disease. The tests used in the study of nerve affections (*q. v.*) are not given. It may be due to many causes, to exclude any one of which inquiry as to the mode of onset, duration, and character of the pain must be made. Then the structures underneath and about the seat of pain must be examined. 1. The *skin:* to exclude any swelling or tumor or herpes zoster, and to determine the tender nerve-points. 2. The *muscle:* to exclude myalgia or pleurodynia. Examine for tenderness; note the effect of movement; does full breathing increase the pain? Palpate with the fingers and with the whole hand. Negative results exclude any muscular affection. 3. The *nerves.* (*a*) Tender points; (*b*) herpes; (*c*) the vasomotor appearance. The presence of anæmia, other neuroses and neurasthenic phenomena, or toxic conditions, as malaria, lead, or gout, lend color to the view that the pain is neuralgic. 4. The *pleura.* Auscultate for friction if pleuritis. Inquire for cough. Note the character and effect of breathing. 5. The *pericardium.* Note friction of pericarditis or thrill by palpation. Is the heart disturbed in function? 6. The *heart.* It is rare that disease of this organ causes pain, although it may be present in dilatation. Is it affected in a reflex manner, causing palpitation or irregularity? Look for distant disease. Angina or pseudo-angina pectoris may be present

7.[1] The *stomach* and *colon*. A dilated stomach or loaded colon may cause pain by pressure upward. Gastralgia may also be the cause. 8. The *spine*. Determine if it is diseased or if there is pressure by an aneurism or a mediastinal growth. If a local cause is not ascertained, look for a central or reflex disorder.

Although any one of the above conditions may cause pain in the side, it is usually (1) a reflex pain from gastric disorder; (2) pain from neuritis; (3) a true neuralgia from anæmia; (4) a neuralgia from heart-fatigue. (Hilton.)

It is to be observed that every local tissue must be examined, and questions asked as to the various attributes of the pain.

Pain in the Loins.

When acute, without fever, pain in the loins may be due to lumbago, to a sudden uterine retroversion, to a suddenly moved kidney, or to calculus of the kidney; with fever, acute Bright's disease, smallpox, muscular rheumatism, tonsillitis, influenza, dengue, or spinal meningitis must be looked for.

Chronic Pain in the Back; Backache. Backache may be due to many causes. When in the region of the kidneys, they may be at fault. Organic disease (Bright's) may be associated with backache; more frequently, pain, if in one kidney, is due to a calculus or to accumulation of uric-acid gravel. Pressure over the kidney or a sudden jar from a false step will usually excite the pain. It may be constant in moved or movable kidney. When low down, just above or over the sacrum, it is due to disturbance of the pelvic viscera. The uterus, the colon, and rectum (impacted, cancerous) must be examined

Otherwise we may have—(*a*) Pain due to affections of the *muscles*. 1. *Myalgia of rheumatic origin*. Increased by movement, by dampness, by pressure. Often relieved by warmth, by the recumbent posture, or rest. It is associated with symptoms of lithæmia and with the passage of red sand in the urine. When the fascia or the ligaments of the vertebræ are affected, the upright position and pressure in small areas increase the pain; other muscles may be affected alternately. 2. *Myalgia from sprain*. A history of injury is obtained. Usually one side is larger than the other. Tenderness is present and movement increases the pain. There may be increased swelling, vasomotor disturbance, or ecchymoses. A neurosis of the so-called spinal or traumatic

[1] Shoulder-tip pain, due to anastomosis of phrenic nerve with 3d and 4th cervical and to parts of liver and round ligament (Hilton); or of phrenic nerve and subclavius (Rolleston); or of vagus with spinal accessory, which communicates with 3d and 4th cervical. The v. and s. a. are sensitive to pressure. (Embleton.)

Inframammary pain (6th, 7th, and 8th intercostal spaces). The aorta at left side, 3d dorsal vertebra, is in relation with the 4th, 5th, and 6th intercostal nerves through the sympathetic ganglia, through which also the heart sympathetics are in anastomosis The 4th, 5th, and 6th intercostal nerves supply cutaneous branches to the 6th, 7th, and 8th intercostal spaces. The inframammary pain is a reflex neuralgia expressive of some heart-distress. The latter is brought about by exhaustion of the medullary and vasomotor centres. from worry or overwork, or from long-continued irritation of the uterine nerves. In leucorrhœa this pain is most common. (Jacobson: Hilton on "Rest and Pain.")

type (hysteria) attends the pain. 3. *Myalgia from fatigue.* Not only acute fatigue after exertion, but chronic muscle-tire (and nerve-tire). The pain is increased on exertion, after mental, physical, or *emotional* effort. *Neurasthenia, anæmia,* or local exhaustive disease (uterine, gastro-intestinal, etc.) are present. The muscles are usually flabby, and the vertebral column is not supported. The patient lounges or supports the back. Spinal curvatures are observed.

(*b*) Pain due to affections of the *nerves.* Nerve-pain is recognized by the tender points ; by vasomotor phenomena.

(*c*) Pain due to disease of the spine, the membranes, or the cord. (See above.)

CHAPTER V.

THE DATA OBTAINED BY OBSERVATION.

The objective symptoms correspond to phenomena in nature. *Method of procedure; method of the observer.* Inspection, palpation, percussion. The instruments required.

The Objective Symptoms.

The objective symptoms of disease are the most important to ascertain. They are the "handwriting on the wall." The impress of forces for good or evil is observed. In determining them we determine the physical, chemical, and vital condition of the organism; its state after the action of the forces of its environment. The physical and mental status of the patient is measured. He is individualized. The objective symptoms are data by which a complete diagnosis is made. Without such data the diagnosis is mere guesswork—one of probability. With such data alone, if accurately and precisely collected, a positive diagnosis can very frequently be made. A correct diagnosis depends upon the skill and thoroughness of the physician and his ability to interpret the data secured, always provided that clear, succinct data can be obtained

The data obtained by inquiry are carefully recorded, after which the following procedure is conducted. A physical examination of the patient is made, followed by an immediate study, or, if time permits, a study at leisure of the fluids of the body—microscopically, chemically, and bacteriologically. In the physical examination we make a general survey of the individual, and form an estimate of his height and weight. The various organs and tissues are interrogated by the senses applicable to the investigation of each, aided by special instruments. The natural secretions and discharges, abnormal discharges, all exudations or transudations, and cystic fluids are passed upon.

The student will soon learn that the process of ascertaining the objective signs of disease is in no respect different from that which obtains in the study of any object in nature or any like phenomena. The chemist notices the form, the color, the density, etc., of the object under examination; the effects of heat and cold, of various reagents upon its structure; he determines its component parts and ascertains its relation to other objects in nature. From data thus obtained by the use of all his senses he classifies the object. The biologist notes not only the physical appearance of a given form of life, but also the phenomena of the living, sentient matter under all conditions in a varied environment. By comparison and analysis the living being is classified.

By the same powers of observation and the same analytical process, the departures from health are recognized and classified. Is it not, therefore, a wonderful aid to the diagnostician to possess faculties which have been trained to minute observation by previous studies in sciences allied to medicine?

What has been thus imperfectly said is intended to emphasize the fact that no mystery attends the recognition of the objective signs of disease. Abundant opportunities of observing disease at the bedside, patient training, skill in technique, and a systematic procedure are essential.

Method of Procedure.

The method by which the data ascertained by observation are secured is modified by the circumstances under which the patient is seen. It is obvious that the patient who comes to the office, or is not sufficiently ill to be in bed, has sufficient strength to stand, and should be given an exhaustive examination. Moreover, we can inquire into certain abnormalities, as the gait, not visible in bed. On the other hand, in the case of a bed patient, we learn the position he assumes when lying down, and have better opportunities for thorough examination of the various organs. Often the objective examination must be very brief, on account of the patient's extreme illness. It may be advisable, although unfortunate, to exclude one or more methods, as percussion, if there is pain, or auscultation, if there is great restlessness or orthopnœa.

If a complete examination is made, it is well to begin with the exterior. After the *external examination* is made, the *internal examination* is conducted, by grouping together and examining organs functionally related, as the heart and bloodvessels, in diseases of the heart; the nose, larynx, and lungs, in diseases of the latter. The student will do well to begin at the head and take up the organs in their continuity.

Comparison. The results obtained by observation are based upon comparison; the student must bear this constantly in mind. We *compare* the body as a whole with our conception of the normal individual, formed by a study of a large number of persons. We compare symmetrical parts—the right side of the chest with the left, the arm suspected to be the seat of the disease with the healthy arm, etc. The cardinal rule in an examination is to base the significance of ascertained facts upon comparison with known normal conditions.

Methods of Observation.

Securing the Data. To accomplish these ends, examination is made by the sense of *sight* (*inspection*); by the sense of *touch* (*palpation*); by the sense of *hearing* (*auscultation*); and by the sense of hearing applied to the discrimination of sounds developed by *percussion*. By percussion or tapping the part we also elicit the peculiar phenomena known as *reflexes*.

The sense of *taste* is not used to determine the objective phenomena of disease. Some data, such as the odor of the exhalations and discharges, are obtained by the sense of *smell*.

Inspection. By inspection we judge of the physical condition of the whole or a part of the body, as seen in the shape and size and in the color; of the vital condition, by the expression of countenance, by the character of the movements of the body as a whole or in part, by the position in bed, and by the gait. The appearance of fluids (blood) and of discharges is also observed. The results of inspection as to size are confirmed by actual *weighing*.

In order that the data obtained by inspection may be complete and accurate, every portion of the body, and of its internal cavities which can be seen by the unaided or aided eye, should be inspected. The clothing should be removed, and, bearing in mind the proprieties, the whole body should be examined. For this purpose the patient should be under a good light. The light should always fall directly on the surface. The entire surface, of course, need not be exposed at once, and circumstances may be such that only one portion need be examined. Nevertheless, the fact must be insisted upon that patients who have been ill for a considerable time, as well as all grave cases, should be examined all over. It is even more important to do this if the patient is comatose. A node on the tibia, undue prominence of the vertebræ, a special rash about the anus, may afford information which could not be obtained in any other way. It is assumed that the patient has been examined lying down. In nervous diseases and diseases affecting the muscles and bones, the patient's gait, his ability to stand, the method of rising or assuming a sitting posture, and the performance of other customary physiological acts should be observed. For this purpose, as above mentioned, portions of the body can be covered, or a light gown thrown over the patient from head to foot.

Method of the Observer. In order to secure the data in full, the student should teach himself a method of observation by which all the facts are collated in regular systematic order. Whether the examination is general or local, whether the whole of the body is referred to or only a part, as, for instance, the nose, the student should accustom himself to make observations in the following order: First, the shape or contour (expression); second, the size; third, the color; fourth, the movability and the physiological condition of the part on movement. If this plan is pursued, little, if anything, will be overlooked. A similar order should be followed in the investigation of the character of the secretions and excretions of the body.

Inspection of Special Regions. In the inspection of special regions artificial light and special instruments are also required. The artificial light should be secured from an Argand or Welsbach burner, or from a gas-jet with a reflector, or from electricity. To facilitate the examination the room should be darkened and head-mirrors used as reflectors. A number of these have been devised, any one of which is suitable if it fits the head well and can be adjusted with comfort, so that the observer can throw the light on the part he wishes to examine, and, at the same time, peer through the centre of the mirror. A special arrangement of the patient and the light is required. The patient should sit in an easy, comfortable, erect position, with the light on a level with the part to be examined, a little behind, and to his right or

left, according to the convenience of the examiner. Special apparatus is required for the examination of each cavity: mirror, tongue-depressor, and specula for the throat, an ophthalmoscope for the eye, etc. (See respective sections.)

Palpation. The results of inspection are confirmed, when possible, by palpation, and the sense of touch supplies additional data. The nutrition of the parts is ascertained. The density, the resistance, the special character of the part, whether solid or liquid, are determined by this method of examination. On examination of the skin, the degree of dryness or moisture, the character of the skin, whether smooth or rough, the density of the part—as to degree of thickness and resistance—are all ascertained by means of the sense of touch. The presence or absence of pitting is observed, and the nature of swellings ascertained. In a similar manner local areas are examined. The same routine method should become habitual with the student. First, the shape and contour; second, the size; third, the color, its change on pressure, etc.; fourth, the movability of the part, and the character of the normal movements, as when a joint is under observation; fifth, the resistance and density of the part examined, or special characteristics revealed by touch—the elasticity of the skin, firmness of muscles, and, in swellings, the presence or absence of fluctuation. Other phenomena are detected, which are vital, in contrast to the above, which are physical. By palpation, alone or with instruments, we determine the sensibility of the part, the presence or absence of tenderness, the temperature, and the degree of moisture. In the examination of special regions by means of palpation some phenomena are determined peculiar to the system under examination, and dependent upon its physiological or functional action. Thus, in palpation of the chest, in addition to its movement, we note the vibrations transmitted to the hand when the patient is asked to speak, or detect abnormal vibrations from the friction of two rough surfaces together (pleura), or from the throwing of fluids into agitation: fremitus, friction, and râles are thus transmitted.

Knowledge of the action of the heart and of its position is obtained by palpation; thrills are detected, abnormal impulses felt. (For method of procedure, see respective organs.)

Auscultation. By auscultation we hear and analyze the sounds that attend respiration, the movements of the heart and of the blood in the bloodvessels. Abnormal sounds may be created in the pleura and pericardium—and in hollow viscera, as the œsophagus, stomach, and intestines—and their presence is likewise ascertained by auscultation. (See Diseases of the Lungs and Heart.) The character of the voice as to the quality and degree of loudness is studied to determine abnormalities in the respiratory tract or any speech defects of central or peripheral origin.

Percussion. By percussion, sounds are elicited which indicate the physical condition of the part percussed. In health the lungs and the

gastro-intestinal tract contain air in certain proportions, and therefore the sounds yielded by percussion are always of a known character. Any change from the normal sound is indicative of disease, of abnormal structure, or of alterations in the normal relations of the parts. Percussion determines these changes, and, in addition, enables us to estimate the size of organs. It is possible to determine the size of the liver, the heart, or the spleen, because of the relationship of these airless, non-resonant bodies to the air-containing structures around them. As this method of securing data is of the greatest use in pulmonary and abdominal diseases, the mode of procedure will be described in the chapters on Diseases of the Lungs and Abdomen.

Other Methods to Secure Data. In addition to the data obtained by the above methods, valuable and essential data are obtained by chemical, microscopical, and bacteriological examinations of the fluids, discharges, exudations, and transudations, and by aspiration and special examination of the fluids obtained from the natural cavities, or from cysts of the body. Bacteriological diagnosis and exploratory puncture will be considered in a special chapter.

CHAPTER VI.

THE DATA OBTAINED BY OBSERVATION—(*Continued*).

The *first sight* impressions. General abnormal vital conditions. *Fits* or *seizures*. *Coma*. *Collapse*. *Shock*. 1. The *personal appearance*. 2. The *apparent age*. 3. The *temperament* and *constitution*. 4. The *attitude* and *gait*. 5. The *general form* and *nutrition*. The *size*—enlargement, diminution. The *weight*.

GENERAL EXAMINATION OF THE EXTERIOR.

The general appearance of the patient affords an idea of the ability he has to cope with the antagonistic forces of his environment, or to overcome the deleterious effects of his occupation. It indicates the effect of present or past disease or of inherited disease. The *first sight*, striking impression, is always to be noted. "Very sick," "comatose," "collapsed," etc., or "robust," "cyanosed," etc., are speaking memoranda. To the experienced practitioner, the opinion formed at first glance is often of great diagnostic significance. It may happen that the patient is suffering from some unusually abnormal vital condition, a study of which must be made before the exhaustive survey of the case we are about to enter upon is conducted.

General Abnormal Vital Conditions. Impairment of consciousness and fits are readily recognized. The two often go hand-in-hand, but in some instances, as in fainting-fits, consciousness is not lost. The following list includes the various forms with their associate phenomena. Only those are mentioned which occur instantaneously. For their symptomatology and diagnosis the appropriate sections on special diagnosis must be consulted.

1. Unconsciousness. *a*. Syncope. The face is pale but calm, the pulse feeble or imperceptible, the extremities cool; nausea or hurried breathing may precede. The breathing is quiet in the attack. The pupils respond to light. No pain. (See Heart Disease.)

b. Cerebral Disease. (Spasm is sometimes associated.) Head-pain, congested face, hemiplegia, facial palsies, pupils irregular and irresponsive, cornea not sensitive, incontinence of urine.

c. Intoxications. Alcohol, opium, and other narcotics; uræmia, diabetes, toxæmia from infections, sunstroke.

2. Fits. *a*. Epilepsy. (1) "Haut mal:" aura, convulsions; (*a*) tonic, respiratory muscles affected, face livid, stupor afterward; (*b*) clonic, tongue bitten, stupor follows. (2) "Petit mal:" pallor sudden, no convulsions.

b. Infantile Convulsions. Usually reflex from indigestion; may be the onset of a specific fever or due to high temperature.

c. Puerperal Convulsions. Headache, amaurosis, œdema, suppressed and albuminous urine; clonic convulsions, tongue bitten, complete coma. (See Uræmia.)

d. Uræmia. Unilateral or bilateral clonic convulsions. (See Renal Disease.)

e. Alcoholism and Sunstroke.

f. Organic Brain Diseases (syphilis, tumor, softening, etc.).

g. Fits with Partial or no Loss of Consciousness. Hystero-epilepsy, focal or Jacksonian epilepsy, hysteria, cerebral embolism, thrombosis, or hemorrhage, spasms of various kinds.

h. Fits with Vertiginous Movement. The forms of vertigo are gastric, aural, and labyrinthine (Ménière's, also paroxysmal), ocular, cerebellar, from congestion of the brain (reflex), epileptic.

3. Collapse. Collapse may occur in a person in apparent health and be the first indication of disease, as in rupture of a large blood-vessel causing internal hemorrhage. Or it may occur in the course of disease, as typhoid fever, when intestinal hemorrhage takes place.

The symptoms are those of prostration, with partial loss of consciousness, or the mind is perfectly clear. The face is pale, pinched, and bathed with perspiration. (See Hippocratic Facies.) The skin is cool and clammy. The hands are cold. The skin is wrinkled. The eyes are sunken and encircled by dark rings. The voice is weak or suppressed. The pulse is rapid and thready, or may be absent at the wrists. The heart-sounds are indistinct. The temperature falls. The respiration may be hurried or shallow, sighing and gasping. The urine is scanty or may be absent. Collapse is due to hemorrhage, external or internal; to perforation of abdominal viscera; to peritonitis; to excessive watery discharge, as in cholera or serous purging. It may be due to pernicious malarial fever. Coma attends this form.

4. Shock is a condition in which the vital powers are blunted or stunned, with or without mental terror or anxiety. It is likely to be seen in injury, surgical operation, hemorrhage, angina pectoris, severe pain from any cause, any sudden cerebral or spinal lesion, undue mental and emotional strain. Its presence points to a grave antecedent condition, near or remote. The symptoms are those of collapse.

Location of Disease. A general view of the exterior will often indicate which system is the probable seat of the disease. For instance, violent respiratory action points to the lungs; paralysis, to the nervous system; the enlarged abdomen, to disease of the viscera in that region. The apparently hasty view has already given the practitioner much information.

We then note with more deliberation (1) the personal appearance; (2) the apparent age; (3) the temperament and constitution of the patient or the evidence of any diathesis or cachexia; (4) the position assumed in standing, walking, or in bed; (5) the general form and nutrition.

1. The Personal Appearance.

From the general appearance, the patient's habits as to industry, neatness, or care of dress may be observed; these habits are of diag-

nostic importance, particularly in brain affections. The appearance also shows frequently whether the patient is addicted to alcohol or to the use of narcotics. Moreover, the slit-boot, to relieve the swelling of gout, the loosely fastened boots from swollen ankles, the unduly worn sole as in spastic paralysis, the unbuttoned waist-band because of dropsy or increased weight, the stained trousers from drops of urine, are seeming trifles, but of diagnostic value.

The occupation of the patient is often important in throwing light upon his disease; the brown, weather-beaten face of the farm laborer, sailor, or driver contrasts strongly with that of the merchant, clergyman, or clerk. A machinist can often be recognized by his grimy, oily hands. All this information can be obtained at a glance, and many details can be added before the patient has taken his seat in the consulting-room.

2. The Apparent Age.

The apparent age of the patient should be estimated from his appearance, and compared with the *exact* age when this is learned later. In this way the physician will be able to judge whether the patient is aging too rapidly or bearing his age well. An obvious advantage of noting the patient's age is that it enables us at once to exclude a large number of diseases which are not found in the period of life to which the patient belongs. For example, if the patient is a child, we need not consider the chronic degenerations and the visceral cirrhoses which appear in middle and later life. Conversely, in an old person we do not expect to meet with the exanthemata which affect children almost exclusively. So, too, typhoid fever and pulmonary tuberculosis are more common in adolescence and early manhood than in childhood and old age. Again, in very young girls, the question of menstruation and its difficulties never have to be considered. *Gray hair* in a person under thirty-five generally indicates a feeble constitution and premature age. *Loss of hair* is not significant, for, apart from a tendency to baldness which is very marked in some families, professional men who do much brain-work, especially in hot, close rooms, are apt to become bald much sooner than other men. The presence of *wrinkles* at the corners of the eyes and of "crow's feet," and of dull, dry, lustreless eyebrows, should be noted as indicating aging, whether the person has lived long or not. In women approaching forty, who do not gain in flesh, there is often a suggestive prominence of the angles of the jaw and sternomastoid muscles, with a certain loss of roundness and elasticity of the cheeks. The latter appearance, however, may be due to loss of molar teeth.

3. The Temperament and Constitution of the Patient.

In former times emphasis was laid upon appearances which pointed to a particular diathesis or type of inherited constitution. Five varieties of diathesis were described to which general appearances pointed. They were the gouty or sanguine-arthritic, the strumous, the nervous, the bilious, and the lymphatic. While certain appearances point to the

occurrence of groups of individuals who may be classified under one of these diatheses, it is well not to lay too much stress upon them for diagnostic purposes. As pointed out by Gairdner, it is not proper to designate the diathesis off-hand. Individual appearances should be carefully noted, so that only after the completed examination a final conclusion as to the diathesis can be drawn.

In the *gouty* or *sanguine* diathesis the osseous system and muscles are well developed, the nutrition active, and the patient usually robust in appearance. The digestion is good, respirations deep, the circulation is well carried on (as shown by the florid skin and the large heart), the pulse is firm and steady, and the pressure in the arteries is high. The head is large and the jaw prominent, the teeth good. The hair is of strong growth. The individual with such diathesis is predisposed to the arterial changes of advancing age. Apoplexy, aneurism, and angina pectoris, or complications resulting from the senile changes in the heart and arteries, develop.

In the *strumous* diathesis the bones and the glandular system are changed and the appearance of the face is expressive; the bones of the chest are small; the long bones are slender, while their epiphyses are large; the forehead is broad and prominent, the lips full, the alæ nasi thick, the teeth are carious, the lower jaw light and thin, the hair is fine and often of a light hue, the eyelashes long, the eyebrows arched, often heavy. In this diathesis the nutritive changes are poor, inflammations are usually sluggish; disease of the bones, of the glands, and forms of tuberculosis are apt to be more severe.

In the *nervous* diathesis we see small, active, restless beings, with small bones and large muscles. They are full of energy, and carry on large business or mental operations. The features are well formed, the eyes active. Such types readily become the victims of overwork and of early breaking-down of the nervous system and of dyspepsia. They possess idiosyncrasies toward drugs, particularly opiates.

In persons of the *bilious* diathesis we find a dark skin, dark hair, muddy conjunctivæ. They are usually not well nourished. Their digestion is poor, and they are subject to attacks of so-called biliousness. Sick headaches are common. Fatigue is not borne well.

In the *lymphatic* diathesis there is lack of energy and sluggishness of nutritive processes; such persons are unable to keep up in the wear and tear of life. They are usually pallid and have soft muscles.

In addition to diathesis, *cachexiæ* are also noted. Cachexiæ arise from the ravages of disease, especially when the number of the red cells of the blood is reduced and the hæmoglobin diminished. Cachexiæ are caused especially by syphilis, gout, and chronic malarial poisoning. In cancer of some part of the digestive apparatus—and, indeed, in all forms of chronic disease of the digestive tract—a cachexia is seen. The anæmia from poisoning with lead, arsenic, and other metallic poisons produces an appearance to which the term cachexia has been applied, although in truth it only resembles one. Each form of cachexia takes its name from its cause, as the syphilitic or the cancerous cachexia.

4. The Attitude and Gait of the Patient.

The *attitude* of the patient gives information as to his physical vigor, and, to a certain extent, of his alertness of mind. A man vigorous of mind and body will stand firmly upon both feet, with back straight, shoulders square, and head erect. When one is depressed by care or disease the shoulders have a tendency to droop and the head to fall forward. Indecision and a vacillating disposition are sometimes indicated by the patient standing first on one foot and then upon the other while talking, or by an unsteady look from the eye.

When one shoulder is lower than the other and the patient is of phthisical build, pale, and emaciated, the attitude is strongly suggestive of phthisis or chronic pleurisy on the side on which the shoulder is depressed. Sometimes, in acute pleurisy, the patient will walk with the shoulder depressed and the arm firmly pressed against the affected side, so as to restrict its movements as much as possible.

Decubitus. The attitude of the patient in bed is often significant. He may assume the *active dorsal*, or the *side position*, with the body arranged so that it is comfortable and unconstrained. Then slight indisposition only is present. On the other hand, the *side* position, the *dorsal* position, or the *upright* or *semi-upright* position may be assumed.

To the close observer the attitude of a patient in bed is sometimes reassuring. He lies easily upon his back, or turned slightly to one side with the arms uncovered, and may even turn or sit up to meet the physician as he enters the room—all these signs point to moderate illness or to the approach of convalescence.

Side Position. A patient with acute pleurisy or pneumonia will lie on the affected side so as to limit its motion as much as possible. The breathing will be shallow and frequent, the expression of the face anxious, and occasionally a spasm of pain contracts it as the patient coughs or is obliged to take a full breath. He usually lies on the affected side because fixation is thus secured and pain on inspiration is diminished, and also because there is a greater liberty for expansion of the free, healthy side. If effusions are present, by lying on the side of the effusion pressure is removed from the heart and the unaffected lung, an obvious advantage.

At times, in case of thoracic aneurism, if situated on one side, or of movable thoracic tumors, the patient will lie on the side which is the seat of the disease.

The dorsal position, as assumed in health or slight disease, has been referred to. When the position is assumed in grave disease it is called *passive* dorsal, because it is often assumed without volition of the patient.

In grave cases of typhoid or other low fevers the patient lies upon the back and shows a marked tendency to slip down in the bed. The expression of the face is heavy or vacant. The lips and teeth require constant cleansing to keep them from sordes; the tongue is dry and glazed or covered with sordes; the tendons of the wrists twitch convulsively, and the patient lies with open or half-open eyes (coma vigil), picking at the bedclothes or at imaginary objects which float before his eyes.

A healthy baby a few months old finds motion an almost ceaseless delight. It will lie on its back, kick up its feet, play with its toes or some object that attracts it, crowing, wriggling, squirming. In *rickets*, on the contrary, the little patient lies as quiet as possible, even refraining from crying, because all motion is painful. In *cerebro-spinal meningitis* the head is drawn backward and downward and the muscles at the back of the neck are rigidly contracted.

In *acute disease* involving the peritoneum or neighboring organs, such as acute peritonitis, appendicitis, or endometritis, the patient lies on the back with the legs flexed upon the thighs and the thighs upon the abdomen. Motion is avoided as much as possible, and so is any pressure upon the abdomen.

The LATERAL or DORSAL POSITION, with legs drawn up and trunk and head drawn down to meet them, occurs with groans of pain and possibly involuntary bearing-down in hepatic and intestinal colic and during the throes of labor.

THE SEMI-UPRIGHT OR UPRIGHT SITTING POSITION. In an acute attack of *asthma* the patient is found sitting up in bed, or in a chair, possibly by an open window. The expression of the face is anxious, the skin dusky or pale, and moist. The breathing is loud, noisy, and scraping. The demand for oxygen is imperative, difficulty is experienced in inspiration and expiration, not enough air for physiological purposes being able to enter the alveoli; expiration is prolonged and labored (expiratory dyspnœa). The patient sits with the chin raised and head erect, the hands grasping the arms of a chair or the bedclothing, so that, by fixing the chest, the accessory muscles of respiration can be of the greatest assistance in supplementing the diaphragm. In emphysema, in its late stages, or when complicated with bronchitis and asthma, the same position is assumed almost constantly.

In *pericarditis* with effusion, in *large pleural effusions*, and in *advanced heart disease* with anasarca, the patient is unable to lie down on account of the smothering feeling which the recumbent position induces. In pericarditis the expression of the face is extremely anxious, the patient having a dread of impending death.

In large pleural effusion the expression is not usually so anxious, but the dyspnœa may be intense. The patient is propped up in bed, leaning slightly to the affected side, and devotes all his energies to breathing, avoiding every exertion, such as moving, answering questions, or coughing, which taxes his breathing-muscles still more. One side of his chest may be observed to move violently while the other is motionless.

In heart disease and anasarca dyspnœa frequently amounts to *orthopnœa*. The patient may be found propped up in bed or seated in a large rocking-chair, some patients finding greater comfort in the latter. The face is pale, livid, or jaundiced, and may be swollen, while the cellular tissue throughout the body is œdematous, and the cavities, especially the peritoneum, are more or less filled with fluid. In diaphragmatic pleurisy the position assumed is very characteristic—the erect sitting posture, with the body leaning forward and laterally, to relieve the pain.

The Prone Position. Rarely the patient is found lying upon the abdomen. He assumes this position because it gives relief to abdominal pain or to colic of any form. Owing to the change in the relative positions of the organs brought about by this posture, the pain of an ulcer of the stomach, of aneurism, or of caries of the vertebræ may be mitigated.

In tetanus *opisthotonos* occurs. The body rests on the head and heels and the trunk is arched upward, because of tonic contraction of the spinal muscles. In strychnine-poisoning with tonic convulsions the same position may be assumed.

Emprosthotonos, vaulted side position, is occasionally assumed in tetanus and also in strychnine-poisoning.

Unclassified Positions. Irregular or bizarre positions are usually assumed in affections of the nervous system, particularly in hysteria.

Restlessness. Often the patient is unable to assume a position, or, at least, to remain fixed in any position. This may occur on account of pain, or because of irritation or anæmia of the nerve-centres. In cases of moderate cerebral hemorrhage, and of shock, there is great restlessness. The patient is restless without the appearance of agitation. In profuse hemorrhage, whether uterine, intestinal, or pulmonary, on account of cerebral anæmia, there is also restlessness with sighing and gasping. The pallor, the quickened pulse, the great thirst, with the history of bleeding, are sufficient to explain the restless state. In *chorea* there is more than restlessness—there is constant twitching of muscles with jerking from one side of the body to the other. The patient does not keep the covers on when in bed, and by her jerky movements often does herself considerable injury.

In *cerebral meningitis* the patient tosses from side to side or lies with the head retracted and pressed deeply into the pillow. The eyes are injected, the pupils contracted, and frequent sharp cries are uttered, especially if the patient be a child.

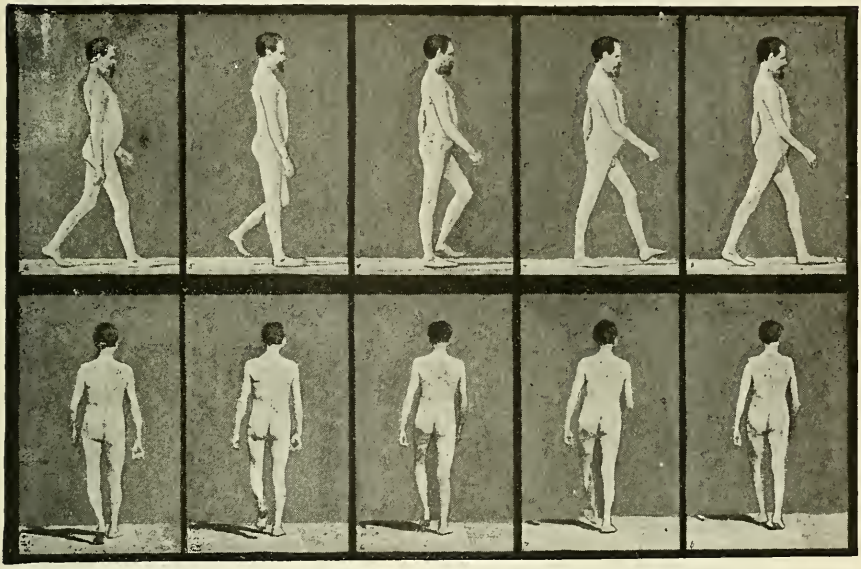
In *hysterical convulsions* the patient, usually a young woman, tosses wildly to and fro, screaming, laughing, or crying; or coma may be mimicked. The moods often change with great suddenness. The appearance is very alarming at first sight; but the pulse and breathing are not much accelerated, there is no fever, and the patient is conscious enough not to injure herself even to the extent of biting the tongue.

Gait. The gait is sometimes characteristic. (See Nervous Diseases.) The *hemiplegic* patient advances the sound limb, and then brings the other up to it by lifting the pelvis and swinging the paralyzed limb around by a movement of circumduction. The shoe is worn down at the toe in an irregular way. Sometimes the shoulder on the sound side is thrown outward and forward, so as to facilitate the raising of the pelvis on the paralyzed side in order that the limb may be circumducted. The arm may be rigid or bent at the elbow, the fingers being flexed upon the palm and the thumb turned in.

In *locomotor ataxia* there is uncertainty in the gait, which may only be felt by the patient or be apparent to the observer also. There is irregularity in the line of progression, or the movements become very jerky and erratic. As there is very little motion at the knee, because it is spasmodically braced, the pelvis is slightly tilted until the foot is

released; the foot is then raised unnecessarily high, jerked rapidly forward and outward and brought down with a sudden stamp, or flail-like action, on the heel. The patient's centre of gravity undergoes several changes at each step, so that he swings from side to side. He cannot walk in the dark, and, at a later stage, requires the aid of canes to prevent him from falling forward.

FIG. 2



Gait in a case of locomotor ataxia; instantaneous serial photographs. (MUYBRIDGE and DERCUM.)

In *paralysis agitans* the attitude and gait of the patient are peculiar. The head and body are thrown forward and fixed in that position; the arms are slightly abducted and partly flexed, the hands being in the position in which a pen is held or a pill rolled. The legs are also bent at the knees. Rhythmical tremors affect the hands first and then the rest of the body, the head and neck usually escaping. On attempting to walk the gait is *festinating*—that is to say, each step becomes more rapid than the preceding, until the patient is prevented from falling only by catching hold of something. The tremors cease during sleep, and are independent of voluntary motion. (See Fig. 3.)

In *spastic paraplegia* the patient walks with two sticks. He leans on the left one, arches the back, and then lifts the pelvis and the right limb as far from the ground as possible, but cannot quite clear it. The leg is rigid and the foot dragged around in a semi-circle. The toe has a marked tendency to stick to the ground, and is brought forward with a scraping sound. The knees have a tendency to interlock, and the foot which is brought forward is apt to cross in front of the other.

In *disseminated insular sclerosis* the gait is somewhat jerky and resembles the gait of ataxia or of tumor of the cerebellum. Of course, the

disease that causes such peculiarity in gait cannot be established without first observing the mental and nervous phenomena that attend such affections.

FIG. 3.

Side view of a case of paralysis agitans, showing forward inclination of trunk. Tendency to propulsion. (DERCUM.)

FIG. 4.

Spastic paraplegia, cross-legged progression. (DERCUM.)

In *hysterical paraplegia* there is sometimes complete loss of power of standing or of walking. The patient falls if an attempt is made to compel her to stand. Or she walks with the knees and the hips semi-flexed or in awkward attitudes, implying greater muscular exertion than necessary for the normal gait. It is recognized by the fact of its occurrence in young subjects in whom other striking phenomena of hysteria are observed. (See page 73.)

Cross-legged Progression. This form of gait is seen in children with spastic paraplegia, and occurs because of contracture in the calf muscles. When the child begins to walk, one foot gets over in front of the other. Sometimes a swinging oscillation of the body occurs, which may persist throughout adult life. (See Fig. 4.)

The gait of *pseudo-hypertrophic muscular paralysis* is known as the waddling gait. This oscillating character is assumed in order that the body be so inclined "as to bring the centre of gravity over each foot

on which the patient successively throws his weight, because the weak gluteus medius cannot counteract the inclination toward the leg that is off the ground, unless the balance is exact" (Gowers). The *position* assumed in getting up from the floor, as described by Gowers, is pathognomonic. The patient turns over in the all-fours position, raises the

FIG. 5.

Typical pseudo-muscular hypertrophy. (DERCUM.)

FIG. 6.

Hysterical astasia-abasia. (LLOYD.)

trunk with his arms, rests the trunk upon the extended hands, then extends the knees, pushes back with the hands until he can grasp one knee with the corresponding hand, then grasps the other knee, and pushes up the trunk by gradually raising the point of support for the hand upon the thigh. (Fig. 5).

The *swaying* gait, like that of a drunken man (cerebellar titubation), is significant of cerebellar disease. (See Station.)

Feebleness of the gait attends general paresis and the early stage of chronic myelitis, but, of course, is of no significance unless it is attended by other symptoms of these affections.

The gait of *paramyoclonus multiplex* and of *Thomsen's disease* is also peculiar. (See Muscles.)

Station. Astasia and abasia are terms employed to define the loss of power of standing and of walking, respectively, without paralysis. Both may occur. (See Fig. 6.) They are usually due to hysteria.

Ataxic Astasia in Locomotor Ataxia. The inability to stand is observed under many circumstances. Either with (1) the eyes closed, or (2) the eyes open and the toes and heels in contact, or (3) with the eyes open and feet apart. The latter occurs in the highest degree of ataxia, and may be followed later by complete loss of power of standing.

Swaying. If a healthy person stands with the eyes shut the body will sway slightly. In a patient with locomotor ataxia swaying is seen in increased degree.

In *pseudo-hypertrophic paralysis,* if the patient stands, there is that extreme curvature of the spine known as lordosis. It disappears entirely when the pelvis is supported, as in the sitting posture. In the latter stages of this affection there is posterior or lateral convexity of the spine with astasia.

In the paroxysms of *Ménière's disease* the loss of power of standing may be absolute. The patient may be hurled to the ground and be quite unable to rise or sit up. The nature of the paroxysm is suspected on account of the sudden onset and the complaint of vertigo, together with the ear symptoms that attend this affection.

In disease of the *middle lobe* of the *cerebellum,* swaying from side to side, or in large waves, is observed. The appearance is like that of a drunken person. While the walk is peculiar the patient can usually sit up.

5. General Form and Nutrition.

The general form and nutrition of the body are estimated by the color of the skin, the amount of subcutaneous fat, the degree of muscularity, the size and shape of the osseous system. Hence we estimate the degree of physical development of the individual by the size, the weight, and the condition of the muscles, as well as by the state of other tissues. To recognize lack of development is often to be able to explain phenomena of a functional nature which otherwise could not be accounted for. The color will be considered under the head of the condition of the skin.

Importance of such Observation. It is extremely important that these observations should be made, particularly in childhood and adolescence. Not only are marked departures from the normal significant, but slight deviations point to the occurrence of processes which modify nutrition. Unless lack of development is detected, it is frequently impossible to explain the occurrence of some functional disorder, as neuralgia, or of derangement of the viscera, or of indefinable ill health, as the result of which the patient shows inaptitude for exertion or inability to conduct the usual affairs of life. The recognition of malnutrition, as shown in lack of tone of muscles, or diminution of weight, is often sufficient to point the way to successful treatment by hygienic methods.

Size. Change in size may be *general* or *local*. General *increase* or *diminution* in size, not necessarily abnormal, is due to enlargement or diminution of the muscles and fat, singly or combined. When large accumulations of fat take place the word *obesity* is applied to the condition. The estimation of the patient's size as compared with his weight is usually based upon the amount of subcutaneous fat. The general accumulation can readily be recognized by rotundity of the exterior.

Size affords some information as to the degree of development of our patients and as to the kind of diseases to which they are most liable. While there is no absolute standard by which to compare the relative proportion of height to girth in individual cases, yet there is a type generally recognized as being usual, and variations from it give rise to such expressions as *stout*, *spare*, *slender*, *thin*, *tall*, and *short*. *Stout* usually expresses an increase in girth and a moderate excess of flesh over the normal. When used in this sense it becomes synonymous with lusty, and indicates an increase of flesh which is well distributed and due to healthy, active nutrition without impairment of physical activity. In some cases, especially in women, stoutness is used as a euphemism for corpulency, but not often for that excess of fat properly called obesity. Stoutness, in the sense of lustiness, up to middle life is an indication of physical and often of mental vigor. It is often found in gouty and rheumatic subjects. A tendency to take on flesh after the age of forty-five, especially if the person's occupation is sedentary and his habit of body inactive, is not to be regarded as favorable. It may be compared to a warrior's persisting in wearing an increasingly heavy weight of armor after the campaign is over. Increased weight under such circumstances is not increased strength, but increased burden, and the burden becomes greater with advancing years. Those who are under forty and stout, in the sense of having too much fat in proportion to bone and muscle, bear fevers and exhausting diseases badly. Women at the menopause are very prone to take on flesh rapidly. Fat subjects after middle life, and to an increasing degree after that period, are liable to fatty degeneration of the heart, blood-vessels, and important viscera.

Persons who are tall and thin, especially if they have become tall rapidly after puberty, are commonly looked upon as delicate, and as especially liable to consumption. There is reason for this view. But if they live to be twenty-five or more, without disease of the lungs or pleura, they may then live to a great age.

Some patients have an appearance which is well described and understood by the word "*spare*." The form is compactly put together, but with small bones and a scanty allowance of fat. There is a tendency to leanness rather than to roundness of form.

In still others muscle and bone predominate, and the form is apt to be angular, as in those described as *wiry*. They are often possessed of great muscular power and resistance to strain. Those of spare and wiry habit bear disease very well. Inspection alone may leave one in doubt whether to regard an individual as thin and delicate or spare. Light will be obtained from the patient's occupation and the amount of physical exertion of which he is capable, and also from the tonicity

and hardness of his muscles. If one stops to think a moment, he will see that, for the same amount of heart and lung capacity, a man will be better off if spare than if corpulent; because in the latter case he has an additional load to carry, and he has to nourish and keep up a thick blanket of fat from which he derives no adequate advantage. Hence a person of spare build, who survives childhood and adolescence without disease, probably has, on the whole, a better prospect for long life than a stout person.

Normal Habit. In estimating the patient's size or weight it is important to ascertain if he has a regular habit of taking on flesh at certain periods of the year, for instance, or if it has developed suddenly or followed acute disease.

Weight. Nothing has yet been said of the weight, but, as it affords a precise estimation of the size, particularly if considered in relation to the height and age, the following discussion will include the two points, size and weight.

While the eye can estimate approximately the weight of the body and the degree of emaciation, the physician should make it a rule to ascertain the weight accurately by means of scales. Machines are now made which can be used for weighing the patient and at the same time noting the exact height. It is particularly important to note the weight from time to time. In the course of wasting disease we learn the effects of treatment, or, on the other hand, the march of disease in spite of treatment. In obscure cases, as in tuberculosis, persistent loss of flesh is a serious diagnostic and prognostic symptom. After acute disease, if the patient is weighed every week, the onset of insidious sequelæ, as tuberculosis, may be detected.

The relation of body-weight to height is of importance. It is also important to know the average weight of the individual in different periods of life. The progressive increase in weight which should take place after birth should be remembered, as the opposite is positive evidence of malnutrition.

Mr. Hutchinson's table enables us to judge the average weight of a healthy man of a given height:

A man of	4	ft.	6	in.	to	5	ft.	0	in.	ought to weigh about	92.26	lbs.
"	"	5	"	0	"	5	"	1	"	"	"	115.52	"
"	"	5	"	2	"	5	"	3	"	"	"	127.86	"
"	"	5	"	4	"	5	"	5	"	"	"	139.17	"
"	"	5	"	6	"	5	"	7	"	"	"	144.29	"
"	"	5	"	8	"	5	"	9	"	"	"	157.76	"
"	"	5	"	10	"	5	"	11	"	"	"	170.86	"
"	"	5	"	11	"	6	"	0	"	"	"	177.25	"

In some life insurance tables in this country the average weight for the height is lower, especially in persons over five feet ten inches.

Weight in Disease. The question of *weight* is an important one in disease. As has been stated, persons with an excess of fat do not bear fevers and exhausting processes so well as those who have a relatively larger proportion of firm muscles. Remember, if emaciation is present, to ascertain its amount and degree, its possible relation to unusual mental care or to acute disease. Slow progressive emaciation

is of serious moment, as evidence of tuberculosis or disorder of assimilation. Remember the wasting that is associated with great hunger, excessive thirst, and polyuria in diabetes mellitus. On the other hand, such symptoms as occasional cough, slight evening fever, and impairment of resonance at one apex of the lung become much more significant of incipient phthisis if accompanied by loss of weight. At any stage of phthisis a maintenance of the body-weight is one of the most favorable elements in prognosis.

Again, while loss of weight attends all the diseases of the digestive tract which interfere seriously with nutrition, it progresses more rapidly and steadily, and attains a greater degree, in malignant disease than in the mechanical or functional diseases. Hence the question of loss of weight is important in deciding between chronic catarrhal gastritis and gastric carcinoma. But still more important is the question of the time during which loss of flesh has been taking place, and whether it has been progressive or interrupted by periods of gain in weight. If during two or three years the patient has been vomiting occasionally, and losing flesh, but gaining again from time to time, it is much more significant of gastric catarrh than of gastric cancer.

False Increase of Weight. In certain cases of great anasarca, and in malignant disease of the abdomen, especially huge cysts of the ovary in women, and sarcoma of the kidney in children, there may be actual increase of weight due to the accumulation of water or to the new growth, though the rest of the body is manifestly emaciated.

Weight in Children. In babies and children fat is more likely to be a sign of good health than in adults. Nevertheless the quality of the flesh is to be taken into consideration. There are fat and flabby babies and children, and there are others who are fat but whose flesh has a firm, solid feel. The former often gain and lose flesh rapidly, and, when ill, do not appear to have much resisting power. The size of a child gives a good idea of its nutrition. A child may have its growth stunted by bad food and unfavorable hygienic conditions, or the stunting may be the result of exhausting disease, such as whooping-cough.

Increase in size and weight then may be due to changes in (1) the skeleton (see Chapter XIII.); (2) the muscles; (3) the adipose tissue; (4) the subcutaneous connective tissue, giving rise to accumulations of serum, mucin, or connective tissue dystrophies (see Chapter X.). *Diminution* in size is due to changes in (1) the skeleton; (2) the muscles, and (3) the adipose tissue. The word *emaciation* is applied to excessive atrophy of fat and muscles. If it is accompanied by great exhaustion and apparent loss of fluid, the word *marasmus* is employed.

Degree of Loss. The whole body may exhibit considerable loss of flesh, the cheek bones and temporal fossæ being distinctly visible, the muscles soft, the limbs wasted, and the subcutaneous fat diminished. It is important to notice whether flesh has been lost or not, and how much, and how long a time the loss has been going on. Such facts furnish the clue not only to diagnosis but to treatment also. Flesh is lost in almost all diseases, acute or chronic, but it becomes of

special moment in diagnosis in the latter. It is most noticeable in tuberculosis, cancer, marasmus, cirrhosis of liver and kidneys, diabetes, in anæmias, and in cachectic conditions due to prolonged suppuration or chronic diarrhœa, in gastric neurasthenia and *anorexia nervosa*.

Local Change in Size. There may be local increase or diminution in size, alone or combined. It is not to be forgotten that accumulations of fat may take place in special portions of the body ; the abdomen is the favorite seat for excessive accumulation, particularly in women and in men of sedentary life, with habits of excessive indulgence in food and drink. When one part is increased in size and another growing progressively small the disparity indicates disease (see below). The face is swollen, especially under the eyes and above the jaws, in the dropsy of large white kidney and in parotitis. The neck may be enlarged in the sterno-clavicular notch or laterally above the clavicles in aneurism. The thyroid, as a whole, or either lobe, is enlarged in goitrous affections and in Graves's disease.

The face may be thin and even much emaciated, while the abdomen is greatly distended from dropsy or from tumors of the various abdominal viscera or glands. The chest is enlarged or contracted. Local decrease in size in thorax or abdomen is significant of tumors.

The head is much increased in size in chronic *hydrocephalus*, while the face remains small. The bones of the cranium are enlarged in *leontiasis ossea*. The head, face, and neck enlarge in the affection described by Allen Starr as *megalocephalie*. (See Chapter VII.)

The loss in flesh in the *extremities* or special muscles may be local and atrophic in character, as in some diseases of the nervous system, such as neuritis, infantile palsy, hemiplegia, and monoplegia. Loss of flesh of the arms is said to be a symptom in cystic ovarian tumor.

The increase in size may also be local, as in hydrocephalus, elephantiasis, dystrophies, myxœdema, œdema, and various tumors.

The General Musculature. The state of the muscles must always be learned. It has been referred to in the discussion on emaciation. A few words more seem necessary. It must be remembered that a person can be obese and yet have poor muscular development, or have little fat and fair muscle. General lack of muscular development or muscular weakness is an important sign of malnutrition, and may explain the nature of many symptoms. The muscular weakness can be approximated by the degree of firmness of the muscle. Weakness of the muscles of the spine, with resulting curvature, or inability to keep the erect posture, is sufficient cause for the occurrence of neuralgic pains in the course of related nerve-trunks, and for the displacement of organs within the thorax or abdomen, often causing functional disturbance. Various uterine displacements and functional disorders may be mitigated by toning up the nutrition of the muscles of the trunk. Forms of indigestion, sluggishness of secretions, particularly of the bowels, follow in the wake of debilitated muscles and pass away as such muscles gain tone. It may be that the indigestion has not taken place because the muscles are weak, although in a measure there is relation between them ; but the weak, flabby muscles are pronounced indications of a state of the system which may develop indigestion. More-

over, weakened abdominal walls, separated recti muscles, and *diastasis* favor dropping of the liver, stomach, and other organs, causing gastro-enteroptosis with its train of symptoms. The detection of muscular deficiency leads to correct lines of treatment. Atrophy of muscles occurs because of disuse, because of sedentary occupation or of a life of ease and luxury, with improper nutrition. It is sure to follow improper assimilation, as seen in extreme degree in *anorexia nervosa*.

CHAPTER VII.

THE DATA OBTAINED BY OBSERVATION—(*Continued*).

The *face*—the facial expression. The *head*. *Mumps*—facial hemiatrophy. Hydrocephalus. The *hair*. The *lips*. The *neck*—the thyroid gland—*exophthalmic goitre*—the bloodvessels of the neck.

The Face and its Expression.

The face is a mirror in which are reflected all degrees of ill health, from that which amounts only to temporary indisposition and depression up to the gravest cachexia. The face reflects also the degree of intelligence of the patient and his mental condition at the time, as well as his emotions, and, in a large measure, his character. The face is usually a fairly good index of the temper of the individual; benevolence, amiability, and purity are written as plainly on some faces as anger, lust, dishonesty on others. (See Nose and Mouth in respective chapters on special diagnosis.)

The face frequently affords us valuable information concerning the health, habits, and temperament of the individual. Everyone is familiar with the bright eye and animated countenance of a friend which lead us to say, "You are looking very well to-day," and with that slight pallor, diminished clearness of the conjunctiva, with perhaps a dark circle under each eye, which lead us to infer that he is depressed or has passed a sleepless night. The face also gives unmistakable evidence of alcoholism by its bloated appearance, injected or glassy eye, dull expression, and nervousness when the patient is addressed suddenly.

Full-blooded persons, disposed to endarterial changes, frequently as the result of gout, often have, at a little distance, the *ruddy* appearance of blooming health. Closer inspection, however, shows that the ruddy color is due to a dilated or congested condition of the minute bloodvessels. This condition, when associated with high tension in the arteries and accentuation of the aortic second sound, is highly suggestive of chronic nephritis. (For color and complexion, see the Skin, Chapter X.)

Moreover, the face tells of the presence or absence of pain, and, to a certain extent, of its character. Everyone has witnessed the sudden contraction of the brow and eyelids and the involuntary sucking in of the breath when some one has bitten upon a tender tooth. Other faces bear the imprint of long-continued more or less constant suffering. According to Eustace Smith, pain in the head in children is indicated by contraction of the brows; pain in the chest, by sharpness of the nostrils; and in the belly, by a drawing of the upper lip. (See the Face in Children and Pain, Chapter IV.)

It will be seen that the expression, the color, and the outline of the face are valuable indications of disease.

The master mind in clinical medicine, the late Austin Flint, Sr., tersely described the various appearances of the face in disease, with their clinical significance, as follows :

The Facies of Renal Disease. In some cases of acute albuminuria and of chronic parenchymatous nephritis—the large white kidney of Bright—puffiness of the face from œdema, with notable pallor, renders the aspect highly diagnostic.

The Malarial Facies. Pallor of the face, sallowness, and slight puffiness, if renal disease be excluded, point to malarial disease.

The Facies of Carcinoma. Notable anæmia, a waxy or straw-colored complexion, and more or less emaciation, in combination, render the aspect marked in some cases of malignant disease. In a patient over forty years of age this aspect has considerable diagnostic import, although it is by no means always present when malignant disease exists.

The Typhoid Facies. In the middle and later periods of typhoid fever the countenance is often dull, besotted, expressionless. This facies may be present in the typhoid state, which is incident to diseases other than typhoid fever —*e. g.*, pneumonia. Coexisting with a dusky hue of the skin and congestive redness of the conjunctiva, it distinguishes typhus as contrasted with typhoid fever.

The Facies of Acute Peritonitis. The upper lip raised so as to expose the front teeth gives an aspect which characterizes, in a certain proportion of cases, acute peritonitis. It is often wanting, but when present it is strongly diagnostic.

The Facies of Acute Pneumonia and Hectic Fever. Circumscribed redness of one or both of the cheeks, with abruptly defined borders, is diagnostic of acute pneumonia. If it be observed in a case of chronic pulmonary disease it denotes the so-called hectic fever, and is a sign of phthisis. The wan, emaciated appearance with the bright eye and hurriedly expanding nostrils excites our fears that the progress of the latter affection is most rapid.

The Facies of Exophthalmic Goitre. Projection of the eyeballs, giving to the face a remarkably staring and sometimes ferocious expression, conjoined with enlargement of the thyroid body and frequency of the pulse, is distinctive of the affection known as exophthalmic goitre—Graves's or Basedow's disease.

The Choleraic Facies. In the collapse stage of cholera the face is contracted, sometimes wrinkled ; the cheeks are hollow, the eyes sunken, the skin is livid, and the expression denotes indifference. This combination of traits is quite distinctive. They are, however, to a certain extent combined in the state of collapse which occurs in some cases of pernicious intermittent fever and in other pathological connections.

The Hippocratic Facies. This facies denotes the moribund state. The skin is pale, with a leaden or livid hue ; the eyes are sunken, the eyelids separated, and the cornea loses its transparency ; the nose is pinched and the eyes are contracted ; the temples are hollow and the lower jaw drops. Hippocrates described this facies in graphic terms, and the name Hippocratic has ever since been used to designate it.

The Face in Children. Inspection is even more important in the case of children than in adults. The pale, pinched, weazened face of some babies who have snuffles, ulcers, or striated lines at the corners of the mouth, and look prematurely aged, with prominent forehead and a depressed nasal bridge and retroussé tip, characterizes *inherited syphilis*. In older subjects the undeveloped face and skull are striking. In *rickets* the head is unusually large with flattened vertex, projecting forehead, and open fontanelle. In *hydrocephalus* the head becomes very much enlarged, the eyes prominent, the bones of the *face* remaining small, the expression vacant. In *adenoid disease* of the pharynx, with tonsillar hypertrophy, the dull apathetic expression, with the thickened lips, the small nasal orifices, and the gaping mouth are characteristic. In *cretins* the thickened lips, the protruded tongue with saliva dribbling from the open mouth, the flattened nose, with the idiotic expression and pallid, waxy skin, are easily recognized. To a lessened degree such appearances are seen in "backward" children, who, it may be said, are undeveloped cretins. In *measles* the red, swollen face, the reddened, weeping eyes, and running nose make a very striking picture. An irritating, excoriating discharge from the nose in a child may indicate the existence of a nasal *diphtheria*.

The Face in Nervous Disease. All varieties of mental aberration are reflected in the face; the suspicious, at times revengeful, look of the delusional monomaniac; the wild look and excited manner of the maniac; the plaintive, depressed, injured look of melancholia; the vacant, listless, peaceable, animal-like look of dementia—a look which changes to animation only at the sight of food or some coveted luxury. All these expressions come to be recognized very readily by those who see much of the insane. In addition, in hysteria expressions of varied emotions are seen; in neurasthenia a worn and wearied aspect of countenance is noticeable.

The face often tells of the existence of some organic nervous disorder. The peculiar heavy expression, drooping eyelids, though they close improperly, and sluggishly moving lips, betoken the early stage of the facio-humero-scapular type of *muscular atrophy*, and is sometimes seen in *Friedreich's ataxia*.

Change in the expression and appearance of the face more frequently occurs because of change in the function and nutrition of the muscles, on account of central or peripheral disease of the nervous system. On this account we have facial spasm or tremor, and unilateral, bilateral, or local facial paralysis. Further consideration of these conditions will be found in the local examination of the muscles (Chapter XII.) and in Diseases of the Nervous System.

In *peripheral facial palsy* the paralyzed side of the face has a staring, vacant expression, owing to the fact that the eyelid is motionless. The angle of the mouth on the affected side is depressed. The whole paralyzed side is devoid of wrinkles, has a smoothed-out, glazed appearance; tears flow over the cheeks and saliva dribbles from the corner of the mouth. The contrast with the normal side is most marked when the patient smiles or frowns. (See Fig. 7.)

In *glosso-labial palsy* there is progressive palsy, with tremulousness

of tongue and lips; progressive failure of articulation and dribbling of saliva. Sometimes the patient is able to open the lips but unable to close them without the aid of the hand. In *paralysis agitans* the mask-like expression of immobility has been described as Parkinson's mask.

A slow, hesitating, thick manner of speaking, with a tendency to slur the labial and lingual consonants, when associated with irregularity of the pupils, slight tremulousness of the lips, and the loss of the fine adjustment of other muscular movements, such as writing, is very suggestive of *general paralysis of the insane*, especially when the condition develops in a middle-aged man.

FACIAL HEMIATROPHY is a peculiar affection, characterized by progressive wasting of the bones and soft tissues of one side of the face. The disease is rare; it begins, as a rule, in childhood, but may develop in later life. The local change is diffuse; in some instances, however, it slowly spreads from a spot in the skin, involving, in succession, the tissues underneath. The skin changes in color and the hair falls out. The eye is sunken on the affected side, on account of wasting of the tissues of the orbit. The bone of the upper jaw atrophies to a more

FIG. 7.

Complete facial palsy. Patient unable to close eye of the affected side. (DERCUM.)

FIG. 8.

Facial hemiatrophy. (LYMAN.)

advanced degree than the other bones which undergo wasting. Because of the wasting of the alveolar processes the teeth become loose and fall out. The wasting is sharply limited by the middle line. (See Fig. 8.) The disorder is easily recognized. The patient looks as if the face were made up of two halves from different persons. It must not be mistaken for *facial asymmetry* that is associated with congenital wry-neck. The contraction of the sterno-mastoid muscle from birth distinguishes the affection.

The outline of the face and any change in the shape of the head should next be observed. Both changes, as seen in *myxœdema* and *scleroderma* (see Skin, Chapter X.), are described. The striking changes in *acromegalia*, *rickets*, and *osteitis deformans* are described in Chapter

XIII., on Bones and Joints. In *leprosy* the face is characteristic; the leonine countenance—*facies-leontina*—is the result of the tuberous outgrowths about the eyes and forehead.

Enlargement of the Face. Swelling. Other changes in the outline of the face and skull are significant. The face is swollen and deformed in erysipelas and smallpox, and, to a moderate degree, in measles. The specific eruption serves to distinguish each one. The puffiness of the eyelids and general swelling of the face in the course of Bright's disease will be referred to. (See Œdema.)

Œdema of the face occurs in *trichinosis*. It occurs at two periods in the course of the disease. It is seen in the eyelids in the beginning of the disease and disappears after a few days. Later it returns with pain, tension, and restriction of the movement of the eye-muscles.

Mumps. In *mumps* the swelling is characteristic. It usually begins on one side. The swelling of the parotid gland is observed in front of the ear, then it extends below and around it and behind the ramus of the jaw. Unless there is much collateral œdema the outline of the gland is preserved. The gland is tender and boggy, not indurated. Viewing the face from the front, the midlateral aspects are seen to bulge. The ears stand out from the head. The jaws are fixed. The submaxillary glands are usually enlarged.

The data to be considered in the study of an infectious disease are pointed out in the chapter devoted to those affections. In addition to such data the diagnostic features of mumps are the symptoms of the invasion of the general symptoms and the local signs.

The symptoms of the invasion are sudden, with chilliness, a rise in temperature, which is generally moderate (101° to 103°), and pain at the angle of the jaw. The corresponding parotid rapidly begins to swell, as well as the adjacent cellular tissue. Along with pain on movement of the jaws, any acid liquid, as vinegar, which stimulates salivary secretion, increases the pain. At times the submaxillary glands are involved instead of the parotids, or they may be enlarged and painful several days before the parotid is affected. The disease may be limited to one side or involve the opposite side, as the process in the one first attacked subsides. Rarely it is bilateral from the start. When the swelling has lasted from three to five days the fever subsides and the swelling begins to disappear rapidly. At this time, however, the opposite side may be attacked or the testicles become inflamed. Usually it is the right testicle. In girls and women the ovary or mamma is rarely inflamed. Resolution is extremely rapid, and usually the disease is not followed by sequelæ. Sometimes, however, deafness is left. In fact, sudden deafness sometimes announces the commencement of an attack.

If to these facts we add the data obtained in the social history, the age of the patient under fifteen, and the history of exposure or the presence of an epidemic, the diagnosis is easily made.

It must be borne in mind that *parotid swelling*, inflammation, with or without suppuration, may occur in the course of various infections, notably typhoid fever and septicæmia. It may also be traumatic. Chronic enlargement of the parotid occurs in syphilis. In some cases

(Osler and Kümmel) the submaxillary and lachrymal glands are conjointly enlarged with the parotid.

The Lips. Color. The lips are pale in anæmia, and livid in cyanosis from chronic lung or heart disease with feeble circulation. Vesicles (herpes) are apt to appear upon them in common colds, in certain febrile diseases, particularly pneumonia, and with many women during or immediately following menstruation. A child with hereditary syphilis may show ugly fissures, or the scars which result from them, at the angles of the mouth. In facial palsy the angle of the mouth on the paralyzed side is depressed and free from wrinkles. In glosso-labial-laryngeal palsy the lips tremble, twitch, and may have to be closed with the fingers after they have been opened. In general paralysis of the insane the lips tremble, and speech is "thick," hesitating, and uncertain, with a tendency to elide syllables and slur the labial consonants.

Hair. The hair often indicates the state of the nutrition of the individual. Changes in it may be significant of syphilis or other internal morbid processes. The abnormal growths and changes in the texture due to local parasitic disease will not be referred to. Undue and rapid falling out of the hair in patches, known as alopecia, is indicative of syphilis and of profound intoxication by the virus of this disease. The hair can be pulled out in large masses without difficulty or pain. This falling of the hair must not be confounded with the excessive falling out which takes place in the convalescence of acute disease, particularly of typhoid fever, nor with that following an attack of gout or erysipelas.

Color of the Hair. Obscure paralysis or anæmia may be explained by noting if the hair is artificially colored. Lead and other poisonings have repeatedly arisen from the use of hair-dyes. Other changes in the color are often significant. Early gray hair may go hand-in-hand with premature endarteritis. The term "canities" is applied to the diminished development of pigment. Premature gray color in defined patches occurs in nerve-lesions, as paralysis of one of the branches of the fifth pair, and is a trophic change. Sudden change in the color of the hair, usually to gray, takes place at times under the influence of fright, mental anxiety, or deep emotion.

"Green" hair is seen in brass-founders and workers in copper-mines; "blue" hair in laborers in cobalt-mines and persons employed in the manufacture of indigo. Chemicals applied to the hair change its color —peroxide of hydrogen bleaches the hair, pyrogallic acid turns it black. Drugs administered internally, as jaborandi and its alkaloid, change the color to dark hues.

The Head.

The *posture* of the head and abnormal *movements* are due to affections of the muscles of the neck, and will be considered in a study of local affections of muscles. (See Chapter XII.)

Enlargement. Change in the size and shape of the head is seen in rickets, acromegalia, and otitis deformans, along with other skeletal changes, and are discussed in the chapter on the Bones and Joints.

Enlargement is due, however, to local hypertrophy of the bones, to hypertrophy of the soft tissues (myxœdema and leprosy), and to enlargement of the contents of the cranium. Enlargement of the bones is seen in *leontiasis ossea*. In osseous hypertrophy the bones are thickened. Gowers states such thickenings may simulate hydrocephalus at any age. He thinks it doubtful whether the nature of osseous hypertrophy can be ascertained during life.

Enlargement due to increase of cranial contents is seen in hydrocephalus.

Hydrocephalus. The enlargement of the skull is very conspicuous, and the disproportion of the cranium to the face is striking. The cranium is rounded or globular in shape, and the fontanelles are seen

FIG. 9.

Congenital hydrocephalus. Female, aged seventeen. (The thinness of the hair could not be represented.

to be very large, tense, and bulging, and the sutures widely separated. The disproportion in size between the face and head is increased by the projection of the anterior portion of the skull. The axis of the eyes is directed downward, and they are partly covered by the eyelids, because of the oblique direction of the orbital plates. The head is supported with difficulty. The eyeballs roll from side to side. There is frequently strabismus. The skin is stretched tightly over the cranium, and the hair is scanty. (See Fig. 9.)

Diminution in the size of the head is seen in microcephalus (circumference less than seventeen inches). It is usually abnormal in shape.

Fontanelles. After a consideration of the size and shape of the head we turn our attention to an examination of the fontanelles and the bones of the head. The fontanelles in a healthy child, with the exception of the anterior, close in the early weeks of life. The anterior close from the sixteenth to the twentieth month. We note whether they are open or closed, prominent or depressed. New openings or fontanelles and loose bone plates, the normal fonanelles remaining open, are seen in so-called *craniotabes*—a condition found in congenital syphilis and rarely in rhachitis.

Prominence or *fulness* may be temporary or permanent. When the former, a passing fever with cerebral congestion may be the cause; when the latter, hydrocephalus and other brain affections in which there is increase of internal pressure. *Depression* of the fontanelles occurs in general atrophy, marasmus, and in wasting diseases generally. It is present in collapse, and is of grave prognostic omen. In pneumonia and other respiratory affections with dyspnœa, retraction is observed. The former affection, with cerebral symptoms, is thus distinguished from cerebral meningitis in which the fontanelles bulge. The fontanelles are neither prominent nor depressed in *rickets*, a point of distinction between this affection and hydrocephalus or enlargement from other internal causes. They may remain open, moreover, long after the usual period of closure in rhachitis, even to the third or fourth year.

The Bones. The *bones* of the cranium may be thickened; they may be the seat of periostitis, of necrosis, and caries. Necrosis and caries of the frontal bone are almost pathognomonic of syphilis. Necrosis of the jaw bone belongs to phosphorus-poisoning. The mastoid and petrous portions of the temporal bone should be examined in many affections. The symptoms that should call our attention to these bones are pain and tenderness over the mastoid, rigors, and fever, with the symptoms of thrombosis of the cerebral sinuses, pain in the head, convulsions, and strabismus. Examination in this region should extend to the *occipito-atlantal articulation*. Disease of this articulation, and particularly tubercular disease, causes stiffness of the neck or falling forward of the head. On account of the stiffness, associated with difficulty of deglutition and pain, the writer has seen it mistaken for retro-pharyngeal abscess.

Auscultation and *percussion*. We have thus far limited our examination of the head to inspection and palpation. Auscultation has been practised, and at one time it was thought the continuous murmur heard over the vertex in children was due to intracranial disease. Osler, however, pointed out its occurrence in healthy children, hence, unless heard in adults, its presence is not of diagnostic significance. McEwen, of Glasgow, has found that in cerebral abscess and tumor and also in meningitis, secondary to ear disease, a difference in the percussion-note was found over the affected area, and at the same time the percussion resistance was increased. The site of disease was indicated by a note higher in pitch than the usual osteal note. Comparison of the two sides must be made.

The Neck.

The position and movements of the larynx and trachea, the thyroid gland, the lymphatic glands, and the vessels of the neck should be observed.

The *larynx* and *trachea* occupy the median line in health, but may be deflected to the right or left. The deflection is more readily noticed at the lower part of the neck, and can be ascertained by comparing its position with the normal relation to the adjacent muscles. The change in position is due to disease within the thorax. An aneurism or a mediastinal tumor may cause this alteration. In cases of chronic fibroid phthisis the trachea is pulled to the side of the affected lung. When the respiratory movement of the larynx and trachea is excessive and associated with dyspnœa the source of the dyspnœa is of laryngeal origin. When, on the other hand, the movements are lessened, or the organs remain fixed, notwithstanding violent efforts at respiration, the dyspnœa is due to disease in the mediastinum, as enlargement of the mediastinal glands, or aneurism pressing upon a bronchus. *Tracheal tugging* may be seen, but is usually determined by palpation. It is particularly characteristic of aneurism of the descending portion of the aorta. The aneurismal sac presses upon the bronchus, and, with each pulsation of the vessel, tugs or pulls downward upon the trachea, which tugging is transmitted to the hand. (See Diseases of the Vessels.)

Thyroid Gland. It may be enlarged or atrophied. *Atrophy* is shown by absence of fulness, which would otherwise be present. (See Myxœdema and Acromegalia.)

Enlargement of the thyroid can be detected without much difficulty. It may be limited to one lobe, or both lobes may be affected. It may vary in size from a small localized swelling to large masses which fill the median and lateral sides of the neck, pressing upon the trachea and extending into the thorax. On palpation the swelling may be soft or hard. In the fibrous forms the swelling is not very large and is very much indurated. In the cystic forms of the thyroid enlargement fluctuation may often be detected; it may be localized to a small area of the lobe, or may be detected over the entire affected lobe. In some cases, on palpation, a purring or thrill is transmitted to the fingers. The thrill is synchronous with the heart's action and due to increased vascularity of the gland. Auscultation under these circumstances reveals a systolic murmur.

Causes. Enlargement of the thyroid gland may be due to simple hypertrophy, to fibro-cystic enlargement, or to enlargement in which the vascularity is more prominent, as in exophthalmic goitre. 1. In simple *hypertrophy* the enlargement is often intermittent, increasing in size at each menstrual period, or coming on in pregnancy, to disappear after labor. It may then disappear entirely or return at the menopause. 2. The *fibro-cystic* enlargement which occurs in countries in endemic form is persistent. 3. The enlargement of exophthalmia generally continues throughout the course of the disease. (See below.)

Exophthalmic Goitre. Exophthalmic goitre, Graves's or Basedow's disease, is far more frequent in women than in men. It may develop

at any age, but is most common in early adult life. A neurotic heredity, exhausting disease, general debility, and anæmia are predisposing causes, while sudden fright or shock is the most common exciting cause. Graves's disease begins slowly.

The data just recorded are those of the social and family history, and with the objective symptoms to be described complete the picture of this affection.

Of the three classic symptoms, *rapidity of the heart's action*, with palpitation, *enlargement of the thyroid*, and *prominence of the eyes* (exophthalmos), the first is the essential symptom. It is also usually the earliest. Either enlargement of the thyroid or exophthalmos may be absent for months or years, and in some instances throughout the disease.

1. Tachycardia. Attacks of palpitation may recur at intervals for a long time before their true nature is suspected. In these attacks the behavior of the heart is much like that which occurs under the influence of fright or great excitement. The frequency may not be over 100 or 120 in the early attacks, the rate being normal in the intervals. In the later and severe attacks, however, the pulse beats 160 or 180 or even 200. It is small and regular. The heart beats with increased force; the sounds are loud, sharp, and clear, occasionally being heard several feet from the patient. In time the heart becomes hypertrophied and dilated, and there is often a loud, basic, systolic murmur.

The larger arteries and even sometimes the smaller ones show the vascular disturbance by increased pulsation, sometimes with thrill.

2. The Thyroid Gland. The *thyroid* is usually the next to become affected. It enlarges slowly from vascular dilatation, the swelling at first subsiding in the intervals between attacks, but subsequently persisting. The right lobe may be larger than the left. The enlargement is painless, soft, and compressible. It may pulsate with or without thrill, and over it can be heard hæmic murmurs.

3. The Eyes. Prominence of the *eyes* is the most conspicuous feature of well-marked cases. Like enlargement of the thyroid, it varies in degree, and rarely is wholly absent. The protrusion allows the white sclerotic to show above and below the cornea, giving the eyes an unnatural, startled, staring appearance. The protrusion may be so great that the eyelids cannot close; more commonly they close, but when the eyeball is simply directed downward the upper eyelids do not follow but remain spasmodically elevated or lag behind the movement of the eyeball (Von Graefe's sign). The eyeball may become inflamed and even slough from undue exposure. In rare instances one eyeball alone is affected, and in these cases the lobe of the thyroid of the opposite side is enlarged. Stelwag's sign (widening of the palpebral fissures) is the third ocular sign of significance in exophthalmic goitre. Finally, Möbius calls attention to the frequency of insufficiency of the internal recti muscles.

In addition to these characteristic symptoms loss of *flesh* and *strength*, moderate pyrexia of irregular type, impaired appetite, diarrhœa, and despondency are observed. The *diarrhœa* is of the nervous type—increased peristalsis without local catarrh. Menstruation is apt to be irregular or to cease. Tinnitus aurium, headache, and vertigo are not

uncommon, and sometimes there is profuse *sweating*. A restless, nervous excitement (Charcot) is very common. *Muscular tremor* (Marie), occurring on voluntary movement, is frequently observed, and, with diarrhœa, is almost as common as the three primary symptoms. Œdema of the feet is often seen if there is coexisting mitral disease. Transitory vasomotor œdema of the eyelids, the face, hands, and the supraclavicular and infraclavicular regions occurs. It is usually circumscribed, and may not pit on pressure.

FIG. 10.

Exophthalmic goitre.

Graves's disease, as a rule, runs a chronic course, lasting for years. A few cases that have run an acute course of a few weeks, some ending in recovery and some in death, have, however, been reported. Moreover, there may be recurring attacks with apparent recovery in the intervals.

Death results from gradual weakening of the heart and its direct and indirect effects. It may be hastened also by uncontrollable diarrhœa, acute mania, and epilepsy. The disease may also be complicated with hemorrhages, and these may be the immediate cause of death.

Enlargement of the thyroid gland from the above-mentioned causes must be distinguished from enlargement due to abscess, cancer, sarcoma, or adenoma. Abscess usually follows infectious diseases; in the writer's case it followed typhoid fever. With carcinoma and sarcoma there is anæmia and gradual loss of flesh. It must also be distinguished from other tumors in this region. It particularly must not be confounded with enlargement on the right side due to an innominate aneurism. (See Aneurism.)

The Vessels of the Neck. Changes take place in the arteries and veins, observed by inspection, palpation, and auscultation. (For a description of these changes, see Arteries and Veins.)

The Lymphatic Glands. (See Chapter XI.)

CHAPTER VIII.

THE DATA OBTAINED BY OBSERVATION—(*Continued*).

THE EYE AND EAR.

The Eye. Indirectly the eye and the skin are the external structures that present the most evidence of disease in other organs. This is true of the eye, because of the comparative ease of its examination, and because it is a highly specialized organ, bearing close relationship to the vascular and nervous system. Its special functions are subservient to the highest physiological cerebral action; hence any perturbation of or organic change in the cerebrum is expressed in altered eye function, either of movement or of vision. Its nerve and vascular connection with the brain render it sensitive to internal change. In diseases of the nervous system the eye is the one organ the examination of which is essential to make a diagnosis. Constant reference in the chapter on Nervous Disease will be made to this section, and the converse holds that in the study of this section reference must be made to the nervous system. But diseases of the heart, the kidneys and systemic conditions, such as gout, rheumatism, diabetes, etc., find expression often in some eye change.

Much may be gained from an inspection of the eye and its adnexa regarding the state of the general system. This is at once evident when we reflect that of the twelve pairs of cranial nerves four pairs are devoted solely to this important organ, while in the eye itself we have unfolded to our gaze a living nerve-head, the optic papilla, and the retinal vessels, which offer to our view the perfect cycle of the supply of an organ with arterial and the escape of its venous blood. Moreover, the eye presents in compact form representation of nearly all the tissues of the body.

In order to insure that nothing shall escape scrutiny in the inspection of the eye, it is necessary to follow some settled plan of investigation, and for this purpose it is well to pursue an anatomical order, proceeding from the superficial to the deeper structures.

The Lids. Œdema is not an infrequent symptom of renal disease (see Œdema of the Face), and may occur in cases of profound anæmia and chlorosis; it may indicate the prolonged use of arsenic, or it may be originated by disease of the orbit or some of the periorbital sinuses of the same side. The dropsy may accumulate during the night and be seen in the morning on rising. Morning puffiness is natural to some individuals. Both it and the swollen face following a debauch are not to be confounded with œdema.

Ptosis, or drooping of the eyelid, may be congenital, more usually it is a symptom of disease within the brain. (See Paralysis of the Third Nerve.)

Lagophthalmus is that condition in which the lids can be closed but imperfectly, and follows paralysis of the orbicularis muscle, due to lesions of the portio dura. According to Bull and Hansen, paralysis of the orbicularis muscle is of common occurrence in leprosy.

Blepharospasm, or active closure of the lids from spasm, is of a reflex nature, originated by excitation of a filament of the fifth nerve. It is always present to a greater or less degree in *photophobia* or intolerance of light; this latter symptom is a frequent associate of ocular disorders, and is also found in certain stages of meningitis, cerebral tumors, typhus, measles, etc. It accompanies many forms of headache, especially migraine, and it may be the expression of a hyperæsthesia of the retina in nervous subjects, apart from any actual inflammation of the retina. Cramp of the orbicularis muscle has been noted quite often as a symptom of hysteria. *Nictitation*, or undue winking of the eyelids, occurs not infrequently in children as part of a habit of chorea.

Styes or small boils which form on the tarsal margin, and blepharitis or inflammation of the margin of the lids, while often due to an error of refraction, may denote some defect in the general health, such as anæmia or scrofula.

Vaccinal eruption may appear on the eyelids, occurring as a small ulcer with an indurated border and yellow floor at the commissures, and is usually attended by some swelling of the lids and face and by enlargement of the preauricular glands.

Chancre may appear either as a primary or secondary sore, and is generally situated in the conjunctiva lining the lids.

Malignant pustule, or specific anthrax, is seen at times, though rarely, on the lids of those who are exposed to infection from diseased animals or decayed animal matter.

Xanthelasma consists in the formation of small, irregular, opaque, yellowish patches, slightly elevated above the surrounding skin. These areas may either remain localized or the disease may involve the palms of the hands, the flexures of the fingers, and the inside of the mouth. (See Tongue.)

The Orbit. Exophthalmus, or proptosis, abnormal prominence or protrusion of the eyeball, is usually occasioned by some disease of the orbit or of the neighboring sinuses which encroaches upon the cavity of the orbit. It is one of the diagnostic features of exophthalmic goitre (see Exophthalmic Goitre), and may also be caused by paralysis of the ocular muscles. It has been seen, though rarely, after spontaneous hemorrhages into the orbit in cases of hæmophilia and scurvy.

Enophthalmus, or retraction of the eyeball, may be the result of exhausting diseases, such as peritonitis, or secondary to some orbital lesion. It is very pronounced in the sudden atrophy that occurs in cholera from loss of water.

Extraocular Muscles. Before detailing briefly the measures employed for the detection of paralysis of the extraocular muscles, and that the subject may be grasped more readily, a few words of explanation will be given regarding the anatomy and physiology of the muscles engaged in the ocular movements.

The eyeball is suspended in the orbital cavity by means of six muscles—the four recti, superior, inferior, internal, and external, and the superior and inferior oblique. Of these the four recti and the superior oblique have their origin at the apex of the orbit, while the inferior oblique rises from its lower inner wall. These muscles exercise their action upon the movements of the globes in three pairs, each pair being composed of two antagonistic muscles; the rectus internus and externus; the rectus superior and inferior, and the superior and inferior obliques. The sixth nerve supplies the external rectus, the fourth the superior oblique, the remaining four muscles receiving their impulses from the third nerve.

When all of the muscles are in a state of equal tension and the visual axes are directed straightforward in the horizontal plane, the eyes are then said to be in *the primary position*. Any deviation from this is known as *a secondary position*, the simplest of these being direct lateral or vertical movements. Thus, the rotation of the eye directly inward is accomplished by the rectus internus, outward by the rectus externus, upward by the superior rectus and the inferior oblique, and downward by the inferior rectus and the superior oblique. Oblique movements of the eyeball, however, are more complicated and necessitate the action of a third muscle to regulate the torsion which the eye undergoes when it is moved from the perpendicular. This is occasioned by the fact that while the plane of the points of origin and insertion of the rectus externus and internus corresponds with the horizontal plane of the eyeball, that of the rectus superior and inferior and of the oblique muscles do not correspond with the vertical and horizontal planes respectively. Therefore, so soon as the globe is moved into the oblique position, it rotates or undergoes a certaion amount of torsion. Thus, the superior rectus, in addition to elevating the eye, rotates the upper part of the cornea toward the nose, while the inferior rectus, in direct antagonism to it, depresses the eye and rotates the upper half of the cornea externally; these muscles exercising their greatest degree of torsion when the eyeball is turned inward and either upward or downward. The superior oblique depresses the eye and rotates the upper part of the cornea internally, while the inferior oblique elevates the eye and rotates the upper half of the cornea externally. The obliques, in antagonism to the superior and inferior recti muscles, exercise their maximum amount of torsion, therefore, when the eye is rotated externally and either upward or downward.

It appears from the foregoing that in inward and downward motion the rectus internus and inferior and the superior oblique will be brought into play; in outward and upward, the rectus externus and superior and the inferior oblique; and in outward and downward movements the rectus externus and inferior and the superior oblique.

Manner of Detecting Palsies of the Extraocular Muscles. Normally, the movements of both eyeballs are in perfect association and harmony, so that the images of objects fall upon corresponding points of both retinæ, and single vision obtains. If this harmonious action be interrupted by paralysis of one or more of the extraocular

muscles, however, then this no longer happens, and limitation in the movement and deviation of the affected eye is the result, coupled with double vision or diplopia.

Limitation in the Movements and Deviation of the Affected Eye. In studying limitations of motion in the eyes, the examiner seats himself before the patient and requests the latter to follow with his eyes the movements of a candle which is carried through all the different meridians of the visual fields, any muscular deviation being made evident by a failure in correspondence of the images from the candle reflected from the cornea, as well as by the lagging in the movements of the eye in the deviation of the action of the affected muscle. There are three general laws which have been formulated which should be borne in mind in this connection. 1. The limitation in motion as well as the diplopia increases toward the side of the affected muscle. 2. The secondary deviation (the deviation which the sound eye makes while the affected eye is fixing the candle) is greater than the primary deviation (the deviation of the affected eye while the sound eye fixes). 3. The image formed on the retina of the affected eye is projected in the direction of the paralyzed muscle.

Diplopia. The character of the diplopia varies according to the muscle or muscles whose function has been disturbed. Generally speaking, diplopia is either simple or homonymous, or crossed or heteronymous. In the former the image of the affected eye lies on the corresponding side and betokens convergence of the visual axes, while in the latter the image of the affected eye is projected to the opposite side and indicates divergence of the visual axes. In order to ascertain the relation of the two images to the respective eyes, it is essential that the diplopia should be carefully tested.

Test for Diplopia. For this purpose the patient is seated in a darkened room with a red glass placed before one of the eyes, in order to facilitate the identification of each image by its color, and a lighted candle is held on a level with the head about five metres off. Having noticed any deviation which the eyes make in the primary position, upon a chart especially constructed for this purpose, the candle is moved through the different meridians of the visual field, the patient being requested to regard the flame with both eyes while the head remains quiet, each deviation being carefully noted on the chart.

After the deviations have been recorded the diagnosis of the affected muscle or group of muscles will be much facilitated by the following rules: If the diplopia be lateral, then the paralysis is either of the rectus internus or externus. If, in addition, the images are crossed, then the internus is at fault, but if they are homonymous the externus is paralyzed. If the diplopia be vertical, and in the upper field, then the paralysis is either of the rectus superior or the oblique inferior. If the images be crossed, paralysis of the superior rectus is indicated, but if they be homonymous, implication of the inferior oblique is designated. If the diplopia be vertical and in the lower field, then the paralysis is either of the rectus inferior or obliquus superior—crossed images indicating paralysis of the rectus and homonymous that of the oblique muscle.

Additional Symptoms. In addition to the study of the anomalies in motion and of the diplopia, considerable information may also often be gained by noting the position of the head in ocular paralyses. Thus, in paralysis of the sixth nerve, the face is turned toward the paralyzed side; in paralysis of the fourth nerve, it is turned downward and toward the shoulder of the paralyzed side; and in paralysis of the third nerve, the face looks toward the shoulder of the same side. Not rarely dizziness is complained of and false projection of the field of vision, causing patients to make faulty estimation of distance.

The Clinical Significance of Disturbances in the Motility of the Extraocular Muscles. In addition to the significance which paralysis of the eye muscles bears to lesions of the brain and of the cranial nerves, and which will be dwelt upon at length later, diplopia may proceed from some much less serious disturbance, as, for example, derangements of the digestive organs or alcoholic intoxicants. Transient attacks of diplopia may be among the earliest symptoms of tabes dorsalis, and may occur at the very beginning of cerebral meningitis.

Monocular diplopia is a rare symptom, and when it can be dissociated from some local disturbance in the media of the eye, may be attributed to hysteria.

Ocular deviations or *paralytic squint*, as has just been described, must be differentiated from *concomitant squint* or *strabismus*. In this latter variety there is no great restriction in movements of the eyes in any direction, the faulty position of the visual axis remaining constant while the eyes are moved from side to side, and the secondary deviation being equal to the primary. This is the condition which is commonly known as cast or cross-eye, and usually makes its appearance in children with high degrees of far-sightedness.

Nystagmus is a spasmodic condition of the muscles of the eye, producing rapid oscillations of the ball, usually horizontal, sometimes rotary and rarely vertical. It is of great value as a symptom, being found in many brain lesions, usually those of the restiform bodies, the vermiform process, and of the cerebellum. It is also seen in Friedreich's ataxia, in miners, and often as the result of visual defects.

Muscular Insufficiencies. Of late years much attention has been given by ophthalmologists and neurologists to the study of *errors in the extraocular muscle balance* in different reflex psychoses. While the assertion which has been made by some, that chorea and even epilepsy may be originated by such deviations, is extreme, it is nevertheless quite true that many forms of headache, of vertigo, of nausea, and of vague neuralgic pain of a cephalgic type can be traced to this source. It is important, therefore, that the clinician should be acquainted with such errors, and should be familiar with the methods employed for their detection.

The device of Maddox is usually employed for this purpose. This consists of a glass cylinder which is fitted into a linear opening, which is made in a metallic disk. The patient is seated before a candle flame five metres off and requested to regard the flame with both eyes. The rod is then placed before one of the eyes perpendicularly and an image

of a perpendicular streak of light obtained from that eye. If the streak of light be deviated toward the same side as the eye before which it is held, a condition of excessive convergence or *esophoria* is present; but if the streak deviates toward the opposite side, then a divergence of the visual axes or *exophoria* exists. If the streak be on a higher or lower level than the flame, vertical imbalance or *hyperphoria* is present. Balance of the muscles is known as *orthophoria*.

The Conjunctiva. The conjunctiva being a transparent though vascular membrane, any changes in the amount or the constitution of the blood will at once evidence itself in its folds. Thus, in anæmia there is always a pallor of the conjunctival vessels, while in plethora there is usually a passive dilatation of the vessels which gives the eye an injected appearance, and occasions the "bloated eye" of the drunkard. In jaundice the conjunctiva is yellow. Spontaneous hemorrhages into the membrane are seen in whooping-cough, asthma, epilepsy, and in calcareous degeneration of the bloodvessels, and it may be the seat of hemorrhagic infarcts in ulcerative endocarditis.

Inflammation of the conjunctiva is an early symptom in measles, and in typhus fever it is a constant sign, and serves to distinguish this affection from typhoid. It is also present in yellow fever, and may likewise constitute one of the earliest signs of meningeal and cerebral diseases. A passive hyperæmia follows disease of the cervical sympathetic.

The Cornea. The cornea being an avascular membrane, deriving its nourishment from the surrounding structures, it is very prone to undergo inflammation whenever the vitality of the system becomes much lowered, and as a result of this inflammation opacities remain which have a very deleterious action upon vision. These opacities may be either superficial or interstitial. When superficial they are not infrequently the result of burns, traumatisms, and extension of the inflammation from the surrounding conjunctiva; in many cases they denote, however, that the eye has been the seat of a phlyctenular inflammation, a form of ocular disease which is quite common in scrofulous children and in individuals below par.

Superficial ulceration of the cornea is observed also in all fevers of a typhoid type, when the patient lies in a semi-conscious state with the lids but partly closed. Dust and bacteria gather between the lids, and as the patient winks but seldom a crust forms on the cornea, which is followed by extensive ulceration. Abscesses of the cornea form in the stage of desquamation of variola, and must be differentiated from those which arise in the pustular variety of the disease at an earlier period. Ulcers also form in the seventh week of typhoid, being usually coincident with abscesses in the scalp and skin of the back.

The type of interstitial opacities of the cornea is seen in inherited syphilis. Indeed, to the trained eye, the appearance of the haze in this class of cases is so characteristic that the diagnosis of the systemic affection might be made for the eye alone. Malaria and scrofula may also produce similar types of corneal inflammation. The small areas of opacity which form in the upper and lower parts of the cornea near the limbus, and which at times encircle the cornea, are known as arcus senilis. This is commonly supposed to be indicative of arterial sclero-

sis, although the author has never found ground to warrant this assertion. It may always be diagnosed from a somewhat similar opacity of inflammatory origin by the fact that in the latter variety, the opacity being due to an inflammation usually beginning at the corneo-scleral margin, the haze is continuous with the conjunction of the two membranes; whilst in arcus senilis there is a zone of clear corneal tissue between the margin of the cornea and the rim of the opacity.

After lesions of the fifth nerve the cornea may ulcerate from traumatic and trophic causes, and after paralysis of the seventh nerve it may suffer from exposure due to inability to close the lids.

Iris. Inflammation of the iris is a common symptom of secondary syphilis; it occurs under the form of a gummatous infiltration of the membrane in the tertiary variety, and is seen, though rarely, in inherited syphilis. It is not an infrequent symptom of chronic rheumatism and gout, and may be caused by tuberculosis and rheumatoid arthritis.

The Pupil. The pupil may react either directly or indirectly to *light stimulus*. In order to observe this, the patient is seated before a window and requested to gaze at the sky. The examiner, stationed in front of the patient with his back to the window, excludes one eye by placing his hand over it, and notes the size of the pupil under diffuse daylight. The eye is then covered with the other hand, and the dilatation which should follow is also approximated. The hand is then withdrawn, and, if nothing prevents, the iris will contract to the same size as that which existed at the commencement of the test. The fellow eye is then to be tried in a similar manner. This is known as the *direct reflex action* of the pupil; *indirect* or *consensual reflex action* being the contraction or dilatation which occurs in the shaded eye when the exposed eye is being examined, and should correspond precisely with the movements of the pupil of that eye.

Having noted the reaction of the irides to light stimulus, the patient is now directed to transfer his gaze to the examiner's finger, which should be made to slowly approach the eye, whilst its fellow is screened off as in the former test. The amount of the contraction induced by this *accommodative effort* is carefully noted, and the same procedure repeated in the fellow eye. The obstructing hand is finally removed, and the patient being requested to look fixedly at the tip of the surgeon's finger with both eyes, observation is made of the contraction of the pupil, which should be induced by the *effort at convergence* which is occasioned by approximating the finger to the eyes in the median line.

Hippus is a spasmodic alternating contraction and dilatation of the pupil, which is seen at times in mania, hysteria, and other allied disorders. Rhythmical alterations in the size of the pupils occur frequently in the so-called Cheyne-Stokes respiration; the pupil contracting during the period of apnœa and dilating with the first few breaths.

Modification in the Size and Behavior of the Pupils as the Result of Disease. Pupillary reaction to light is a reflex phenomenon, the optic nerve being the afferent nerve, and the third nerve the efferent nerve, supplying the sphincter of the iris; communicating fibres between the corpora quadrigemina and the centre from the third

nerve making such a reflex possible. The mechanism of pupillary reaction being of an extremely complicated nature, and necessitating the activity of a number of nerves and nuclei, it is not strange that anomalies in its behavior should be frequently met with in disorders of the central nervous system.

Dilatation of the pupil (*mydriasis*), apart from local diseases, of which glaucoma is the type, may be produced by certain psychical emotions, such as fright and emotion, or it may be caused by diseased processes giving rise to irritation of the pupil—dilating centre or fibres (irritative or spasmodic mydriasis), or by paralysis of the pupil—contracting centre or fibres (paralytic mydriasis or iridoplegia).

Irritation mydriasis occurs (*a*) in hyperæmia of the cervical portion of the spinal cord and in spinal meningitis; (*b*) in the early stages of new growths in the cervical portion of the cord; (*c*) in cases of intracranial tumor and other diseases causing high intracranial pressure, according to Raehlmann, although Leeser points out that these may also give rise to paralytic mydriasis; (*d*) in the spinal irritation of chlorotic or anæmic people, after severe illness, etc.; (*e*) as a premonitory sign of tabes dorsalis; (*f*) in cases of intestinal worms, owing to the stimulation of the sensitive nerves of the bowel, and sometimes in other forms of intestinal irritation; (*g*) in psychical excitement—*e. g.*, acute mania, melancholia, progressive paralysis of the insane (often, then, unilateral, with myosis in the other eye). (After Swanzy.)

Paralytic mydriasis (*iridoplegia*) may be due either to a paralysis of the pupil contracting centre or as a result of the stimulus not being conducted from the retina to that centre. It may be found under the former circumstances: (*a*) Sometimes in progressive paralysis where at first there was myosis; (*b*) in various diseased processes at the base of the brain affecting the centre of the third nerve; (*c*) in a late stage of thrombosis of the cavernous sinus; (*d*) in orbital processes which cause pressure on the ciliary nerves. (After Swanzy.)

It is said to be present in acute dementia, when there is œdema of the cortex, and is found in cerebral softening. It occurs in irritation of the cervical sympathetic and occasionally in aortic insufficiency.

Contraction of the Pupil (*Myosis*). Having excluded myosis from local causes, especially from the sequelæ of iritis, it will be found that contraction of the pupil may be caused by a disease process irritating the pupil-contracting centre or nerve-fibres (the irritative myosis of Leeser), or by one causing paralysis of the pupil-dilating centre or nerve-fibres (the paralytic myosis of Leeser), or by a combination of both.

Irritation myosis is found in (*a*) the early stages at least of all inflammatory affection of the brain and its meninges, in simple, tubercular, and cerebro-spinal meningitis. When, in these diseases, the medium myosis gives place to mydriasis, the change is a serious prognostic sign, indicating the stage of depression with paralysis of the third nerve; (*b*) in cerebral apoplexy the pupil is at first contracted, according to Berthold, who points out that this contraction is a diagnostic sign between apoplexy and embolism, in which latter the pupil is unaltered; (*c*) in the early stages of intracranial tumors situated at

the origin of the third nerve or in its course ; (*d*) at the beginning of a hysterical or of an epileptic attack ; (*e*) in tobacco amblyopia, probably from stimulation of the pupil-contracting centre by the nicotine ; (*f*) in persons following certain trades, as the result of long maintained effort of accommodation (watchmakers, jewelers, etc.), the pupil-contracting centre being subject to an almost constant stimulus ; (*g*) as a reflex action in ciliary neurosis ; consequently, in many diseased conditions of those parts of the eye supplied by the fifth nerve. (After Swanzy.)

Paralytic myosis occurs in spinal lesions above the dorsal vertebra—*e. g.*, injuries and inflammations, especially of the chronic form. The contracted pupil occurring in gray degeneration of the posterior columns of the spinal cord has been long known as spinal myosis. In the simple form of this myosis the pupil has but a medium contraction, and reacts both to light and on convergence. This condition is found in the early stages alone, when the disease has attacked merely the cilio-spinal centre, or higher up, as far as the medulla oblongata ; later on, when Meynert's fibres become engaged, we have the Argyll-Robertson pupil. The very minute pupil often seen in tabes dorsalis is probably due to secondary contraction of the sphincter pupillæ.

Paralytic myosis is also found in general paralysis of the insane. In acute mania the pupil is usually much dilated, and when this mydriasis is changed for myosis approaching general paralysis may be prognosticated. Myosis, following on irritation mydriasis, is also found in myelitis of the cervical portion of the cord. In bulbar paralysis, if paralytic myosis occurs, the disease is probably complicated with progressive muscular atrophy, or with sclerosis of the brain and spinal cord. Myosis may also be due to paralysis of the cervical sympathetic, resulting from injury, from pressure of an aneurism of the carotid, innominate, or aorta, or from pressure of enlarged lymphatic glands. In apoplexy of the pons varolii myosis is present, but it is not yet certain whether it is an irritation myosis or a paralytic myosis.

Inequality of the pupils may denote lesion of the third nerve, affection of the cervical sympathetic in the cervical region of the spinal cord, general paralysis of the insane, or some unilateral lesion of the brain.

The Lens. *Cataract.* An opacity in the crystalline lens should always awaken the suspicion of its being due to diabetes, as cataract is of not infrequent occurrence in this disease. Although renal disease also has been held accountable by some for the occurrence of cataract, no satisfactory evidence has been given to prove this assertion.

The Eye Ground. In order to study the remaining structures of the eye, it is necessary to have recourse to *the ophthalmoscope.* The essential part of this instrument consists in a concave mirror, whereby the light from a lamp which is placed back and slightly to the side of the patient's head may be projected into the interior of the eye about to be examined. This mirror is provided with a small central aperture, through which the examiner looks and studies the details of the back of the eye or fundus oculi, as it is technically called. When the instrument is held close to the eye, and the eye-ground studied without the intermediation of other means, the procedure is known as the direct

method of ophthalmoscopical examination. In the indirect method, on the other hand, the ophthalmoscope is held about sixteen inches from the eye and an inverted image of the fundus obtained by means of a convex lens, which is interposed between the ophthalmoscope and the eye, and serves to collect the rays of light into a focus between the lens and the eye of the examiner. The former method possesses the advantage of magnifying the interior of the eye about fourteen times, while the indirect, although of less magnifying power, permits of the examination of a greater part of the fundus at a glance.

The ophthalmoscope, in addition to giving us information in regard to the condition of the media of the eye, as, for example, of the existence of commencing cataracts, or of opacities within the vitreous humor, unfolds to our gaze the head of the optic nerve as well as the retina and the choroid, and renders patent to our view the different diseases to which they are liable.[1]

Retinitis. The systemic affection which is accompanied by a lesion of the retina more often than any other is disease of the kidneys, especially chronic interstitial nephritis. Indeed, about 30 per cent. of all cases of this variety of renal lesion have an ocular manifestation. Retinitis may also be seen as an early symptom in the nephritis of scarlet fever and pregnancy. Its occurrence in the cirrhotic kidney is of gloomy import, for patients with a retinal complication in this disease usually die within two years of its first appearance. Retinitis may also be occasioned by pernicious anæmia, leukæmia, diabetes, syphilis, and heart disease.

Choroiditis is usually the result of syphilis, but may in rare instances be the seat of tubercles. Gout may also originate a subacute inflammation of the membrane.

Optic Neuritis. The optic nerve being really a prolongation of the brain, and being, of a consequence, so often liable to be affected in cerebral disorders, it is of the utmost importance that the clinician should be able to recognize changes in its appearance. Indeed, it is safe to say that the study of a "nervous case," so called, is never complete without the report of the ophthalmoscopic findings.

Papillitis, or choked disk, an inflammation of the head of the optic nerve, is rarely idiopathic, but is occasioned by cerebral growths and by meningitis, especially of the base of the brain, and by the same constitutional diseases which originate retinitis. It also occurs in acute fevers, and it may be the result of suppression of the menstruation. Usually, however, choked disk is the result of an intracranial tumor, occurring in 90 per cent. of all such cases, and as it is an early sign, its detection has frequently been the means of the discovery of many intracranial neoplasms. As a rule, tumors of the cerebellum and those of the cerebrum which interfere with the circulation in the lymph passages of the brain originate it, the size and the character of the tumor not seemingly influencing its production.

The variety of optic neuritis which has just been discussed is an

[1] It has not been thought proper in a work of this kind to give further details regarding ophthalmoscopy, the student being referred to special text-books upon ophthalmology for a perusal of that important subject.

ascending neuritis, the inflammation beginning at the intraocular termination of the nerve and spreading upward from this to the brain. There is also an interstitial or descending neuritis which is commonly caused by meningitis. Retrobulbar or toxic neuritis is a variety of inflammation of the optic nerve where the disease confines itself to the bundle of nerve-fibres which go to supply the macular regions. This disease is commonly caused by alcohol and tobacco, although it may be originated by quinine, the salicylates, lead, and iodoform. It may also be caused by rheumatism and catching cold, and there is a rare form where the disease is transmitted through certain families from generation to generation.

Optic Atrophy. This may be secondary to some inflammation of the optic nerve or retina, or it may be a primary disease.

Secondary or consecutive atrophy is usually the result of optic neuritis; it may, however, be originated by local causes either within the eye or the orbit. *Primary atrophy*, on the other hand, though occasionally idiopathic, is generally found associated with some disease of the spinal cord, especially with locomotor ataxia. In this affection it is frequently an early sign, and it has been noted by Benedikt, of Vienna, that when this occurs it is rare for a tabetic patient to become ataxic. It has also been remarked that cases in which blindness is well advanced suffer but little from the pains which are characteristic of this disease. Simple atrophy occurs also in lateral and insular sclerosis, and is frequently seen in general paralysis of the insane.

Before proceeding further with the consideration of the cerebral expansion of the optic nerve, it becomes necessary to study the methods which are used in the determination of the visual acuity, both central and peripheral, as these are valuable and often necessary adjuncts in establishing the diagnosis of many obscure cases of cerebral disease.

Central vision is tested by means of black letters printed on a white test card, those devised by Snellen being usually employed on account of the admirable system upon which they are founded. The patient is seated five metres away from the card, and one eye being blindfolded he is requested to read the lowest line of letters which he can distinguish. If the vision fails to correspond to the standard, it is necessary to exclude hypermetropia, myopia, and astigmatism by means of convex, concave, and cylindrical lenses before it can be definitely asserted that the vision is lowered as the result of disease.

Peripheral vision, or the extent of space of which the eye is conscious when it is fixed on any given point, may be estimated in several ways; it is accomplished, however, most accurately by means of the perimeter. This is an instrument which consists of an upright rest for the chin and a semi-circular arc or bar, graded in degrees, which revolves upon a middle point, and is capable of describing a hemisphere in space. The eye under examination being directed straight ahead at the fixation point, the fellow eye being blindfolded, the test object, a small square of white paper, is brought from the periphery toward fixation. The patient is then asked to indicate the instant the object is perceived, and the examiner marks the degree upon a chart provided for the purpose. If the perimeter be not at hand, the field

may be obtained fairly accurately as follows: The patient is seated opposite the surgeon with one eye bandaged. He is then directed to look at the corresponding eye of the examiner whilst the observer's finger is slowly brought in from the periphery toward the eye through the different meridians. In this way the surgeon can ascertain whether

Fig. 11.

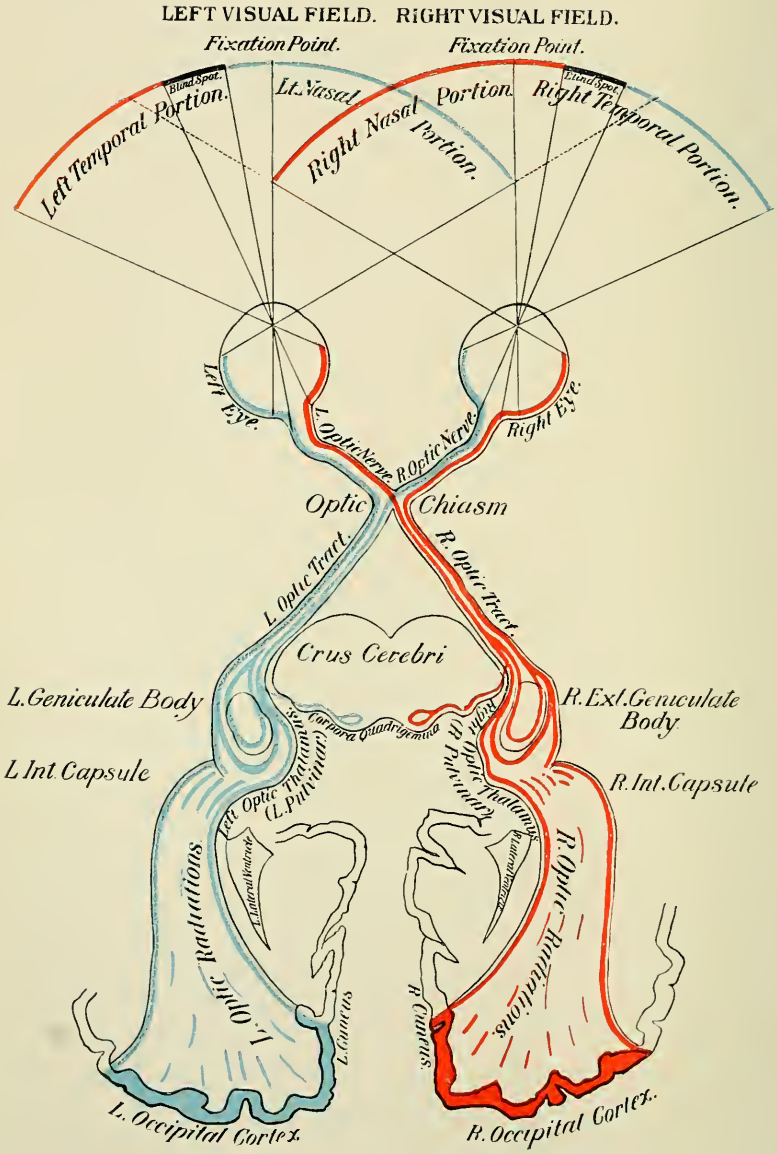
The McHardy perimeter.

the patient permits his eye to wander from the fixation point, and at the same time he can compare the extent of the patient's field with that of his own. The field for form or white extends over 150° horizontally and 110° vertically, that of the different colors falling within this in the following order—yellow, blue, red, and green.

Scotoma. As the patient's macula corresponds to the fixation point in the visual field, the physiological blind spot which is occasioned by the entrance of the optic nerve into the eye will be found in the temporal portion of the field. Pathological blind spots are known as scotoma, and these may be either central, paracentral, or disseminated. When central, they indicate either a disease of the macula or of the fibres of the optic nerves supplying the macula, so that a central scotoma is one of the diagnostic features of retrobulbar neuritis.

Hemianopsia. This term is used to imply a defect in one-half the field of vision, the defect being named according to the blind area. Thus, temporal hemianopsia means that the eye cannot perceive objects when situated in the outer half of the field. The most common form

PLATE I.


LEFT VISUAL FIELD.
RIGHT VISUAL FIELD.
Fixation Point.
Fixation Point.
Blind Spot.
Blind Spot.
Left Temporal Portion.
Lt. Nasal Portion.
Right Nasal Portion.
Right Temporal Portion.
Left Eye.
L. Optic Nerve.
R. Optic Nerve.
Right Eye.
Optic Chiasm
L. Optic Tract.
R. Optic Tract.
Crus Cerebri
L. Geniculate Body
R. Ext. Geniculate Body
L. Int. Capsule
R. Int. Capsule
Tubercula Quadrigemina
Left Optic Thalamus (L. Pulvinar)
Right Optic Thalamus (R. Pulvinar)
L. Optic Radiations
R. Optic Radiations
L. Calcarina
R. Calcarina
L. Occipital Cortex.
R. Occipital Cortex.

of hemianopsia is the loss of the temporal field in one eye and of the nasal field in the other, this condition being known as lateral homonymous hemianopsia. If the temporal portions of both fields are lost, the defect is known as bitemporal hemianopsia; binasal hemianopsia, indicating a loss in the nasal fields of both eyes. Superior and inferior hemianopsia are very rare.

It is often possible by studying the changes in the visual fields to locate quite definitely the seat of the cerebral lesion. By a reference to the diagram (Fig. 12) it will be at once evident that *a lesion of the*

FIG. 12.

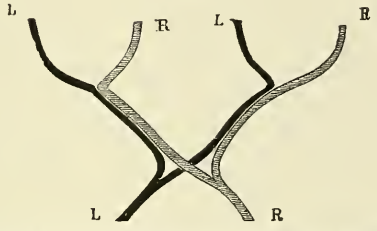


Diagram showing the course of the optic fibres in the chiasm. (HIRT.)

chiasm would necessarily comprise the crossed fibres of the optic nerve, and would occasion bitemporal hemianopsia. Such a lesion may be due to basilar meningitis, periostitis, hyperostitis, fracture of the body of the sphenoid, distentions of the infundibulum, and of the third ventricle, or to tumors, especially those of the pituitary body, and finally syphilitic gumma. If due to the latter cause, there may be transient recurrent attacks of the hemianopsia. Bitemporal hemianopsia is also an early symptom of acromegalia. The lesion in superior and inferior hemianopsia is usually in the chiasm also, affecting its superior or inferior portions; these defects in the fields may, however, be caused by symmetrical cortical lesions and by optic neuritis. (See Plate I.)

If the lesion affects the outer angle of the chiasm, then monocular nasal hemianopsia is the result.

LESIONS OF THE TRACT AND CENTRES. As shown in Plate I., the optic tract after crossing the crus to the hinder part of the optic thalamus divides into two branches, one going to the thalamus and the external geniculate bodies and to the anterior quadrigeminal bodies from which fibres pass into the hinder part of the internal capsule, and entering the occipital lobe, form the fibres of the optic radiations terminating in the cuneus, the perceptive visual centres; while the fibres of the other branch pass to the internal geniculate bodies and the posterior quadrigeminal bodies.

A lesion affecting the optic fibres anywhere posterior to the optic chiasm will produce lateral hemianopsia, so that this symptom of itself is of little value in localizations. There are, however, certain accessory symptoms which, when taken in conjunction with it, will often serve to establish the seat of the lesion in most instances. Thus, in hemianopsia from lesions of the optic tract there is an absence of the symptoms which occur when the cortex is affected—as mind-blindness, word-

blindness, etc.—while other symptoms indicating a basal lesion are apt to be present, as, for example, implication of the cranial nerves, especially those supplying the ocular muscles. Lesions of the optic tract are also frequently associated with a disease of the crus cerebri, so that hemianæsthesia or hemiplegia of the opposite side of the body would be associated with the hemianopsia. There is, however, a sign which enables us at once to say definitely whether the lesion be in the optic tract or not, and this is known as the *Wernicke or pupillary inaction sign.* This is elicited as follows: The patient is seated in a darkened room with one eye blindfolded, and is directed to look straight ahead into the darkness. The eye being slightly illuminated by an assistant by means of the diffuse light from a plane mirror, which is reflected into the eye from a light placed behind the patient's head, the examiner slowly throws a small beam of concentrated light from a concave mirror upon the blind half of the retina. If the pupil fails to react, the lesion is then in the geniculate bodies or in the tract, inasmuch as the failure in the pupillary activity indicates that the lesion must have involved the sensory motor arc of the pupil as well as the visual fibres. Although when present the Wernicke sign is of great value, recent observations have shown that its absence is not conclusive. Lesions of the optic tract may be due either to neoplasms or to tubercular or gummatous meningitis, or more rarely they may be the result of cerebral softening and hemorrhage. As yet clinical evidence is too meagre to permit of a diagnosis of lesions of the primary optic ganglia (pulvinar anterior corpora quadrigemina and external geniculate bodies), although in lesions of the pulvinar two typical symptoms occur—viz., hemianopsia and athetosis—and sometimes hemianæsthesia may be present. In like manner, also, while it is generally believed that lesions of the optic radiations cause homonymous hemianopsia, it has not been definitely proven that these fibres have solely to do with vision.

The hemianopsia is usually thought to depend upon cortical lesions in the occipital lobe, when it is unaccompanied by any of the accessory symptoms which have just been detailed. The chief diagnostic symptom of a central lesion, however, is what is designated as negative vision, "vision nulle," for in these cases the patient has no subjective sensations of the defect in his visual field. Cortical hemianopsia may also be incomplete, but a quadrant of the field being lost.

Transitory hemianopsia, or scintillating scotoma, is the occurrence of symmetrical defects in the field of vision which usually conform to the hemianopic type, and in which a play of lights frequently appears as a precursor of an attack of migraine. (See Migraine.)

Visual hallucinations may also be hemianopic in character, and are due to irritation of the visual memory centre.

Hysterical amblyopia may manifest itself either in complete blindness or central scotoma, but more commonly as defective central vision with concentric contraction and reversal of the visual fields.

Paralysis of the Motor Nerves of the Eyeball. Although in the section which dealt with the diseases of the ocular muscles the various forms of ocular deviation and the different varieties of diplopia

which resulted therefrom were mentioned at length, it is necessary to refer still further to their causes and to point out their connection with cerebral diseases.

Paralysis of the orbital muscles may be due to orbital lesions or to those at the base of the brain ; they may indicate pontine lesions, or they may be originated by causes operating higher up in the cerebrum above the nuclei. In making the differential diagnosis between central and peripheral palsies, it must be remembered that those of central origin are frequently associated with other symptoms which denote intracranial involvement, while peripheral palsies are generally isolated and often complete.

PERIPHERAL PARALYSES of the orbital muscles are generally the result of either rheumatism or syphilis. When due to the latter disease they are usually tertiary manifestations, and especially is this apt to be the case if the third nerve is involved, which seems to be singularly prone to be attacked by gumma of the base. Paralysis of the sixth nerve is frequently of rheumatic origin.

Syphilis causes fully one-half the cases of central paralysis, affecting either the nuclei of the nerves or the neighboring brain structure, the third and fourth ventricles, or the aqueduct of Sylvius.

Diphtheria usually causes a paralysis of the ciliary muscle ; it may, however, affect one or more of the external muscles. Diabetes is complicated at times by paralysis of the external rectus. Influenza, herpes zoster, and whooping-cough are also rare causes of ocular palsies. Paralysis of the eye muscles is seen in paretic dementia, bulbar paralysis, and in multiple and posterior sclerosis. In locomotor ataxia they may be transient and appear at an early stage of the disease. Ocular palsies have also been caused by poisoning by lead, nicotine, sulphuric acid, carbonic oxide, and tainted meat.

COMPLETE PARALYSIS OF THE THIRD NERVE causes the following symptoms : The upper lid droops, the pupil is partially dilated and immovable, and the power of accommodation is lost. The globe is slightly protruded and strongly diverged externally by the two unaffected muscles (the external rectus and the superior oblique). In *incomplete paralysis of the third nerve, as well as in paralysis of the fourth and sixth nerves*, the diagnosis is made by a study of the deviations and by the character of the diplopia, which has been already referred to.

There is a peculiar form of intermitting *paralysis of the third nerve*, known as ophthalmoplegic migraine, which occurs in the young and is associated with headache and at times with vomiting.

Paralysis of the ciliary muscle, or cycloplegia, follows a lesion of the trunk of the oculomotor nerve or of the anterior part of its nucleus. It is quite common as a sequel of diphtheria, and occurs, though rarely, in connection with spinal disease.

Ophthalmoplegia externa and interna refer to paralyses of all or nearly all of the external and internal muscles. As the lesion in this affection is central, it is frequently known also as nuclear paralysis. In its *acute form* it is due either to an acute inflammatory process in the nuclei or to hemorrhage, while *the chronic* depends upon a degenerate atrophy of the nerve nuclei, similar to that which is seen in progressive

muscular atrophy and in chronic bulbar paralysis, with which they may become associated.

In conjugate lateral deviations of the eyes, although the axes of vision of both eyes are deviated from the middle line, yet they remain parallel with one another. This condition is generally the result of a cortical lesion which involves the movements of the eyes to the right or to the left, and is usually the result of apoplexy. A spasm deviation of the eyes in the same direction occurs as the result of irritative lesions of the brain, involving the association centres or tracts, and also in hysteria.

The Localizing Value of Paralysis of the Orbital Muscles.[1]

Paralysis of the Third Nerve. Ptosis, the most frequent symptom of diseases of this nerve, may be present as a focal symptom in cortical lesions without paralysis of any other branch of the third nerve. This would seem to indicate a special centre for the elevator of the lids, and though not definitely ascertained, such a centre is believed to exist in front of the upper extremity of the ascending frontal convolution close to the centre. Ptosis on the side of the lesion, without paralysis of the other branches of the third nerve, has been seen in disease of the pons, and again by forming a factor of a crossed paralysis may seem to localize a lesion in the crus cerebri, although when the third nerve is paralyzed by a lesion in this situation it is usually involved as a whole.

Crossed hemiplegia is a term used to express a disease of the crus cerebri when there is paralysis of the third nerve on the side of the lesion, with hemiplegia, hemianæsthesia, and often facial and sometimes hypoglossal paralysis of the opposite side of the body.

Complete paralysis of every branch of the third nerve without any other paralysis is almost always basal; so, also, are those cases in which when there is hemiplegia it is slight as compared with the degree of the third-nerve paralysis. Lesion of the interpeduncular space and thrombosis of the cavernous sinus also indicate third-nerve palsies; but in the latter the other orbital nerves, as well as the fifth and the optic nerve, may be involved as well Third-nerve symptoms may also be distant symptoms of tumors of the cerebral hemispheres, more particularly if accompanied by violent general head symptoms.

As a symptom of cerebral lesion *solitary paralysis of the fourth nerve* is rare. When present it is apt to be produced by a basal lesion. In combination with paralysis of the third nerve it speaks for a lesion in the cerebral peduncle extending back to the valve of Vieussens.

When *paralysis of the sixth nerve* occurs as the only focal sign it is probably due to disease of the base as a distinct symptom. On account of the lengthened course these nerves take over the most prominent part of the pons, which renders them readily affected by distant pressure; they are more liable to provide a distant symptom than any other cranial nerve. Thus paralysis of this nerve is not infrequently a distant symptom of tumor of the cerebellum, whereas paralysis of the

[1] This section has been epitomized from the excellent article on the subject in Swanzy's Hand Book of Diseases of the Eye.

third nerve is more apt to be a distant symptom in a lesion of the cerebral hemisphere.

Paralysis of the sixth nerve, simultaneous in its onset with hemiplegia of the opposite side of the body, indicates a lesion in the pons, usually a hemorrhage, on the side corresponding to the paralyzed nerve. Basal paralysis of the nerve is frequently double, especially in syphilis. In combination with paralysis of the facial, paralysis of the sixth nerve is referable to a pontine lesion.

The Ear.

Subjective Symptoms. Buzzing, roaring, hissing, singing, and other sounds in the ear—*tinnitus aurium*—are symptoms which may or may not be due to disease of the ear. If associated with vertigo, it may be due to *Ménière's disease.* They may be the *aura* preceding an epileptic attack or the subjective phenomena attending syncope. Many drugs when pushed to physiological effects cause tinnitus.

The External Ear. The external ear should always be examined. The thin ear may show the anæmic or chlorotic hue more strikingly than other portions of the body, or the opposite condition may be more vividly shown. *Hæmatoma auris* is seen in general paralysis of the insane and in other forms of insanity. It is a trophoneurosis. The ear is thickened and deformed, on account of effusion of blood between the cartilages and the perichondrium. It is discolored, and simulates the subcutaneous effusion due to injury. *Tophi* are observed in the external ears of patients with a gouty diathesis. They are small, hard, gritty accretions, seen in the external ear along the margin or in the depressions. They consist of urate of soda.

The Discharge. When cerebral symptoms or symptoms of infection (pyæmia) are present the presence or absence of ear discharge must be ascertained. Middle-ear disease very frequently results in inflammation of the mastoid, and from thence the sinuses and adjacent membranes of the brain become inflamed; or the ear suppuration may be the primary focus from which general infection has taken place. It may not be possible in all cases to observe a discharge. It may have diminished or disappeared on account of the fever. Tenderness and œdema over the mastoid, perforation or bulging of the ear-drum, as well as other inflammatory signs, point to the occurrence of suppuration of the middle ear and mastoid cells. It must not be forgotten that a bloody discharge from the ear may take place in fractures of the skull. The ears must also be examined in cases of coma from injury, or if the origin of coma is obscure.

The Auditory Nerve. *The Hearing.* The power and acuteness of *hearing* must be tested. This may be done with the voice, a watch, or a tuning-fork. Normally, the instrument should be heard at an equal distance from either ear. If both sides are equally affected the hearing of a patient must be compared with that of a healthy person. The ticking of a watch should be heard at a distance of about three feet. The tuning-fork is used by placing it on the skull. In some cases the voice may be easily heard, while the ticking of a watch can be distin-

guished only with great difficulty. The tuning-fork is used to determine by bone conduction whether deafness is due to obstruction or disease of the auditory nerve. If it is due to obstruction the vibrating tuning fork placed on the vortex is heard better on the deaf side on contact with the skull than when held close to the ear (Rinne's test). Obstructive deafness, is always due to disease of (1) the external meatus, (2) the tympanic membrane and middle ear, or (3) the Eustachian tube.

Deafness from internal ear disease may be due to affections of the labyrinth—as inflammation, caries, and necrosis—or of the auditory nerve. The tuning-fork is not heard on contact with the skull. The auditory nerve may be diseased in its course, or the auditory centre may be affected. (See Nervous Diseases, Part II., Chapter VIII.)

It must not be forgotten that certain drugs, as quinine and the salicylates, may cause deafness. It may be an early and premonitory symptom of typhoid fever, or cerebro-spinal meningitis, and may occur early or late in the course of mumps. Deafness due to *occupation* is worthy of mention. It is not uncommon in blacksmiths, boiler-makers, locomotive engineers, and firemen. In some instances the patients can hear better in the noise incident to their work than when the surroundings are absolutely quiet.

Hyperæsthesia of the Auditory Nerve. Very rarely in certain cases of facial paralysis, and not seldom in hysteria, there is abnormal acuteness of hearing (oxyacoia). In some individuals suffering from hemicrania or tic douloureux, and in meningitis, the hearing of certain sounds—for example, high musical notes and whistling—is accompanied by pain. Nervous patients often complain of subjective noises, buzzing, roaring, hissing, and singing—the so-called tinnitus aurium.

Paralysis of the Auditory Nerve. No case of absolute unilateral deafness, due to a focal lesion in a hemisphere, has as yet been observed. Deafness from disease of the auditory nucleus is very rare. That due to disease of the peripheral nerve is much more common. We may have a rheumatic auditory paralysis similar to that of the facial nerve, or the deafness may be due to pressure from a tumor or inflammatory exudate at the base of the brain, or disease of the mastoid process of the temporal bone. The localization of the lesion is often extremely difficult. The only positive point is, that labyrinthine disease is apt to be accompanied by vertigo while in disease of the nerve-trunk vertigo is absent.

Ménière's Disease. *Aural Vertigo.* We may define vertigo as a subjective feeling of motion referred by the patient either to his own body or to surrounding objects, with loss of equilibrium and without unconsciousness.

In this disease, first described by P. Ménière in 1861, there is paroxysmal vertigo (sometimes so sudden and intense as to throw the patient to the ground), tinnitus aurium, nausea, pallor, clammy sweat, and vomiting. The severity of the attacks varies greatly. There may be momentary unconsciousness. There is sometimes jerking of the eyeballs, nystagmus, or diplopia. The disease is paroxysmal in character, but slight vertigo and tinnitus are apt to persist between the

attacks. Some deafness is present. The attacks may vary in frequency from several in a day to only one in several months.

Paralyzing Vertigo. Gerlier describes a remarkable form of paroxysmal vertigo accompanied by weakness, paresis in the extremities, drooping of the eyelids, marked lassitude, and depression without unconsciousness. It occurs only in men, and is epidemic in the Canton of Geneva.

Hysterical or *functional* deafness is recognized by (1) its association with undoubted symptoms of hysteria; (2) its sudden occurrence after shock, emotional disturbance, or trauma; (3) the absence of a cause in the auditory apparatus for the deafness; (4) impairment of bone-conduction and aërial conduction to the same degree; (5) the frequent coexistence of anæsthesia of the pinna and external meatus; (6) frequently recovery takes place suddenly.

Hysterical *deaf-mutism* is a rare condition, characterized by (1) sudden origin; (2) absolute aphasia and aphonia; (3) absence of signs of paralysis of the lips and tongue and of any paralytic phenomena except hysterical hemiplegia; (4) preservation of intellectual faculties and power of writing; (5) frequent coexistence of hysterical stigmata; (6) usually rapid recovery.

CHAPTER IX.

THE DATA OBTAINED BY OBSERVATION—(*Continued*).

The *extremities*—*hands*. The shape—temperament—occupation—"claw-hands"—"seal-fin hands"—rheumatoid arthritis—nervous affections—"spade" hands—large bones of acromegalia—osteo-arthropathy—wrist-drop. The movements—spasm—tremor. The *skin*—color—moisture. *Fingers*. Heberden's nodosities—contraction of fascia—Dupuytren's contraction—deviations in shape. The *nails*. Trophoneuroses—cold hands and feet. Raynaud's disease—erythro melalgia.

THE EXTREMITIES.

The Hands.

The Shape. We bear in mind the variation in the form of the hand in different types of individuals—the broad and heavy hand of the sanguine, the slender, dexterous hand of an individual of the nervous temperament (see Chapter VI.), the large joints of the hand of so-called strumous persons, and the effeminate hand of the one who is inclined to tuberculosis, present sharp contrasts. Then, too, the "occupation" hand indicates in a general sense the disease the patient is liable to—none more striking than the hand of the miner, the blue-black dottings of which sharply indicate the possibility of anthracosis. Finally, we note the broad hand and clubbed fingers that are seen in congenital heart disease. The withered hand of age and wasting of the hands, as in phthisis or malignant disease, need not be referred to, as they are part of the general process.

FIG. 13.

Pseudo-muscular atrophy. Claw-hand. (GRAY.)

Presenting more striking changes in shape are the peculiarly deformed hands seen in affections of the muscles and joints. These deformities will be described in the respective sections (Chapters XII.

and XIII.), although in passing they may be grouped together. First we have the "claw-hand" of progressive muscular atrophy, of inflam-

FIG. 14.

Rheumatoid arthritis. The tapering fingers are seen. The phalangeal joints are swollen; many are anchylosed. The wrist is stiff. The muscles are atrophied; the forearm-muscles much wasted.

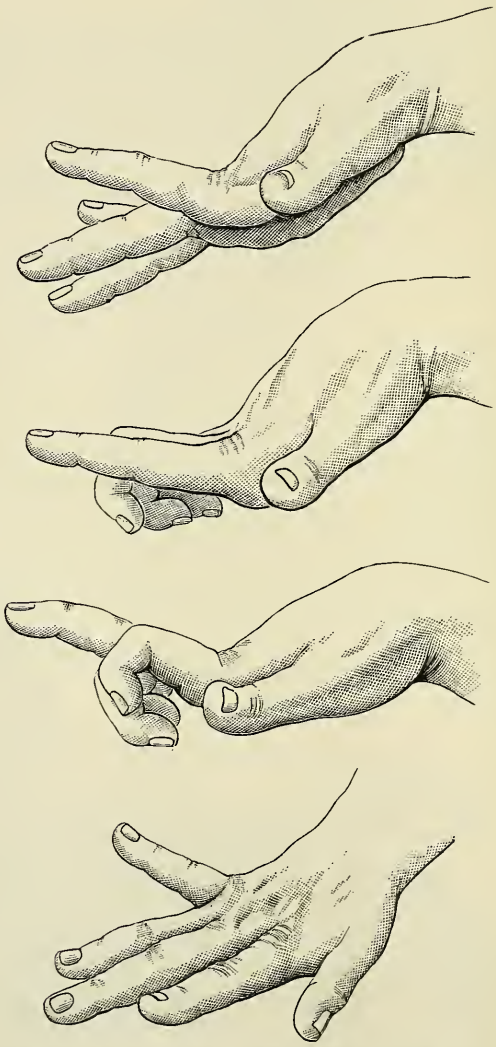
FIG. 15.

Photograph of a case of lead-paralysis affecting the extensor muscles. (GRAY.)

mation of the ulnar and median nerve, and of chronic poliomyelitis; the "seal-fin" hand of chronic gout and rheumatoid arthritis, spasm of the extensor muscles causing deflection to the ulnar side. The

gnarled hand of rheumatoid arthritis and the knotted hand of gout are characteristic. In the former the tapering, shining fingers, the bulbous phalangeal joints, the pallid, clammy surface, dotted with freckles, the locked joints, the atrophied muscles, combined with exquisite tenderness of the involved parts, make a picture never to be forgotten. The

FIG. 16.



Examples of the positions of the fingers in the movements of athetosis. (STRÜMPELL.)

peculiar deformity occurring in scleroderma is described in the chapter devoted to the skin. Then we have the deformity resulting from flexion of the hand on the forearm, the forearm on the arm as seen in *cerebral palsies* of children and in the *hemiplegias*.

The "spade-like" hands of *myxœdema* and the enlarged bones of the hands of *acromegalia* and *pulmonary osteo-arthropathy* are described in other sections.

Deformities of the hand from other causes than the ones just mentioned are often observed. Temporary contractures occur in tetany, in temporary hemiplegia or monoplegia, and in paralysis of the extensors. Dropping of the hand from the radius toward the ulna occurs in acute poliomyelitis from paralysis of the extensors. Then we have paralysis of the median, ulnar, and other nerves, with their characteristic deformity. (See Nervous Diseases.) So-called *wrist-drop* is seen in *peripheral neuritis* (musculo-spiral nerve), and may be unilateral or bilateral. The hand hangs from the wrist on account of paralysis of the extensor muscles. Both hands may drop, although it sometimes happens that one is affected from a few days to a few weeks before the other.

Movements. One can infer the limitation of movements of the hands in the affections described above. The stiffened and immobile hand of chronic rheumatism, in which enlarged joints are prominent, contrast with the painfully locked hand of rheumatoid arthritis. Involuntary movements, as tremors and spasms, are also observed. The *tremor* of age, of hysteria, of paralysis agitans, of exophthalmic goitre, of mercurial and other intoxications, and of disseminated sclerosis, is most marked in the hands. It is in the hands and arms we see that most significant tremor or twitching with aimless picking at the bedclothes, described in an account of the typhoid state (Chapter XIV.), known as *subsultus tendinum*. *Twitching* and *spasm* of the hand or arm are seen in convulsive disorders, and may be unilateral or bilateral, as in hysteria, chorea, epilepsy, true and Jacksonian, tetanus, and tetany. When permanent, it is seen as an expression of a chronic cerebral process, as hydrocephalus. Alternating spasm and relaxation of the fingers, hand, and arm are seen in athetosis.

Having noted the shape and movement of the hand, we direct attention to the skin, the nails, and the fingers.

The Skin.

The *skin* of the hand need not be considered apart from the skin of the rest of the body. It is smooth or rough, dry and harsh, moist and warm, under the same circumstances that affect the skin generally. In rheumatoid arthritis it has been described as peculiar. Both the dorsal surface and the palm are moist and very soft, and the former is dotted with freckles. In progressive muscular atrophy and exophthalmic goitre the skin is moist. The cold, clammy skin of one laboring under excitement, as may be caused by the first visit to the physician, is well known.

Color. The color of the hands is significant of the state of the circulation and the condition of the blood. The blue finger-tips and the pallid hand accompany similar color changes in the lips, and are early signs of cyanosis and of anæmia.

The *swellings* of the hand, inflammatory or œdematous, do not differ from swellings of the joints or the subcutaneous connective tissues in other portions of the body. Several exceptions are to be noted. First,

the swelling that attends articular *rheumatism* with involvement of the wrist-joint extends over the dorsum of the hand frequently, while the fingers are free from the process. Second, a localized swelling on the dorsum of the hand is often due to a *ganglion* from a local affection of the tendons. Third, Gubler's *tumor* is a swelling that is seen in wrist-drop from displacement backward of the carpal bones. Fourth, long-continued inflammatory swelling, with subsequent rupture of the skin, is seen in *mycetoma*. Finally, traumatic injuries produce tendo-synovitis, bone affections, and palmar abscess. Syphilis and gonorrhœa may be causal factors in the production of such processes, it is important to remember. (See Chapters X. and XIII.)

The Fingers.

In gout and rheumatism the joints of the *fingers* are enlarged and painful. The swellings of the joints of each condition cannot well be distinguished. In gout, tophi, hard, white, sometimes glistening masses are likely to be present in the joints or along the tendons, on account of great accumulation of urate of soda. They are more prominent on the dorsal surface of the joints, and sometimes break through the skin, so that the "chalk-like" concretion exudes. It was said by Sir Thomas Watson that a gouty subject under his care used his joints to keep tally while playing cards.

FIG. 17.

Heberden's nodes.

Heberden's Nodes. *Haygarth's nodosities.* The term "end-joint arthritis" is also applied to this condition. This node belongs to the first of the three divisions Charcot makes of rheumatoid arthritis. The nodules develop gradually at the sides of the distal phalanges. The subject may be in good health, or may have had attacks of gout, or have suffered from acid dyspepsia. At first the joints may be a little swollen and tender. The swelling and tenderness may be periodical, and the size may be increased with each fresh paroxysm. The tubercles are seen at the side of the dorsal surface of the second phalanx, the corresponding cartilage becomes soft, the ends of the bone may be eburnated. A moderate anchylosis takes place. The nodules are often considered of good prognostic omen; it is even said that they are a sign of longevity. It is certain that the large joints are rarely involved when these nodules are present.

The tips of the fingers may be bulbous, or *club-shaped*, in some cases of phthisis and of other forms of chronic lung disease, and also of chronic heart disease. It is most common, however, in bronchitis and phthisis. The clubbing is associated with changes in the nails (see below and illustration of pulmonary osteo-arthropathy).

Deviations in Position and Shape. Eversion is characteristic of rheumatoid arthritis, but deviations due to abnormal flexion or extension are the most characteristic. *Flexion* of the first phalanx of the little finger is due to contraction of the palmar fascia or to paralysis of the common extensor from disease of the musculo-spiral nerve. Contraction of the fascia of the hand, causing more or less flexion of the little and ring fingers, is frequently seen, and may be an indication of gouty diathesis. It is certain that these contractions are seen in several members or generations of a family in which gout is prevalent. It is called Dupuytren's contraction.

Abnormal *extension* is usually very marked. Hyper-extension of the middle phalanx is due to paralysis of the flexor sublimis from disease of the median nerve; hyper-extension of the distal phalanges to paralysis of the flexor profundus muscle from disease of the median and ulnar nerves. Extension of the proximal phalanx, with extreme flexion of the two distal phalanges, contributes to form the "claw-hand." (See Muscles.) Contractions due to chorea or to central lesions, as post-hemiplegic contractions, will be considered under special diagnosis. It is thus seen that the peculiar combined extension and flexion, causing abnormal shape of hands and fingers, is due to (1) local joint inflammation (subluxations); (2) local neuritis and paralysis; (3) progressive (spinal) muscular atrophy; (4) idiopathic muscular atrophy, rarely.

The Circulation. Raynaud's Disease. Local Asphyxia. The hands or fingers become pale and intensely cold; they are the seat of numbness, and are without sensation. The term "*dead fingers*" graphically describes the appearance. The pallor usually comes on suddenly, and continues for a variable period. As the pallor disappears there is a gradual return of warmth, and the color changes to a livid red, dark blue, or even blackish hue. The paroxysms of alternating pallid and livid hue may occur several times in twenty-four hours. In some cases the lividity becomes so intense that gangrene ensues in small superficial spots, or even involves the whole finger. Pain may or may not be present, and does not increase when the hand hangs down. In my experience it is more frequently present and excruciating at the time the fingers are "dead." The tip of the nose and the lobe of the ear may be affected, and occasionally other parts of the surface. The sensitiveness to touch is markedly lessened. *Raynaud's disease* occurs usually in ill-nourished subjects, or after an acute disease, as typhoid fever. It may be associated with vascular spasm in internal organs, giving rise to epilepsy, hæmoglobinuria, temporary aphasia, or hemiplegia. It is usually worse in cold weather.

Erythromelalgia. Local changes in *color* are often due to neuritis either of the trunk or of the terminal endings of the nerves. When such changes are associated with pain we use the term erythromelalgia.

It is characterized by redness of the surface with increased temperature; it is usually seen in the extremities and is limited to the distribution of the affected nerve. It is worse in summer, increased by artificial heat, and aggravated when the extremity is dependent or pressed upon. The redness is attended by burning and extreme local discomfort, in which all sorts of sensations are described—tearing of the finger-nails, pulling or pricking of the skin, twistings of thousands of needles, and other forms of torture. I know of no peripheral pain which is the source of greater agony.

Glossy skin is seen after nerve-injuries and neuritis, and in central affections in which the trophic nerves are involved. The skin is shiny, smooth, drawn very tightly over the surface, and sometimes atrophied. Red and pale mottling may be seen. The surface is free from hair. Burning pain precedes and accompanies the change. (See Nails.)

The Nails.

The Shape. The appearance of the nails enables us to estimate the duration of certain diseases, or the time when convalescence began; it also indicates local interference with the nutrition of the parts. Thus, curving of the nails, with the club-shape of the finger-ends, occurs only

FIG. 18.

Clubbed fingers with curved nails (middle finger slightly flexed).

in chronic diseases, as phthisis or emphysema, or in chronic cardiac disease and aneurism. In the latter it is sometimes found on one hand only. It is sometimes seen in other chronic wasting diseases. The nails may curve transversely or longitudinally. When transversely the appearance is like that of a filbert, and when longitudinally they are said to be incurvated. This change in shape may occur without clubbing of the fingers. The shape is altered in acromegalia and pulmonary osteo-arthropathy. (See Chapter XIII.)

Color. White marks on the surface are usually seen after an illness, and may indicate the date of recovery. The marks develop at the root of the nail, and as the nail grows the marks approach the tips of the fingers, and thus their position denotes the time that has elapsed since convalescence set in. If they are seen half-way up the nails, convalescence is probably of three months' standing. We get a good idea of the condition of the blood in the capillaries from the appearance of the tissue under the nails. If there is anæmia, pressure on the finger-tips will drive the blood from the capillaries. Stephen Mackenzie's rule, that if such pressure completely empties the vessels so that they become pale, it indicates that the globular richness of the blood is reduced one-half, is a fair and rapid test of the degree of the anæmia. The purplish and bluish-black discoloration of *cyanosis* previously referred to is first seen under the nails. Sometimes the *capillaries pulsate*, and this pulsation is more visible under the nails than in any other part of the body except the retina. It may occur in aortic regurgitation.

Nutritive Changes. The nails undergo chronic inflammation with destruction in various skin affections, and the matrix is the seat of acute inflammation in onychia. Onychia may be simple or syphilitic. Its presence may indicate the organic origin of otherwise obscure nervous symptoms. It may be only a simple inflammation, or it may result in the loss of the nail and necrosis of the bone.

Deformity of the nails (toe) occurs in acute and chronic myelitis. In *locomotor ataxia* the nails fall out.

In *neuritis* the trophic change is marked; the growth is arrested, and the nail becomes dark and brittle and curved in its long axis, while lateral arching takes place. The cutis underneath thickens and the skin at the base retracts. The fingers may be clubbed. When growth is resumed a distinct roughened line of demarcation is seen. In leprous neuritis there is destruction of nails and phalanges. Atrophy and ulceration at the base of the nails, followed by necrosis of the phalanges, is seen in so-called Morvan's disease, which is not really a disease but a symptom of neuritis or syringomyelia. Enlargement with thickening and sometimes twisting occurs after fevers, as typhoid, or in the course of syphilis and in sclerodactyle. The nutrition is changed in Raynaud's disease. In some cases the nails become dry, scaly, and cracked, or hypertrophied entirely. In the hemiplegia from cerebral apoplexy the growth is arrested on the paralyzed side. This is tested by staining the nails of the two hands at the same level with nitric acid; the relative position of the stain upon corresponding nails of the two hands will show whether there has been growth or not. The return of functional power is indicated by renewed growth.

The Feet.

Enlargement or deformities of the feet and legs may be due to changes in the joints, the bones, and the subcutaneous connective tissue. Hence we would have swelling due to œdema and myxœdema, and enlargements due to acromegalia and pulmonary osteo-arthropathy.

The chapters so frequently referred to will contain a discussion of these subjects, and to the Chapter on Joints must be referred all articular changes. It must be recalled that *pain* may be due to flat-foot and to neuralgia of the third interosseous nerve. (See Pain.) Flat-foot must always be looked for when inability to walk is complained of. Changes in the shape of the foot from muscular affections will be described, bearing in mind that "claw-foot" is a prototype of "claw-hand," found in progressive muscular atrophy and in Friedreich's ataxia.

Three *nutritional changes* take place in the feet that are of diagnostic significance: perforating ulcer of the foot, a trophic change occurring in locomotor ataxia; gangrene, the result of endarteritis (usually senile), or occurring in the course of diabetes mellitus; mycetoma, or "Madura foot." Perforating ulcer usually begins as a blister, then an abscess, and finally an ulcer.

The *nails* of the feet are subject to the same changes that take place in the nails of the fingers.

Cold Hands and Feet. Patients frequently complain of coldness of the extremities. It is a common and often serious complaint. It is natural to expect a peripheral coldness when the central organ of circulation is weakened. Coldness takes place in the final hours preceding death. It occurs in collapse, in hemorrhage, and in shock. But we also see it in organic disease of the heart, with impairment of the circulation. It is a common vasomotor condition in nervousness, independent of hysteria. It is a marked feature in Nothnagel's angina pectoris vaso motoria, as well as in true and false angina pectoris. A visit to a physician, or excitement from any cause, is likely to be attended by coldness of the hands and feet. Under these circumstances the extremities are often bathed in a cold and clammy perspiration. In senile endarteritis cold hands and feet frequently occur.

They are an index to the state of the peripheral circulation in other parts of the body, as the brain.

The poisons of gout, of rheumatism, and of other diseases, which irritate peripheral and vasomotor nerves, may cause cold hands and feet.

In gastric and intestinal dyspepsia, with the absorption of toxic principles, as leucomaines, this symptom may be present.

Changes of *sensation* in the skin of the extremities will not be considered in this section. They will be taken up in the chapters devoted to the diseases of the nerves. It is sufficient to state that *anæsthesia* in local areas, and due to causes limited to the skin, is seen in morphœa, in the anæsthetic form of leprosy, and in certain ischæmic states (urticaria). It is accompanied by loss of tactile sensibility. *Hyperæsthesia* and *paræsthesia* occur with various local affections, but they are without diagnostic significance except in nervous diseases.

CHAPTER X.

THE DATA OBTAINED BY OBSERVATION—(*Continued*).

The *skin*. The color—redness—pallor—jaundice—cyanosis—the bronzed skin—Addison's disease—hæmochromatosis—chloasma—tinea versicolor—vagabond's disease—argyria—freckles. The nutrition. Moisture and dryness—hyperidrosis—anhidrosis. Scars Hemorrhages—mode of recognition—cause—significance. Eruptions—their clinical significance—nature of the lesion—distribution—associate morbid phenomena—general symptoms. Table of skin diseases—erythema—herpes—erythema nodosum—urticaria—medicinal rashes—erythema of infectious diseases—roseola—miliaria or sudamina. General diagnosis.

THE SKIN.

Color. The portions exposed to the air exhibit more varied and pronounced changes of color than parts that are covered. The changes in color herein described refer more particularly to the face and hands. The color of other parts partakes of the same tint as that of the face, other things being equal, except that the intensity is less. Comparison of the two should always be made, and the mucous membranes examined, as control observations. For the latter the conjunctivæ, lips, and mouth are sufficient, always remembering the possibility of hyperæmia of the conjunctiva from other causes.

Local color changes of the *face* will be particularized in this section. It is not to be forgotten that the color varies with the type—whether blonde or brunette—and that variations in the latter at times easily escape recognition.

The skin in a healthy child is of a faint pink color; as age advances it loses its fresh appearance and becomes paler, except in those whose occupation exposes them to atmospheric influences. In the latter, the skin becomes weather-stained, and may assume a mahogany or reddish-brown hue. In old age, the color is apt to deepen and become duller, while the loss of subcutaneous fat allows the skin to lie in folds, especially about the jaws and neck, and wrinkles are marked, especially between the eyebrows, over the nose, and at the angles of the eyes and mouth.

Apart from these changes, which are physiological or necessarily the result of occupation, the skin exhibits changes which are the result of the habits or health of the individual. Some persons, especially if blondes, retain to old age the fresh, pink skin of childhood. In others is seen early a dull, *muddy complexion*. This is common in those who use coffee to excess and are habitually constipated. In others digestive derangements, particularly constipation, uterine disorders, or gouty derangements produce, in addition to a muddy complexion, crops of acne and comedones, or black-heads. It must be admitted, however,

that some persons preserve a fresh complexion in spite of marked digestive disturbance. Considerable congestion of the superficial blood-vessels, giving a person a *florid* appearance, may be due, especially in a young person, to alcoholic excesses; and there is a popular belief which connects such an appearance, when coupled with a tuberous nose and a crop of angry-looking pustules, with a prolonged use of spirits.

The *sebaceous glands* of the skin of the face merit but a passing notice. Deficiencies or excesses of secretion, or alteration of it, are usually due to local causes. Excessive secretion of sebaceous matter, known as *seborrhœa*, or steatorrhœa, is seen in two forms. First, with oily exudation; second, with drying of the secretion and the formation of crusts. It may be more pronounced in strumous subjects. The opposite condition, or *asteatodes*, is seen in wasting diseases, particularly diabetes, and in xeroderma and ichthyosis.

Color Increased. The Abnormally Red Skin. Physiological hyperæmia has been spoken of. The color is intensified when the capillaries are overfilled or the blood-current is unusually rapid. The hyperæmia may be general or local, and is due to dilatation of the capillaries, possibly from nerve-influences. *General* hyperæmia is seen in fever, in poisoning from atropine, and from organic poisons derived from food or the result of intestinal putrefaction.

Local hyperæmia attends the phenomena of blushing, and comes and goes in nervous persons with every psychical impression. Rarely in neurasthenics the hyperæmias may be extreme, amounting almost to an erythromelalgia. Abnormal redness may be diffused over the whole face or may present the circumscribed flush of phthisis; the local deep-red area, on one cheek, of pneumonia; the evanescent flush of anæmia, with cardiac palpitation; and the creeping flush, with raised border, of erysipelas, appearing on the bridge of the nose or at the nostril. In phthisis, moderate excitement or exertion, the taking of food, or the onset of fever, tinges the cheek with the blush of hectic. In migraine, the burning flesh may be limited to one side. Capillary congestion on the cheeks or on the tip of the nose occurs with the endarteritis of the aged, but is seen also in early life in cases of hepatic cirrhosis or of obstruction of the hepatic circulation from other causes.

Color Lessened. It is caused by diminution of the amount of blood in the capillaries, or because its richness in hæmoglobin has been reduced.

Pallor. Diminished amount of blood in the capillaries occurs from active contraction or spasm of the arterioles, from hemorrhage, or from weak heart. The pallor, therefore, is usually *acute* or *temporary*, and may be recurrent. It attends fright, syncope, or nausea and vomiting. It occurs also in acute poisoning, in acute disease, such as diphtheria, and in hemorrhage. The pallor due to loss of blood may be instantaneous if the hemorrhage is sudden and large, or develop gradually if it is small and continued over a long period. The onset of sudden pallor is of diagnostic significance in diseases in which hemorrhage may occur, as in aneurism, gastric or intestinal ulcer, and typhoid fever. Symptoms of collapse are seen with this form of pallor.

Pallor of long duration, or *chronic pallor*, if we may so term it, is seen in a number of diseases. In all of them there are diminution in the amount of red corpuscles and destruction of the hæmoglobin. It is characteristic of blood affections, as the various forms of anæmia. It does not necessarily occur in leucæmia; indeed, the cheeks and lips may be red. It is seen, in a striking form, in chronic Bright's disease, in cancer, in chronic poisoning, as from lead or arsenic, in chronic catarrh of the stomach or of the bowels, and in chronic infectious processes, as tuberculosis and syphilis.

While *paleness* is recognized as the fundamental or prevailing color of the skin in many of the above-noted affections, a further tinge gives a characteristic hue to the skin; thus, in *chlorosis* there is a *greenish* appearance of the face, which is in striking contrast to the pearly colored conjunctivæ. In *carcinoma* the *yellowish* tinge of the pallor often causes it to be mistaken for jaundice. In *pernicious anæmia* a *straw-colored* appearance of the skin has been frequently described, which may cause it to be mistaken for carcinoma. It is worthy of remark that the cachectic pallor in carcinoma is not likely to occur unless there are primary or secondary deposits in the gastro-intestinal tract or the liver, and it is well known that pernicious anæmia is usually secondary to gastric or hepatic disorder. The peculiar hue of the pallor, therefore, may be due to a common cause in these affections. The pallor that attends *Bright's disease* is usually associated with slight puffiness under the eyelids, or local dropsical accumulations elsewhere. In chronic poisoning with *lead* pallor is associated with a blue line upon the gums and drop-wrist; while in arsenical poisoning there are frequently associated a puffiness of the eyelids and looseness of the bowels.

It is not well to lay much stress upon the variations in hue of the pallor. They are not of diagnostic importance in themselves, but only when associated with the characteristic symptoms and signs of the respective affections in which this hue occurs.

It must not be forgotten that there are a large number of individuals in whom pallor is the normal condition. This is particularly the case with those who lead a sedentary life and are confined within doors. There are a number of occupations which predispose to pallor.

Abnormal Color. I. The Yellow Skin. Jaundice. The yellow coloration is seen not only in the skin but in the scleræ (see the Eye) and the mucous membranes. The discoloration of the skin is not difficult of recognition. It varies in shades from a slight yellow hue to yellow-green or olive-green, and in many forms of jaundice to brownish-yellow. The yellow hue of the skin in jaundice may be preceded and is always accompanied by tingeing of the conjunctivæ; its presence in this situation confirms the observation. The mucous membrane under the tongue early gives evidence of jaundice; or, if the lips are everted and a glass slide pressed evenly on the surface, the yellow discoloration of the mucous membrane will shine through.

The yellow tint of the conjunctivæ must not be confounded with the same color due to subconjunctival fat. The latter is not uniform in the conjunctivæ, but may occupy cone-shaped areas.

The physiological yellow color of the skin that is seen in infants shortly after birth is not a true jaundice, but in all probability arises from excessive destruction of red corpuscles in the over-congested skin. On light pressure with the finger the color changes. It fades from shades of yellow into the genuine flesh-color. The conjunctivæ are natural, and the urine is free from bile-pigment. The fæces are normal. By these symptoms a distinction can be made.

Jaundice is a symptom due to a number of diseases. In the first place, it is most frequently due to disease of the liver; this form is known as *hepatogenous* jaundice. It may possibly be due to destruction of the corpuscles of the blood and liberation of the hæmoglobin, the so-called *hæmatogenous* jaundice. The various causes of the former will be considered under diseases of the liver. The latter is said, not without objection, to be due to destructive agencies in the blood, such as ptomaïnes, which are absorbed in gastro-intestinal disease, or to poisons that develop in the course of pyæmia, yellow fever, malarial and relapsing fevers; it may also be due to snake-bite or to poisons that are imported, as in mineral poisonings, or chloroform, ether, or chloral. In both instances the yellow coloration of the skin is due to coloring-matter of the bile or of the blood, or bilirubin, which is deposited in the cells of the rete mucosum.

Other symptoms due to the same cause are associated with *hepatogenous* jaundice. Their presence may be of diagnostic value in determining the nature of the yellow color of the skin in cases of doubt. These symptoms are: (1) *Itching.* This symptom is intolerable; the surface of the body is often seen to be covered with scratch-marks on account of the irritation of the peripheral ends of the nerves in the skin by bile-pigment. (2) *Slow pulse.* Slowness of the pulse also frequently attends jaundice. (3) *Secretions* and *excretions.* The *saliva,* or expectoration, if present, is bile-tinged, and the *urine* is dark colored, due to the presence of the pigment. (See Urine.) While the excretions are all tinged with bile in the hepatogenous form, the fæces are free from bile, hence they are pale or of an ashy color. On account of the absence of bile in the intestines its physiological effects are lost, and therefore *flatulency* from fermentation becomes an important symptom.

II. The Blue Skin. *Cyanosis.* This peculiar hue is recognized without difficulty. The bluish or bluish-red appearance of the skin is first seen at points furthest from the central organ of circulation, as in the extremities. The mucous membranes, in which the capillary circulation is readily seen, also exhibit the change early. It is early seen also in the finger-tips, particularly underneath the nails, about the phalangeal joints, and in the lips. Subsequently the entire surface of the skin may become dusky or cyanosed, as its cause increases in degree. Its onset, it is said, can be anticipated by the state of the veins on the under part of the tongue; overfilling or extreme distention of these vessels always occurs in cyanosis. At first the color, wherever situated, usually disappears on pressure, but as the hue deepens it remains in spite of pressure.

Causes. Cyanosis is (1) respiratory, due to overfilling of the veins and capillaries with blood not sufficiently oxygenated, or (2) vascular, to an excess of venous blood, oxygenation not being interfered with.

1. Respiratory. All conditions which interfere with the aëration of the blood cause more or less cyanosis. Practically sufficient air cannot get to the blood, or sufficient blood to the air. Obstruction of the air-passages, diminution of respiratory area, and diminished or inefficient respiratory movements prevent oxygen getting into the blood; interference with the circulation in the lungs prevents the blood getting air. Both causes are often combined.

a. Obstruction of the Air-passages. This may occur in the upper respiratory tract, or in the capillary bronchi. (1) *Faucial* obstruction, by pharyngeal abscess or tonsillitis, or, in rare cases, by diphtheria, causes moderate cyanosis. (2) Obstructive *laryngeal* diseases produce cyanosis varying in degree with the amount of obstruction and its persistence. The cyanosis is of *short* duration in spasmodic croup and in laryngismus stridulus; it is prolonged in the more persistent inflammatory affections. Its gradual onset, in moderate degree, as seen by the purple lips or dusky finger-tips, is of serious prognostic import in the course of tuberculous laryngitis even if symptoms of grave obstruction have not arisen. (3) *Tumors,* pressing on the trachea or bronchi, narrowing the air-channel, cause cyanosis. The tumors may be situated in the neck, as the thyroid gland, or within the mediastinum. (4) *Spasm* of the bronchi, as in asthma, occlusion of the bronchioles, as in bronchitis, both acute and chronic, and particularly the grave forms of capillary bronchitis in childhood, cause cyanosis. (5) *Foreign bodies* anywhere in the upper regions of the respiratory tract are fruitful sources of cyanosis.

b. Diminution of the Respiratory Area. Cyanosis from this cause occurs in pneumonia, in œdema of the lungs, in tuberculosis, and in all forms of pleural effusion and of intrathoracic tumors compressing the lung. It is an important diagnostic feature of acute tuberculosis.

c. Diminished or Insufficient Respiratory Movements. Deficient chest-expansion, because the action of the respiratory muscles is interfered with, lessens the respiratory area. This interference may be either on account of muscular or pleuritic pain, on account of paralysis, or, in the case of the diaphragm, on account of upward pressure by accumulations in the abdominal cavity, as large peritoneal effusions, an enlarged liver or spleen, or an abdominal tumor. In bulbar paralysis and peripheral neuritis, in paralysis of the diaphragm, and in spasm of the muscles of respiration (as in tetanus) there is diminished respiratory movement. In forms of progressive muscular atrophy and in other rare affections of the muscles, as trichinosis, cyanosis is also observed for the same reasons.

d. Obstruction of the Pulmonary Vessels. Interference with the circulation within the lungs, from pressure on the pulmonary artery or vein by aneurism or mediastinal tumor, or from disease of the heart itself, is a most frequent cause of cyanosis. In affections of the heart it is not seen until—in the case of valvular disease, for instance—compensation is lost and the right heart is dilated, causing an accumulation of

blood in the lungs. In the latter condition the bronchitis of passive congestion of the mucous membrane is an additional cause for the cyanosis.

2. Cardio-vascular. Obstruction to the flow of venous blood anywhere in the circulation will lead to the development of cyanosis. This is the *cyanosis* of *passive congestion*. Cyanosis due to causes mentioned above is always general. Cyanosis arising from the causes indicated in this section may be *general* or *local*, depending upon the seat of obstruction. *General* cyanosis may occur in (1) congenital heart disease; (2) in valvulitis, when compensation is lost and dilatation has taken place; (3) in incompetency of the valves from dilatation; (4) in weak heart or enfeebled action from pericardial effusion. In congenital heart disease the cyanosis is so great and so persistent that the affection has been termed "blue disease" or "*morbus cæruleus.*"

Local cyanosis is seen when there is obstruction of the venous trunks from external pressure, or from disease of the venous wall, causing thrombosis. It may be limited to the head and upper extremities, in obstruction of the descending cava by tumor or aneurism, or to the lower portion of the trunk and the lower extremities in obstruction of the ascending cava by pressure from tumors within the abdomen and thorax. One extremity may be the seat of local venous stasis from pressure upon the veins, or its occlusion by thrombosis; the arm in cases of cancer of the breast and axillary glands, the leg in cases of femoral phlebitis, represent typical forms of venous stasis. A striking form is due to causes affecting the vasomotor nerves, giving rise to peripheral capillary spasm. (See under Fingers, Raynaud's Disease.)

III. The Bronzed Skin. *Pigmentation. Addison's Disease.* The most marked form of bronzing is seen in Addison's disease—an affection characterized by a gradual loss of strength without much loss of flesh; by gastric uneasiness and occasional vomiting; feeble circulation, and a bronze hue of the skin.

Social History. The disease occurs most frequently during the active period of life, from the age of twenty to forty years, and nearly twice as often in males as in females.

Asthenia. The disease begins insidiously with gradual and progressive *loss* of *strength.* It becomes evident from the patient's languor, weariness on slight exertion, and inaptitude for mental effort that he is suffering with some exhausting disease. The most characteristic symptom is the extreme prostration without any obvious cause. Any exertion requires great effort and may induce *fainting.*

Gastric Symptoms. The appetite is impaired or lost, there is more or less discomfort at the epigastrium, and occasional vomiting.

Perhaps at this time a close inspection may show some discoloration of the skin, but usually this appears later. By degrees the gastric symptoms become more prominent, and vomiting may be so frequent as to shorten life materially. Finally, the patient is unable to leave the bed. Dull pains in the head, back, and abdomen are not uncommon; neuralgic pains in the limbs may be complained of; and Osler states that there is tenderness on pressure in the lumbar region in a considerable proportion of cases.

PLATE II.

Addison's Disease, Showing Bronzing of Skin, and White Areas of Atrophy. (Coleman.)

The pulse is extremely small and feeble; in the later stages it may be absent at the wrist.

Bronzing. The discoloration of the skin is the most striking symptom of the disease when it is well marked. The external surfaces are changed in hue, and delicate portions of the skin underneath the clothing are also bronzed. The discoloration is not removed by pressure. The areas are irregular in shape. The skin is soft and pliable. The pigment which causes the discoloration is deposited in the rete Malpighia.

The pigmentation is never seen in the cornea or in the nails. The axilla, the flexure of joints, the median line, the areola about the nipple and other normal areas of pigment deposit are the common sites. Bronzed areas in sharply circumscribed patches are also seen in the mucous membrane of the lips and cheeks.

Sometimes the whole body becomes of a walnut-juice color, a bronzing which is deeper in exposed surfaces. At times only portions of the body are discolored, in which case the dark hue shades off gradually into the normal hue of the skin. Wilks[1] states that in all the cases which he has seen the scalp, finger-nails, soles of the feet, and palms of the hands escaped pigmentation.

Nevertheless, discoloration of the skin is not an essential symptom of the disease; in some cases it is entirely absent. These cases, especially if associated with much vomiting, run a more acute course than the others, lasting only a few weeks. Such cases have been mistaken for *typhus fever*.

On the other hand, diseases of the suprarenal capsules not usually associated with the Addison symptom-complex, as carcinoma, are attended by pigmentation. In about an equal proportion of cases it is absent, however.

The discoloration of the skin in Addison's disease must not be confounded with that of *sunburn*. The latter discoloration is limited to parts that are exposed to the sun, is more uniform, and the mucous membranes are free. Moreover, the anæmia and debility of Addison's disease do not attend it. The pigmented areas in the mucous membrane of the mouth, seen in a certain class of negroes, must not be mistaken for the pigmentation of Addison's disease. (See Plate II.)

In persons living in filth general discoloration of the skin takes place, known as "*vagabond's disease*;" but because it is so general and the skin is rough and thickened, and other evidences of filth are seen, it can easily be recognized. In the latter stages of *jaundice* the dark-green, olive, or black hue of the skin might be taken for the general bronzing of Addison's disease. The appearance of the conjunctiva is sufficient to indicate the cause of the bronzing. In certain cases of *tuberculous peritonitis*, even if the adrenals are not involved, the peculiar brown discoloration which simulates Addison's disease is present. In *scleroderma* pigmentation occurs, although rarely.

The pigmentation that occurs in *uterine disease* or in *pregnancy* (uterine chloasma) resembles the bronzing of Addison's disease. It is usually

[1] Reynolds' System of Medicine, Philadelphia, 1880, iii. 561.

confined to the forehead and cheeks and the normal pigmentary areas of the skin. The mucous membranes are not affected, although in pregnancy there may be the characteristic change of the vaginal mucous membrane. The vomiting and weakness that attend pregnancy may sometimes lead to confusion—vomiting is early, pigmentation late in pregnancy.

The affections just described must not be confounded with the discoloration—yellowish-brown in hue—of *tinea versicolor*, a parasitic skin disease. The latter is recognized by its color and irregular dissemination. It especially occupies the chest and spreads to the abdomen. It rarely ascends above the neck. It does not usually, therefore, occur in parts exposed to the air, or in parts that are the seat of normal pigmentation. Then, again, the surface desquamates in brownish scales. Examination of the scales in a drop of dilute liquor potassæ, under the microscope, shows both spores and mycelium. The spores are of the fungus *micro-sporon furfur*. Another skin affection is attended by bronzing—*leucoderma*. In *diabetes* bronzing is often seen independently of any parasitic invasion of the skin, and apparently the result of the cachexia. It is possible that it is due to the cirrhosis of the liver which causes the glycosuria. But if the pancreas is primarily at fault the skin change is more likely to occur. In certain forms of hepatic cirrhosis, as so-called Hanot's, or the hypertrophic form, bronzing, undoubtedly the result of blood destruction, *hæmochromatosis*, is seen in rare instances.

At times the bronzing and other characteristic symptoms of Addison's disease are associated with tuberculosis in other organs. Conversely, in cases of phthisis in which there is bronzing, tuberculous disease of the suprarenal capsules may be suspected, and it adds to the gravity of the prognosis.

Argyria. If nitrate of silver is administered over a long period of time, fine black particles of the metal or of the albuminate are deposited in the kidneys, the intestines, and the skin. The corium is the principal seat of the deposition. The discoloration of the skin is gray or grayish-black. It is not changed by pressure, and is usually limited to the face and hands. Small specks may also be noted in the mucous membrane of the mouth. The cornea and nails are not affected. Persons are usually in good health, although the presence of the skin-change, if seen in a patient with coma, would point to the possible presence of epilepsy, on account of which the drug had been taken.

Freckles. Freckles are not usually of special diagnostic significance. Their occurrence in an unusual degree on the back of the hand and forearm has been observed, however, in cases of rheumatoid arthritis.

Hemorrhages.

Hemorrhages in the skin are called, according to their size, *petechiæ*, *ecchymoses*, *vibices*, and *hæmatomata*. The petechiæ and ecchymoses are apt to appear in the hair follicles, and vary in size from a pin-point to a split pea.

Mode of Recognition. They must be distinguished from erythematous and other eruptions. They may be raised above the surface of

the skin; they do not disappear upon pressure, and vary in hue from deep red to yellow-brown, according to their depth beneath the surface and to the degree of absorption that has taken place since the hemorrhage occurred.

Vierordt advises the following test to distinguish them from erythemas: Press a piece of glass (a microscope slide) upon the suspected spot. A hemorrhage is rendered more distinct, while the surrounding part becomes more anæmic. An inflammatory hyperæmia, on the other hand, disappears.

Cause. Hemorrhages may be due to affections of the blood or disease of the bloodvessels. They occur in the course of blood diseases, because such change in the quality of the blood takes place that permits diapedesis more readily. They are more particularly, but not exclusively, seen in dependent parts, especially in the lower extremities.

Significance. While subcutaneous hemorrhages are easily recognized, their diagnostic significance is more difficult to determine, and must depend upon the phenomena with which they are associated. Moreover, the *situation* of the hemorrhage is in a measure an index of its causal origin; thus hemorrhages about joints are usually purpuric or hæmophilic.

1. Hemorrhage with Fever. Subcutaneous hemorrhages in the infections are due to changes in the quality of the blood, and indicate the severity of the infection, or to obstruction of the bloodvessels with emboli. To the former class belong *cerebro-spinal fever* and *measles*, *variola*, and *scarlatina*. In the exanthemata they precede, develop with, or even replace the characteristic eruption, the latter being darker in color than normal. Hemorrhages will probably take place at the same time from the mucous membranes; perhaps the nares will be occluded, and the mouth and fauces filled with clotted blood. In milder infections sordes collect in the mouth only. They indicate the degree of malignancy of these affections. To the same class of affections belong *epidemic hæmoglobinuria* and *morbus maculosus neonatorum*, diseases of newborn infants but little understood, although no doubt of an infectious nature. To these may be added the severe forms of *purpura hemorrhagica*, attended by fever, marked visceral disturbances, skin eruptions, and great œdema.

Hemorrhages due to obstruction of the vessels are known as hemorrhagic infarcts, and are seen in *pyæmia* and *ulcerative endocarditis*. The hemorrhages are small, sometimes elevated, more abundant on the extremities, but distributed over the trunk; they are seen as small areas in the mucous membranes, observed in the conjunctivæ, and, on ophthalmoscopic examination, in the retina. The association of chill, fever, and sweat, the presence of pus in some structures of the body, and the characteristic joint affections point to pyæmia. On the other hand, if due to ulcerative endocarditis, the physical signs of this affection render the recognition of the cause of the hemorrhage clear. Finally, in *rheumatic fever* with involvement of the joints we have the occurrence of *purpura*. (See Erythema, same chapter.)

2. Hemorrhage with Anæmia. Hemorrhages occur in all forms of *anæmia* attended by debility. In idiopathic or pernicious anæmia

they are usually small, but may become extensive. They occur on the extremities, and, usually, on the dorsum of the feet or hands. There may also be retinal hemorrhages. They are also seen in the secondary anæmias that arise in the later stages of tuberculosis and of carcinoma, particularly of the stomach; in the later stages of Bright's disease, and of cirrhosis of the liver.

Scurvy is an affection characterized by anæmia, debility, and wasting, in which there are hemorrhages under the skin as well as from the mucous surfaces. The gums are particularly affected. They bleed easily. Hemorrhages also occur in the deep lymphatic spaces, in the muscles, underneath the periosteum, and in the joints. In *scurvy-rickets* similar hemorrhages are seen. (See Chapter XIII.)

3. Purpura. *Primary purpura* occurs without any known cause. It has been divided, for convenience, into simple and hemorrhagic purpura, though the two probably differ only in intensity.

Secondary purpura occurs in connection with a variety of febrile and constitutional diseases: 1. Scurvy. 2. Hæmophilia. 3. Hodgkin's disease. 4. Splenic leucocythæmia. 5. Pernicious anæmia. 6. Chronic lesions of the kidney and liver, with or without jaundice. 7. Ulcerative endocarditis. 8. Malignant sarcomata. 9. Infectious diseases.

A. In *simple purpura* the hemorrhages are limited to the skin. They consist of: 1. Bright-red spots, varying in size from a pin-head to a silver three-cent piece. These spots are under the skin and are unaffected by pressure. They fade gradually from red to yellow and disappear. 2. Larger spots or streaks called vibices. 3. Ecchymoses.

The disease is said to be most common about the age of puberty. It may come on in the midst of apparent health, or it may follow an illness, as typhoid fever.

Purpura occurs especially upon the legs, the standing position seeming to favor its occurrence. It comes on in successive crops. Sometimes large blebs, filled with thin blood, form under the skin, and gangrene at times occurs.

B. In the *hemorrhagic form*[1] hemorrhages occur from the nose, stomach, bowels, vagina, and bronchi, or into the kidney or other viscus. Cutaneous and submucous hemorrhages also occur.

The onset of these cases is sudden, though there may be a day or two of depression, lassitude, headache, and nausea. The first symptom noticed is generally fever, which is apt to be moderate, then eruption upon the skin is detected, and for a day or two the patient may seem to be only slightly ailing. Copious epistaxis may now occur, or a hæmatemesis or hæmaturia, or all of these and other hemorrhages, may occur the same day. The temperature may be only moderately raised, or it may reach 104° to 105°, or even a higher point. The pulse at first is frequent (120 to 140), but of good volume and tension. Subsequently, in unfavorable cases, it becomes thready and very frequent. Respiration is not affected, and the mind is clear; the face is pale and

[1] See Grave Forms of Purpura Hæmorrhagica. Musser: Trans. Association of American Physicians, vol. vi.

anxious. Hemorrhage may also occur into the choroid and brain-substance, with blindness and paralysis as sequels. It may also occur into the uvula or tonsil.

The *subjective symptoms* are pains in the loins, limbs, epigastrium, or chest. Often these pains announce a fresh hemorrhage, as into the kidney, or a fresh crop of purpuric spots. The degree of anæmia depends upon the copiousness of the hemorrhage and the length of time the disease lasts. Sometimes the hemorrhages cause great exhaustion, with a tendency to collapse.

The urine, in the case of hemorrhage into the kidney, of course contains blood; sometimes casts are also found.

C. Another variety of purpura is known as *peliosis rheumatica*, the peculiar features of which are tender and swollen joints, œdema of the subcutaneous cellular tissue, and purpura associated with urticarial wheals and intense itching (purpura urticans). The subcutaneous hemorrhages consist of petechiæ, vibices, and ecchymoses. There may be such large hemorrhages into the penis, scrotum, and uvula as to result in gangrene and slow separation of the dead tissue by ulceration. Epistaxis may occur, but copious hemorrhages from the stomach, the bowel, or into the kidney or other organs are rare. Endocarditis and pericarditis occur as complications in some cases. The duration is apt to be long, convalescence being delayed by repeated outbreaks of purpura with multiple arthritic symptoms and œdema.

Diagnosis. It is distinguished from *scurvy* by the absence of antecedent debility and anæmia, of spongy gums, of brawny induration in the limbs, and by the fact that the hemorrhages do not usually occur around a hair follicle. In scurvy there is a history of deprivation of vegetable food, whereas purpura may occur in the midst of robust health. As a rule, the cutaneous hemorrhages are larger in scurvy than in purpura.

It is distinguished from *acute infectious diseases*, particularly typhus, cerebro-spinal fever, and smallpox, by the absence of severe constitutional symptoms which characterize the graver forms of these diseases —in which alone a purpuric eruption is likely to be severe enough to cause doubt. Hemorrhages from mucous surfaces are rare in the latter.

Hæmophilia is distinguished by the history the patient gives of being a bleeder by heredity, and the fact that the bleeding has been started by some injury, wound, or operation.

It is distinguished from the hemorrhages of *leukæmia* by the absence of enlarged spleen and liver, and by the fact that there is no excess of leucocytes in the blood.

Malignant sarcoma causing hemorrhages is recognized by the previous history of anæmia and cachexia, and by the detection of primary or secondary growths.

It must not be confounded with *Raynaud's disease*, a vasomotor affection characterized by local syncope, local asphyxia, and gangrene.

4. Hæmophilia. The diagnostic significance of subcutaneous hemorrhage is clearer when associated with profuse hemorrhages in other portions of the body, and when there is also a history of the occur-

rence of such hemorrhages in the family. *Hæmophilia* is a constitutional affection characterized by bleeding, which is spontaneous or occurs upon slight injury. It is nearly always hereditary, but may arise *de novo*.

Males are very much more liable to it than females, the ratio being about 11 to 1. This curious disposition to bleeding may be transmitted for generations, and almost always to the males through the female members of the family—that is to say, the daughter of a bleeder is not usually affected, but she transmits the tendency to her sons, who become bleeders; so, too, the granddaughters are not bleeders, but they in turn transmit the disposition to their male offspring. It generally shows itself early in life, usually before the end of the second year, and almost invariably by puberty.

The affection usually first declares itself by the occurrence of a hemorrhage, either spontaneous or the result of slight injury, the bleeding being far more profuse than would be natural, and in some cases absolutely uncontrollable.

Legg[1] has divided hæmophilia into three degrees, according to the severity of the symptoms. The first is characterized by external and internal bleedings of every kind, and by joint-affections; the second, by spontaneous hemorrhages from mucous membranes, but no traumatic bleeding or ecchymoses, and no joint-affections; the third, by a tendency simply to ecchymoses. The first form is seen most frequently in men; the second most frequently in women; and the third in either sex.

The most frequent seat of hemorrhage is the nose, and the next the gastro-intestinal tract. The bleeding is from the capillaries; it may prove fatal in a few hours, or last for days and weeks with final recovery. Intense anæmia follows the prolonged hemorrhage, but the blood is replaced with remarkable rapidity. All operations, even the most trivial, are extremely dangerous in bleeders. Circumcision, extraction of teeth, and leeching are credited with the most deaths by Grandidier.

Joint-symptoms are very common. The knees, elbows, ankles, and shoulders are the ones most frequently involved. The attack may be marked by pain, redness, swelling, inflammation, and fever; or fever may be absent; or pain alone may be complained of. The attacks are liable to recur, especially in cold, damp weather, and may result in stiffened, deformed joints.

The *diagnosis* is easy when the history of a hereditary tendency to bleed can be obtained. Osler[2] properly remarks that slight joint-trouble and petechiæ are as much a manifestation of the disease as the more severe hemorrhages. In cases in which no history can be secured the diagnosis is made by noting a persistent liability to hemorrhage, without adequate cause, and associated with joint-affections.

Osler gives the following excellent summary of the affections with which hæmophilia can be confounded:

1. The umbilical hemorrhages of infants, due to jaundice or to syphilis, hæmorrhagica neonatorum, etc.

[1] Hæmophilia. London, 1892.
[2] Quoted by Osler, Pepper's System of Medicine, 1885, iii. 932.

2. Purpura simplex, often seen in debilitated, rarely in healthy children, usually confined to the legs, and in some cases associated with rheumatic pains or swellings in the knees and ankles.

3. Peliosis rheumatica.

4. Purpura hæmorrhagica, morbus maculosus Werlhofii, a grave disease, characterized by extensive cutaneous ecchymoses, mucous hemorrhages, but not dependent on any local disease, or, as far as known, on any specific poison.

5. Infective purpura due to the action of some specific poison—smallpox, measles, scarlet fever, cerebro-spinal fever, etc. The hemorrhages may be cutaneous and trivial, or may be in the most aggravated form of interstitial and mucous bleedings, as seen, for example, in black smallpox.

6. Toxic purpura, as in snake-bites and many poisons, such as phosphorus.

7. Simple hemorrhagic diathesis, under which may be included those cases in which, without any hereditary disposition or previous hemorrhagic history, there is a tendency to uncontrollable hemorrhage from a slight wound.

8. Hæmatidrosis, bloody sweats, which occur usually in hysterical or epileptic females, and are in rare instances accompanied by mucous hemorrhages.

5. Hemorrhage in Central Nervous Disease. Neuritis. Purpura in some instances is believed by Mitchell to be due to primary disease of the nervous system ; certainly we do see it in neuritis, in Raynaud's disease, in myelitis, and in locomotor ataxia. It may occur in hysteria, when drops of blood ooze through the skin at the time of the attack (hæmatidrosis).

6. Hemorrhage of Toxic Origin. The *virus* of snakes causes hemorrhages under the skin. In *jaundice* the blood is disintegrated and hemorrhages take place. In malignant types the mucous membrane bleeds and sordes collect on the tongue, lips, and gums. To the same class belong the subcutaneous hemorrhages that follow the administration of certain *drugs*, as copaiba, iodide of potassium, quinine, and belladonna. (See Medicinal Rashes.)

Eruptions.

Diseases of the skin are usually characterized by eruptions. Now, such eruptions may be primary and local (from causes operating directly on the skin) in the sense that they occur independently of any internal affection ; or secondary, the resultant of an internal morbid process. The morbid processes are the same, and morbid processes in the skin do not differ from such processes in other epithelial structures. The anatomical and physiological peculiarity of the part causes the difference in the phenomena. Hence anæmias and hyperæmias, inflammations, acute or chronic, with or without exudation ; hemorrhages, atrophies, and hypertrophies, new growths, and parasitic affections are found in both. But instead of a painless inflammation with transudation of mucus, as in mucous membrane inflammation, we have a more or less painful inflam-

mation, with itching (nerve-supply), and with sebaceous and sudoriferous gland exudation. Otherwise the same symptoms attend each; but ocular examination of the inner mucous membranes is not possible.

While the reader is referred to special works on skin diseases for a description of the primary or local skin affections, the secondary affections will be briefly noted. It must not be forgotten that the local affections—eczemas, parasitic disease, etc.—are modified by the general conditions or state of health of the patient.

Clinical Significance. This depends, first, upon the special character of the eruption, the nature of the lesion; second, its distribution (*a*) in the layers of the skin, (*b*) over the surface of the body; third, its association with other morbid phenomena or various circumstances.

I. The Nature of the Lesion. Observation concerning the nature of the lesion includes (1) its anatomical character, (2) the order of appearance, (3) its uniformity, and (4) the mode of invasion.

A knowledge of anatomical lesions is essential in order to be able to define exactly the morbid process and determine the primary cause of the lesion. For a long period of time the lesions were divided into primary and secondary. The lesions known as scabs, scale, raw surfaces, scratch-marks, and ulcers are always secondary. Scars and maculæ appear latest. The other lesions herein described are primary.

The writer follows Dr. Pye-Smith in the description of them, as well as in most of the matter appertaining to cutaneous affections.

1. *Hyperæmia, or congestion.*

a. Mere overfulness of the vessels from paralysis of the vasomotor nerves, with redness and heat, but without the exudation and tissue changes which accompany inflammation. This hyperæmic blush, readily produced in the physiological laboratory, is rarely seen as an uncomplicated morbid condition (*e. g.*, Trousseau's *tache-cérébrale*).

b. Active, arterial, or inflammatory hyperæmia, varying in color from brilliant scarlet to rose-pink, and combined with heat, tingling, or other sensations.

c. Passive, venous, or congestive hyperæmia, dependent upon retarded circulation and distended venules. The color is purple, bluish, or livid, the surface is cold, and there are no painful sensations.

2. *Pimple, or papule.* A small, solid elevation of the skin.

a. The acute inflammatory papule.

b. The chronic large inflammatory papule, discrete or confluent.

c. A solid non-inflammatory papule.

d. Solid elevations of the skin, which may be called false papules.

3. *Vesicle.* A visible cavity in the skin filled with transparent liquid.

4. *Pustule.* A cutaneous abscess.

5. *Bulla, or bleb.* A very large vesicle.

6. *Scab, or crust.* A dried-up concretion of the contents of a vesicle, pustule, or bleb.

7. *Scale* (*squama*). A dry flake of epidermic cells.

8. *Wheal* (*pomphos*). A flat, solid elevation of the skin, much larger than a papule, and of ephemeral duration.

9. *Scratch-mark.* An injury to the skin, of linear form and curved outline.

10. *Raw.* A surface which has lost its horny layer of epidermis.

11. *Chap* (*rima*). A crack or fissure which goes through the epidermis.

12. *Sore* (*ulcus*). The result of destruction by inflammation, which has reached below the Malpighian layer and has destroyed the papillæ.

13. *Scar* (*cicatrix*). The result of the healing process after an injury or disease deep enough to destroy the papillæ of the part.

14. *Nodule.* A solid elevation of the skin larger than a papule and seated in its deep layer.

15. *Stain* (*macula*). A patch of increased pigmentation of the skin.

16. *Hemorrhage* (*ecchymosis*). When a bloodvessel of the cutis vera gives way a dark-red or purple mark is produced, which (like the macula) does not disappear on pressure.

The recognition of the exact anatomical lesion is not sufficient for diagnosis unless the *mode of invasion* is observed at the same time. The rash often spreads from a single focus, or numerous foci appear and coalesce. The lesion is best studied in the most recent part. Not only is the mode of local invasion to be noted, but also the *uniformity* of the anatomical lesion. Often, instead of a simple lesion, various kinds are present at the same time, or they develop in successive order ; thus, in smallpox, we have first the papule, then the vesicle, and finally the pustule.

II. Distribution. The location of the lesion in the various layers of the skin, and the distribution over the surface of the body, must be observed. *The layers of skin :* (1) The horny layer of the epidermis manifests the pathological changes of hypertrophy, atrophy, dryness, or desquamation of the cuticle. Dead scales result, in addition to the hypertrophies and atrophies indicated in the outline. (2) The eruption in a large number of cases is limited to the living Malpighian layer of the epidermis and to the papillary layer of the cutis. The hyperæmias (erythemata), and inflammations of all kinds, are confined to these layers. In this situation they never leave scars. (3) The deep layer of the cutis is so intimately connected with the subcutaneous tissue that morbid changes in it involve the latter, and even extend more deeply. The affections are more severe, but less numerous than affections of the superficial layers, and are always followed by cicatrices. The changes in the sweat glands, sebaceous glands, hair, and nails, so far as they refer to internal medicine, have been treated in another section.

Area of distribution : The distribution of the eruption over different areas of the body is of great importance in the diagnosis of the various erythemata due to exanthems and to morbid conditions of the gastro-intestinal tract. It will be noted more in detail when the specific eruptions are considered. The student should also bear in mind the relationship of eruptions or cutaneous changes of nutrition (trophic disorders) to the affected nerve-supplies.

III. Associate Morbid Phenomena. The student of internal medicine should particularly observe the associated morbid phenomena, or concomitant circumstances, in order to determine the nature of the skin affection, which may be the expression of internal disorder. The

associated morbid phenomena of diagnostic significance are *fever, jaundice, albuminuria,* and the phenomena of past or present *syphilitic* disease, *tuberculosis, rheumatism,* or the rheumatic habit. The presence of one of these processes or diseases points to particular affections. Thus, a large number of eruptions is attended with fever; another group is of frequent occurrence in the course of rheumatism; another class belongs to syphilis, while a fourth class is associated with anæmia, jaundice, or albuminuria. This subdivision is not based on the nature of the eruption but on its association with other phenomena. It will be learned later that all the groups belong to the *hemorrhages* or the *erythemata.* The true relationship of the two classes of phenomena can be fully ascertained only by inquiry into the history and course of the eruption and, in addition, into the concomitant phenomena. Thus, if the eruption is thought to be due to the exanthemata, the period of incubation, mode of infection, symptoms of the invasion, and the progress of the attack must be inquired into.

General Symptoms. In order to determine accurately the cause of an eruption and appreciate its diagnostic significance, the general health must be inquired into, the condition of the stomach and bowels and the character of the urine must be ascertained. It must be remembered that local skin disorders are influenced, for good or ill, by the general health. Functional disorders of the stomach and bowels are a frequent source of many of the erythemas, while in diabetes pruritus and forms of dermatitis are of common occurrence. The latter are also observed in Bright's disease. The cause for the eruption is the same in both, in all probability—that is, a perverted secretion of the skin, or, if œdema is present, impaired nutrition of the surface.

The subjective symptoms are of great importance in the attempt to ascertain the true nature of an eruption. Pain, itching, burning, smarting, and tenderness are significant of the inflammations. Pain due to inflammation is constant and smarting, burning or throbbing in character. Sometimes, however, pains of a neuralgic character, intermittent and distributed in the course of nerve-trunks, precede the development of eruption. This is seen in herpes zoster. *Itching* is an important symptom in disease of the skin. It is not present in the eruption due to the exanthemata generally, except in smallpox, chickenpox, and rubella. Its absence is a striking peculiarity of the eruptions of syphilis; but in erythema, especially if associated with œdema, it is a most annoying symptom. In other skin diseases, as eczema, psoriasis, and the parasitic affections, it is much more common and of extreme annoyance.

Itching may be present without any anatomical evidence of skin disease. It is seen in the troublesome *pruritus* that occurs in the aged, particularly about the intestinal and genito-urinary orifices, symptomatic of affections of the organs related thereto. It is a symptom which should lead to an examination of the urine, as diabetes is sometimes found to be the fundamental source of the complaint. It has been previously noted that itching occurs to a high degree in jaundice. It is also due to the internal administration of drugs, as opium and morphine, and sometimes quinine.

In addition to the associate pathological phenomena which should be ascertained in the study of skin eruptions, in order to determine their relationship to internal affections, other circumstances should be inquired into, such as the occupation, the character of the clothing, degree of cleanliness of the patient; the effects of climate, the season, temperature, and the state of the air.

The following very concise outline, taken from the work of the above-named author, to whom the writer is indebted for much of the data of this section, is here given to enable the student to appreciate more thoroughly the pathological relations of the various skin diseases. The table also shows at once the relation of the eruptions to the internal disorders which concern us more particularly in this work:

DISEASES OF THE SKIN REGARDED AS PHYSIOLOGICAL PROCESSES.

(*Pathological Arrangement.*)

Acute Inflammations.—Diffuse, *e. g.*, scarlatina, morbilli, syphilis, roseola (eruptive fevers, erythema).

With venous congestion—Erythema nodosum (rheumatism).

With œdema—Urticaria, erythema nodosum (gastro-intestinal disorder and rheumatism).

With necrosis—Furunculus, anthrax (diabetes).

Localized in papules—Enterica (erythemata), syphilis, eczema, prurigo.

Localized in vesicles—Eczema, zona, variola, scabies, herpes, varicella (eruptive fevers, infectious diseases).

Localized in pustules—Impetigo, variola, scabies, syphilis, sycosis, acne.

Localized in blebs—Pemphigus, scabies, rupia.

Desquamating during involution—Scarlatina, etc.

Chronic Inflammations.—With venous congestion—Acne rosacea, pernio.

With over-production of epidermis—Psoriasis, pityriasis rubra.

With œdema—Elephantiasis.

With fatty degeneration—Xanthelasma.

With hypertrophy—Elephantiasis.

With cicatrization—Cheloid.

With ulceration—Lupus, syphilis, lepra.

New growths—Xanthelasma, lupus, lepra, syphilis, cancer.

Atrophy—The senile skin, linæ gravidarum.

Hypertrophy—Ichthyosis, cornu cutaneum, clavis, verruca.

Hemorrhage—Traumatic (*e. g.*, flea bites), typhus, scurvy.

Pigmentation—Syphilitic maculæ, melasma, chloasma, icterus, ephelis.

Congenital malformations—Ichthyosis, cutaneous nævus.

Neurosis—Pruritus (diabetes, jaundice).

Anomalies of Secretion.—Increased, diminished, or perverted—Seborrhœa, xeroderma, hyperidrosis, anidrosis, chromidrosis, etc. Obstructed—Comedo, milium, acne, sudamina.

A glance at the above outline will show that the eruptions which particularly concern us belong to the class of diseases to which the term *erythema* is applied.

Erythema. CLASSIFICATION. Erythemata may be divided, in accordance with the classification of Kaposi, into acute, contagious, exudative dermatoses, represented by measles, scarlatina, rubella, and

smallpox; and the acute, non-contagious, inflammatory dermatoses, which may be further subdivided into: (1) typical forms, idiopathic and toxic, including urticaria, or nettle-rash; (2) varieties of herpes; (3) erythemas due to boils, colds, or erysipelas. The first group of the *non*-contagious form includes the class which should always be considered in connection with the diagnosis of fevers. The skin inflammations closely simulate in their symptoms the eruptive fevers, even to the affections of the mucous membranes. Besnier has named them the *pseudo-exanthems*, and divides them into rubeloids and scarlatinoids. Both simulate eruptive fevers throughout their course, and hence both are acute and febrile. The scarlatiniform erythemas are febrile at the beginning, subacute in course, but of longer duration than the fever they simulate. They are the most common forms, and arise from infectious diseases, such as puerperal fever, septicæmia, and gonorrhœa, or from toxæmia due to drugs or articles of food.

Character of eruption in the non-contagious forms. The erythemata are characterized by (*a*) rose rash with injection of the surface, either (*b*) with general œdema, or with circumscribed local œdema, forming wheals or with papules. In rare cases bullæ are also formed. (*c*) The rash is followed by a branny desquamation. (*d*) The exudation that attends the lesion is always watery, in contradistinction to the sero-purulent or purulent exudation of eczema and scabies. Sometimes slight hemorrhages attend the lesion, as in cases of purpura or of urticaria. (*e*) The course of the erythema is of diagnostic significance. It begins quickly, and is usually attended with febrile symptoms, sometimes mild, again very intense. (*f*) The duration is short; at least it is not indefinite. The erythemas that are recurrent must not be considered to be one process of long duration. (*g*) The locality of the erythema is not of precise diagnostic significance. The eruption is usually symmetrical, and the favorite localities may be defined as the extensor surfaces of the forearms and leg, the face, cheeks, neck, and the chest and abdomen. True erythema does not attack the scalp, the flexures of the joints, the palms (except erythema multiforme), nor the soles. (*h*) The local symptoms that attend erythemata are mild. Local tenderness is more marked than in eczema. Smarting and tingling are complained of, but severe pain and excessive itching are rare. Only when wheals are present do we find pruritus. The rash of erythema does not spread. Patches occasionally unite, but an affected area never enlarges its borders.

The etiology of erythema is involved in obscurity. Although the frequent associate phenomena are not of etiological, they are certainly of diagnostic significance. We may have them occur under the following circumstances: 1. In one class the eruption is symptomatic, depending upon dyspepsia or upon rheumatic fever. 2. In the eruptive fevers, especially scarlatina and measles, in enteric fever and cholera, and in syphilis, there is an early erythema preceding the later true eruption. 3. The most striking instance of the relationship to internal disorder is seen in the rash that arises after the administration of medicine, as copaiba, or after the taking of certain foods. 4. The erythemata occur most commonly in children and young people.

They are very frequent in men. The age at which they occur coincides with that of rheumatism.

Varieties of non-contagious erythemata: First, *erythema multiforme* in simple form, with papules or with exudation; it may disappear in a few hours, or persist for a day or two and form rings (*erythema fugax* or *erythema annulatum*). With the fading of the redness faint desquamation ensues, and there may be a few pigment marks. The annular form is observed in rheumatic fever. In addition to rheumatism erythema multiforme may be found associated with the following affections: Typhoid fever, puerperal fever, gonorrhœa, cholera, infectious endocarditis and osteomyelitis, syphilis, leprosy, vaccination, and surgical septicæmia. Osler has called attention to the visceral complications of erythema exudativa multiforme associated with the skin lesions—viz., gastro-intestinal crises, endocarditis, pericarditis, acute nephritis, and hemorrhage from the mucous surfaces. Arthritis is also seen in some instances. The skin lesions range from simple purpura to local œdema, and from urticaria to large infiltrating hemorrhages of the skin and subcutaneous tissues. The gastro-intestinal crises are attended by colic, with vomiting and diarrhœa.

Erythema læve often appears upon the tense skin of dropsical parts. It may be the result of acupuncture.

Vesicular and Bullous Erythema. To this class belong the affections known as herpes and erythema bullosum.

Herpes zoster is seen in the cutaneous distribution of one or more nerves. It consists of vesicles of flattened form, ranged in clusters of twenty or thirty, lying on a reddened, slightly swollen bed of skin. The number of clusters varies from one to ten. The vesicles develop in quick succession, beginning usually near the roots of the nerve whose branches they follow. A short papular stage precedes the vesicles, and some of the vesicles abort. The eruption tends to dry up in five or six days. The crusts form in yellowish or brownish clusters, which fall off in the third week, leaving purple stains.

When the disease attacks the face it follows the course of the fifth nerve. The several twigs of the trifacial are traced out from their points of emergence from the bony canals. Great swelling of the eyelids sometimes takes place on account of the loose tissue, so that the lesion may be mistaken for erysipelas. Ulceration of the cornea and iris sometimes occurs, and, when lower divisions of the trifacial are affected, vesicles may appear in the mucous membrane of the mouth and palate. The cervical nerves and those of the upper extremity are also affected in their distribution. The eruption on the arm rarely goes below the elbow. When the second and third intercostal nerves are affected the intercostohumeral branch produces an eruption down the inner side of the arm. The eruption occurs frequently on the trunk. Following the course of the dorsal nerves it slants downward as it approaches the pubes.

In the distribution of the disease in the lower limbs the eruption rarely extends below the knee or buttocks. It follows the course of the external cutaneous or anterior crural nerves, or that of the small sciatic. Some of the branches of the sacral nerves are also affected.

The disease is unilateral, and its precise limitation to one-half of the body is of the greatest diagnostic significance.

While fever or general symptoms do not usually attend its course in any marked degree, insomnia and depression are likely to occur, probably on account of the severe neuralgic pain. Pain is the most important subjective symptom. It is localized in the nerves, in the distribution of which the eruption takes place. It is not so likely to be present in the young. The pain may precede the eruption by several days, and persist long after the eruption subsides. This is particularly the case in old people.

Herpes labialis, or facialis, consists of vesicles arranged in groups or clusters upon an inflamed surface. They appear very suddenly upon the upper lid or the alæ of the nose, sometimes on the cheek or chin, and they may appear inside the mouth. They undergo some changes, as in herpes zoster, but are not attended by severe neuralgic pain. They are also symptomatic of an internal disorder, an acute catarrh (cold), or follow a rigor, as in intermittent fever or pneumonia. They may be present in epidemic cerebro-spinal meningitis, but are never present in tuberculous meningitis. Diagnosis of the former disease is confirmed by their presence (Klemperer). Herpes iris and herpes preputialis have no diagnostic significance of internal disease.

Erythema Nodosum. With the erythema there is great œdema. The spots are somewhat painful and tender, but do not itch. The redness of the erythema is modified by the hue of venous congestion. Small hemorrhages may be seen. The patches develop on the legs, their long diameter being parallel to the tibia. They rise slowly into hard masses. They may be seen on the ankles or the calf, and sometimes on the ulna. They occur frequently in those who have suffered from rheumatic fever.

Urticaria is a form of erythema in which wheals, sometimes surrounded by an erythematous blush, are seen. It is an acute inflammatory œdema of the cutis. The serous exudation fills the lymph-spaces and expels blood from the venules. It takes place suddenly, and may be excited by chemical irritation or a mechanical irritant, as the finger drawn across the skin. Small patches, or large white areas, are seen, due to the coalescence of smaller ones (giant urticaria). All parts of the body may be affected, except the scalp, face, and soles of the feet. The eruption is not symmetrical. Its course may be acute, or it may be chronic and transitory, characterized by successive attacks. It is the form of erythema in which intense itching is the most pronounced symptom. There are no other subjective symptoms. The itching causes restlessness and loss of sleep. Urticaria is symptomatic of gastric or intestinal disturbance, or the ingestion of drugs or poisons. Another form follows the tapping of a hydatid cyst. It occurs sometimes in women at each menstrual period, and may be traced to ovarian disorder. It may occur after severe shock to the nervous system, with high fever. It is not an infrequent complication of rheumatic fever. It occurs in men and women equally, but is most frequent in children and adolescents.

Medicinal Rashes. To the erythemata belong most of the so-called medicinal rashes.

The following drugs are known to cause erythema: potassium bromide and iodide, copaiba, cubebs, the essential oils, capsicum, santonin, chloral, opium, morphine, antipyrin, salicylic acid and its compounds, iodoform, belladonna and atropine, tar, carbolic acid, arsenic, cannabis indica, digitalis, mercury, silver, copper, and antitoxin.

Belladonna produces in susceptible persons, or when administered in poisonous doses, a diffuse, bright-red erythema, closely resembling that of scarlet fever, but without the darker red points which interrupt the latter. *Atropine* also produces in some persons, especially on the shoulders, arms, chest, and face, an eruption of disseminated, small, hard vesico-papules, showing no tendency to pustulation. They are seated on an inflammatory base, but are more superficial than acne.

The bromides produce a characteristic pustular eruption which is most intense upon the shoulders, face, chest, and arms. Large doses, or long-continued administration, are generally required to bring it out. It is conspicuous upon the face of some epileptics.

The iodides produce an eruption which is not often pustular, but an erythematous or papular rash is not uncommon. It appears chiefly about the forearms, face, and neck. Vesicles, bullæ, and purpuric spots are also occasionally seen.

The eruption produced by *quinine* is generally erythematous, and is attended with itching and burning; the face and neck are attacked first.

Opium and its alkaloid also produce, in susceptible persons, an erythematous scarlatinoid eruption which is accompanied by intense itching. Itching, especially about the nose, is much more common without eruption.

Copaiba produces a vesico-papular or papular eruption which resembles urticaria and erythema multiforme. It is itchy. It is more apt to be seen on the extremities. It may be purpuric.

The eruption of *cubebs* is a diffused erythema, with millet-sized papules, coalescent here and there. Unlike the eruption of copaiba, it is more copious over the face and trunk than over the extremities.

Antipyrin causes a measles-like or urticaria-like eruption.

Erythemata of Infectious Diseases.

The inflammations of the skin which are symptomatic of a specific infection are also of an erythematous variety. The term *exanthemata* has been applied to the latter, but the eruptions of typhus and typhoid (enterica) belong to the same class. The characteristics and distinctions of the various forms will be described in sections devoted to the respective diseases. The student should remember the associate general phenomena, particularly fever, the onset and the course of which should be carefully observed.

Roseola. Roseola is of a deep rose-color, not arranged in crescentic patches, as in measles, nor scarlet and capable of being resolved into innumerable red points, as in scarlatina. It is not so diffuse as the

latter. It precedes smallpox, scarlatina, measles, cholera, typhoid fever, syphilis, diphtheria, and malaria. In smallpox, in cases of cholera, and after parturition and surgical operations, the rash is copious, but is characterized by being seated over the lower half of the abdomen and the anterior and inner aspects of the thighs. It may appear elsewhere, but is usually confined to that portion of the body.

The erythema of roseola may be mistaken for rubella, measles, or scarlatina. The following are points of distinction: First, it is neither contagious nor epidemic; second, there are no prodromal symptoms; third, the rash does not come out after a definite period of fever; fourth, it is not confined to any special locality; fifth, the fever is of short duration and moderate degree, rarely above 101°; sixth, there is no catarrhal discharge from the eyes or nose or in the pharynx; the fauces and palate are reddened without swelling; seventh, it is not seen in the mouth, like the eruptions of measles or scarlatina; eighth, if present, the fever which precedes the eruption is of only a few hours' duration (in scarlatina it lasts twenty-four hours, in measles seventy-two hours); ninth, the rash is not crescentic as in measles, nor punctiform as in scarlatina, though it must be admitted that severe cases of the affection cannot be easily diagnosticated, the development of the sequelæ alone concluding the diagnosis.

To add to the confusion, an erythema called roseola often precedes the eruption of a particular fever. The association with this class of fevers has been indicated before.

Sufficient reference has been made to the erythemata that attend *rheumatism*. A few other internal (infectious) disorders are associated with the development of an eruption. In *cholera*, during the period of reaction, a rose rash which may resemble erythema, urticaria, or scarlatina appears coincidently with a rise of temperature. It is most frequently seen on the forearms and backs of the hands, but may cover the back and limbs. It may be slightly hemorrhagic and last two or three days. A slight desquamation usually follows. In *influenza* a roseolous eruption, covering the trunks and limbs and becoming papular, is seen in rare cases.

In addition, erythematous eruptions are sometimes seen in the course of *Bright's disease*. Two forms, quite distinct from the previously mentioned erythema læve, are observed: the roseola on the feet, legs, and hands—rarely on the chest and abdomen; and the papular form on the thighs, arms, and shoulders. Itching and other subjective symptoms do not attend the eruption. A form with desquamation may begin on the limbs. These erythemata are common in the later stages of Bright's disease, but are not of ill omen. In acute Bright's disease a transient roseola is observed very rarely; so also is purpura. If there is much anasarca in tubal nephritis, erythema is more common. The eruptions usually appear independently of uræmic symptoms, and disappear during their continuance. They are in all probability allied with the inflammation which attacks the lungs and serous membranes in Bright's disease.

Sudamina. Here may be mentioned another eruption, or condition of skin, common in the course of internal diseases. *Sudamina*, or

miliaria, are small, clear vesicles seen in large numbers, usually on the abdomen, but also on any other part which reflects the light strongly. They are seen during and after the subsidence of profuse sweats. While actual perspiration is seen on the forehead, the trunk may appear free from moisture. When the hand is placed over it, as on the abdomen, the dryness is noted, but at the same time a roughened, nutmeg-grater-like sensation is felt. On close inspection this is observed to be due to the eruption just mentioned. The vesicles are usually of good prognostic omen in the course of febrile diseases, particularly typhoid fever. They are due to the accumulation of perspiration under the epidermis.

General Diagnosis of Skin Affections.

(*Condensed from* PYE-SMITH.)

I. Factitious Eruptions. We must never forget the possibility of the affection before us being artificial. All kinds of dermatites, eczema, erysipelas, pemphigus, impetigo, may be simulated by the application of various irritants. Pigmentation also has often been imitated with success. Such artificial lesions will generally be found upon the arms, rarely on the face, and scarcely ever beyond reach of the patient's hands. Mustard, cantharides, and some other irritants can be distinguished with the aid of the microscope.

II. Traumatic Eruptions. In all cases of dermatitis we should seek for the irritant, and sometimes it is so directly the cause of the disease that the eczema or impetigo in question may be considered purely traumatic, and efficient treatment immediately follows accurate diagnosis : *sublata causa tollitur effectus.*

Pediculi in the hair should be carefully looked for in all cases of impetigo in children ; pediculi vestimentorum in prurigo of old people. The acarus of scabies, fleas, bugs, and gnats may be found. In adults, pediculi pubis may sometimes be found in the axillæ as well as in their proper region, and when they have been destroyed by mercurial ointment the patient is at once relieved from pruritus.

Frequently the irritant must be sought for in the objects which the patient habitually handles. The coarser kinds of brown sugar are a frequent cause of eczema of the hands (grocer's itch). So with many of the " chemicals " used in a variety of modern handicrafts. Constant washing of the hands in washerwomen, in scrubbers, in potmen, and many others produces eczema rimosum. The heat of the sun is the cause of eczema solare and ephelides ; the heat of the fire, of the pigment spots on the shins of elderly people. Sweat, again, is a very common irritant, producing the erythema which usually accompanies sudamina and also intertrigo of opposed surfaces. Scratching, as a cause of traumatic dermatitis, has been repeatedly referred to.

III. Febrile Rashes. We must never forget that a cutaneous eruption may possibly be part of an acute exanthem. The use of a clinical thermometer is a great help in this respect. Variola is frequently mistaken for syphilis and other affections.

IV. Medicinal Rashes. Other cases are due to certain kinds of food or to drugs. They have been described above.

V. Syphilodermata. When we have satisfied ourselves that the eruption before us is not factitious, nor directly traumatic, nor a symptomatic eruption, we may next consider whether or not it is due to syphilis. In this inquiry it is undesirable to ask questions the answers to which are as apt to mislead as to guide aright.

1. We should first consider the *color* of the affected skin, remembering, however, that the pigmentation which gives the so-called coppery or raw-ham tint to a syphilitic eruption is the same which is sooner or later produced by all forms of dermatitis. Psoriasis, chronic eczema, lichen planus, and prurigo may all produce shades which bear the closest resemblance to syphiloderma.

2. The lesions of syphilis are *multiform*. It is rare in any but syphilitic affections to find mere hyperæmia in one part and associated pustules, papules, scales, or ulcers in others; and it is not often that a syphilitic eruption exhibits only a single elementary lesion.

A pustular eruption in an adult should always suggest the question of syphilis when that of scabies has been answered in the negative.

3. Syphilitic eruptions, for some unknown reason, *do not itch*—the exceptions to this rule are remarkably few; they usually occur during the stage of scabbing of pustular rashes or during the healing of tertiary ulcers. An ordinary secondary syphilide may, however, as a rare exception, be so irritating that wheals and scratch-marks are present. On the other hand, psoriasis is often free from irritation, while the degree of itching of eczema, and even of scabies and prurigo, varies greatly.

4. The local *distribution* of syphilitic disease is a great aid in diagnosis. Specific eruptions are certainly not, as a rule, symmetrical; the early roseolous rash is only so because it is general, and therefore, upon a surface like the human body, more or less symmetrical. Moreover, as it chiefly affects the face, chest, and trunk generally, it is near the middle line. But we do not see symmetrical patches of syphilide in corresponding parts of both sides of the face, both sides of the trunk, or the right and left limbs. In all but the earliest syphilides the affected patches are very decidedly and constantly unsymmetrical, irregularly scattered over head, trunk, and limbs, and chiefly remarkable for having no well-marked seats of predilection.

The forehead, especially about the roots of the hair, is, however, very frequently the seat both of the early and middle erythematous, scaly, and pustular syphilides, and the palms of the hands and soles of the feet are frequently symmetrically affected with the later scaly eruption.

Practically, when we find a disease of the skin occupying some unusual position, we should at least consider the question of syphilitic origin.

5. These signs, alone or in combination, serve to distinguish early specific roseola from erythema, eczema, scarlatina, and measles, and the later eruptions from eczema, lichen, impetigo, and psoriasis.

The eruptions of *congenital syphilis* which are most liable to be mistaken are: The so-called pemphigus of infants, which is known by its

affecting the palms and soles; rupia, which, by the form of the crusts and the ulcerated surface beneath, may always be distinguished from impetigo; an erythematous rash of the nates and genitals of infants, which is distinguished from eczema of the same parts, also common at that age, by its coppery color, its blotchy distribution, and more clearly defined margin.

The *tertiary ulcers* of syphilis are distinguished by their presence in unusual places, by their punched-out edges, circular or so-called horseshoe shape, and by the fact that they usually give little pain or discomfort.

Tertiary ulcers have no predilection for the outer side of the leg, but, inasmuch as the part above the inner malleolus is, from anatomical causes, the chosen seat of varicose ulcers, most ulcers in the first position will be syphilitic and in the latter not. Moreover, the age helps in the diagnosis, as varicose ulcers rarely occur before the fortieth year.

Most ulcers on the arms are found to be tertiary syphilitic ulcers.

VI. Tineæ. The next group of skin diseases includes those which are due to vegetable parasites—tinea versicolor of the trunk, eczema marginatum of the perineum and thighs, tinea circinata of the neck and other parts, tinea sycosis of the chin, and tinea tonsurans of the scalp. In all doubtful cases the microscope should be employed.

Tinea of the scalp is rare in adults, and tinea circinata still more so; tinea marginata occurs only in adult males.

VII. Primary Superficial Inflammations. To distinguish the superficial from the deeper kinds of dermatitis, we should notice whether the cutis alone is infiltrated and thickened, or whether it is bound down by adhesions to the subcutaneous tissues. The presence of scars, however slight, is a proof that the process has gone deeper than the papillæ, and has more or less extensively destroyed the papillary layer. Superficial inflammations, excluding those due to acarus, to pediculi, and to other direct irritants, and excluding also those which are the result of vegetable parasites and of syphilis, fall, with respect to their treatment, into three large groups:

The first group, represented by impetigo and most forms of eczema, consists of inflammations which are subacute, and accompanied with burning, itching, and pain, sometimes with a slight degree of fever.

The second group of superficial inflammations of the skin is typically represented by psoriasis, but includes lichen planus, the more chronic, dry, and obstinate forms of eczema, and true prurigo. These affections are chronic, with little irritation, exudation, pain, or active signs.

The third group is that of erythemata.

VIII. The Acne Group. Acne, both in its pathology and etiology, differs from other forms of dermatitis. The age of the patient and its distribution are sufficient for diagnosis. It is at once a superficial and a deep dermatitis, and is often followed by scars. Its treatment consists entirely, or almost entirely, in local applications directed to the correction of the sebaceous affection. With acne may be classed sycosis and furunculus.

IX. Deep Affections. When we have ascertained that the affection of the skin is deep, that is to say, that it goes below the papillary layer, the field of diagnosis is limited.

Excluding erysipelas, which is distinguished by its acute character and febrile symptoms, excluding the pustular affections which affect the skin deeply and produce scars only at isolated points, such as acne, variola, and herpes zoster, and excluding, thirdly, leprosy and other exotic diseases, we have to distinguish in the great majority of cases which come before us in this country—first, traumatic and varicose ulcers; second, gummata and syphilitic ulcers; third, lupus; fourth, rodent ulcer; and fifth, carcinoma of the skin.

With regard to the first of these, we must not assume, because a sore upon the skin is said to be the result of a blow or a kick, that it is purely traumatic, for syphilitic ulcers often arise in this way. Malignant ulcers are rare, and are usually obvious from the age of the patient, the pain they occasion, their tumid margins, and their blood-stained secretions. Moreover, they are, with few exceptions, confined to the neighborhood of the orifices of the body, especially the lower lip, the urethra, the vulva, and the anus. Rodent ulcer, however, is very difficult to diagnose with certainty. Its locality, its slow and painless progress, and its belonging to the latter half of life, usually serve to distinguish it from lupus; and its being single, excessively chronic, and unaccompanied by nodes or other syphilitic lesions, are the best characteristics for diagnosis from a tertiary ulcer.

The Nutrition of the Skin.

Palpation. The color, as determined by inspection, is a fair index of the nutrition of the skin, but further information is obtained by palpation. In *health* the skin is smooth, firm, and elastic. When pinched between the thumb and fingers and then allowed to escape, it slips quickly back into its former position. When pressed or squeezed, it becomes pale from expression of blood, but resumes its natural hue immediately.

The readiness with which the blood returns after pressure shows the character of the capillary circulation of the skin. This is active in health and sluggish in serious disease of the lungs, heart, and blood-vessels. In the eruptive fevers, especially in measles, scarlet fever, and smallpox, sluggish capillary circulation with dusky eruption is a grave sign. In measles it is usually due to pulmonary complications, and in other infectious diseases to the overwhelming effects of the poison.

As age advances the skin becomes less elastic, and in old persons may lie in wrinkles. When pinched between the fingers the skin is more inclined to remain wrinkled. Fat persons whose skin is firm and hard are in much better condition than those whose skin is loose and flabby. The latter condition is frequently met with in babies, particularly those that are fed on artificial foods. When the skin is thin and dry and loses its tone, so that, when pinched into folds, it resumes its smoothness but slowly and sluggishly, it is usually evidence, in a person under fifty, of some grave cachexia, as carcinoma.

Moisture and Dryness of the Skin. Moisture and dryness are in one sense correlated with the nutrition of the skin. It is quite certain that when the skin is abnormally dry its nutrition is impaired.

In health the skin is not perceptibly moist, except as the result of physical exertion or under heat, or as the immediate result of imbibing a hot fluid or a sudorific drug. There is considerable individual difference, however, within the limits of the normal. Rheumatic and strumous persons may have a perceptibly moist and oily skin at all times, while others have a skin which perspires very little, even under influences which usually bring about perspiration.

Perspiration Increased. *Hyperidrosis.* It may be general or local.

A. General increased perspiration is seen—1. With fever. It occurs in the course of *rheumatism,* when the sweats are strong in odor and acid in reaction. It is seen in *tuberculosis,* especially the miliary variety. It is sometimes marked throughout cases of *typhoid fever.* General perspiration also attends the violent muscular action of *tetanus,* but is not seen in *epilepsy.* An example of general sweating is seen in that curious affection to which the term "*sweating sickness*" has been applied. It is a fever the nature of which is not well known, but in which this symptom is most pronounced. Sweating is extreme in *trichinosis.*

2. With normal or subnormal temperature. *a. Sudden, temporary perspiration.* Sweats occur from excitement or slight exertion in patients during *convalescence.* A general profuse perspiration may be of short duration and occur suddenly after fright or shock in health. It is the characteristic perspiration of *collapse.* The forehead is covered with sweat, large drops stand out on the face, the hands and feet are moist or wet with perspiration, and the whole surface of the body "leaks." It is attended by a cold and clammy skin. In the collapse of all forms of shock, or after hemorrhage or profuse discharge, as in cholera, this form of perspiration is seen.

More striking still are the perspirations that suddenly break out in the course of acute disease coincidently with a fall of temperature. We have (1) the critical sweats of pneumonia and relapsing fever; (2) sweats which terminate a paroxysm of intermitting fever, whether of malarial or infectious origin (see Fever); (3) the profuse perspiration that attends pyæmia, breaking out with each fall of temperature to disappear as it rises; (4) the night-sweats that attend tuberculosis and other exhausting diseases. In tuberculosis and in pus-formation or accumulation the oscillation of temperature, with or without chills, followed by sweating, is known as *hectic.* Sudden breaking out of general perspiration, but more notably seen on the face, attends dyspnœa of pulmonary origin and the attacks of dyspnœa in the course of organic heart disease. These perspirations are at times the result of an effort at elimination, on the part of the skin, to relieve the kidneys or bowels, such as the perspiration of *uræmia,* which is attended by a urinous odor. At times it may also occur in jaundice. In the conditions just mentioned there are coolness of the skin and cold extremities.

b. Prolonged Perspiration. In exhausting diseases, general and persistent perspiration may occur, particularly in the later stages, as in tuberculosis, and in any disease attended by persistent dyspnœa.

B. Local increased perspiration (*hyperidrosis localis*) occurs when there is local vasomotor paresis. Thus, in organic diseases of the brain and in affections of the peripheral nerves, in some forms of neuralgia, in migraine and in hysteria, it has been observed. Sometimes one side of the body alone is affected, even in a malarial paroxysm (hemidrosis).

Local sweats are sometimes significant. This is the case particularly with a sweat confined to the head, which occurs usually in children, and is one of the striking characteristics of rickets. With the local sweating the patient rolls his head at night from discomfort. The hair on the back of the head is rubbed off.

Unilateral sweating of the head may arise from destructive pressure on the sympathetic nerves, causing paralysis of the dilator fibres of the cilio-spinal branches, in thoracic aneurism, and in caries of the lower cervical vertebræ. There are usually contraction of the pupil and congestion of the face on the same side.

Diminished Perspiration. *Anidrosis.* The skin is abnormally dry in the early stages of acute disease attended by fever, particularly if the febrile rise takes place suddenly, as in acute digestive disorders of children. In adults, when the disease is accompanied by high fever, as in thermic fever, the skin is dry. In the first day of the eruption of the exanthemata the dryness is marked. Dryness of the skin is of frequent occurrence when there are copious discharges of water from the bowels or the kidneys. In choleraic diarrhœa the dryness occurs suddenly. In some affections, as diabetes and Bright's disease, the dryness extends over a long period of time, and is frequently attended by eruptions or desquamations and by the formation of boils. When there are accumulations of serum in the lymph-spaces of the subcutaneous connective tissue, or changes in the connective tissue, as in dystrophies or myxœdema, or scleroderma, the skin is dry because of the stretching and pressure on the bloodvessels.

Scars. Scars are important proofs of the occurrence of previous disease, especially smallpox, chickenpox, and syphilis. Scars of the first two occur in the form of circular pits, and almost always on the face. Scars of syphilis are larger, circular, or oval in shape, and seen usually to the best advantage on the extremities, but the single scar on the forehead is strikingly suggestive. Scars upon the legs in persons under thirty years of age, when not traumatic, are almost always syphilitic. Scars as the result of suppurating glands are seen most frequently in the neck, but may be found wherever there are glands, especially under the jaw and in the axilla and groin. They are most liable to occur in tuberculous persons, either spontaneously or as the result of the exanthemata, erysipelas, or other infectious disease. When such scars are met with in a person with incipient tuberculosis the prognosis becomes more anxious.

The appearance of the scar indicates its age in a general way, and hence throws light upon the patient's previous history, and also serves as a check upon the accuracy of his statements.

Scars the result of wounds, injuries, or operations may be seen any-

where ; they are of importance only so far as they may furnish a clue to the cause of existing disease. Of such nature are the scars upon the head in cases of brain disease, particularly epilepsy.

The scars of pregnancy, the striæ seen upon the lower part of the abdomen and the upper part of the thigh, must not be confounded with similar scars that occur in great œdema, and which are sometimes found in fat persons. They are also seen after typhoid fever.

CHAPTER XI.

THE DATA OBTAINED BY OBSERVATION—(*Continued*).

The subcutaneous connective tissue. Œdema—causes—mode of recognition—situation—feet, face, arms, and head—œdema of trichinosis—angioneurotic œdema. Myxœdema. Connective tissue dystrophies. Scleroderma. Sarcomata—cysticercus cellulosæ—brawny induration. Subcutaneous nodules. The *lymphatic glands.* Enlargements—local—general. *Adenitis. Hodgkin's disease.* Tuberculosis and leucæmia.

THE SUBCUTANEOUS CONNECTIVE TISSUE AND LYMPHATIC GLANDS.

Enlargements or swellings of the subcutaneous connective tissue, other than the skin tumors and papular eruptions, on any portion of the surface of the body, are due to some change in the tissue or the structure or organs directly underneath the swollen part. Œdema, myxœdema, subcutaneous emphysema, dystrophies, scleroderma, brawny induration, and local subcutaneous swellings are the principal ones to be considered.

Œdema; Dropsy.

The lymph-spaces of the subcutaneous connective tissue become overdistended with serum, causing an accumulation to which the general term *dropsy* is applied. If the accumulation is local and confined to small areas it is known as *œdema.* If it is general, and if, in addition, the large lymph-cavities, the pleura, the peritoneum, and the pericardium contain fluid, it is known as *anasarca.* Accumulation occurs because more fluid is poured out by the vessels than can be removed by the lymphatics and veins. This may depend either upon obstruction of the veins and lymphatics, or excessive exudation from the bloodvessels, or both. The former condition, however, is rare, and usually local, because, unless the obstruction is very great, the veins and lymphatics are able to carry away more fluid than is effused from the capillaries.

1. Excess of fluid transudes when there is *local capillary change* from inflammation or the effects of poisons. The change must be in the capillaries. It was thought that this general process was of an inflammatory nature, but at present it is believed to be due to the influence of poisons, probably absorbed from the intestinal canal, altering the nutrition of the capillary vessels. Thus, the œdema and general dropsy of albuminuria, particularly in the early stage of that affection, are thought to be due to a poison circulating in the blood which also causes the nephritis. Mahomed found a pre-albuminuric

stage of scarlet fever, in which he noted a peculiar reaction of the urine, which gave a blue color with guaiac. A brisk purgative administered when this reaction was noticed would prevent the occurrence of albuminuria, whereas if the drug was withheld albuminuria always followed. The purgative removed the poison which caused the nephritis and œdema.

It is well known that in urticaria there is marked local œdema. Brunton thinks that some poisons circulating in the blood cause paralysis of the secreting power of the sweat-glands, on account of which there is not only effusion from the bloodvessels, but at the same time such changes in the secreting-cells take place as to produce an acid, the local irritative action of which, upon the capillaries, causes a further transudation of fluid. That acids circulating in the blood have the power of creating œdema, the experiments of Cash and Brunton fully demonstrate. While, therefore, in the œdema of Bright's disease in its earliest stage and in urticaria we have this explanation of the phenomena, other factors are causal in other forms of œdema.

2. Increased transudation and obstruction to the flow of lymph are the causes of some forms of œdema. It may be of local origin, as in the œdema over the site of an inflammation or the œdema of an arm or leg from venous occlusion, or it may be of general origin, as in cardiac disease. The obstruction may be in the lymphatics or in the veins. In the former it may occur (*a*) from want of muscular action ; (*b*) from want of inspiratory action of the thorax ; (*c*) diminution of the diastolic suction of the heart ; (*d*) positive pressure on the veins. In the latter, obstruction of the veins is caused by conditions similar to those affecting the lymphatics, and arises from (*a*) want of muscular action ; (*b*) want of movement of the thorax ; and (*c*) feeble action of the heart ; and, in addition, it is likely to be caused by (*d*) complete arrest of blood-flow from external pressure upon the vein or from plugging of the vein. It can readily be seen, with a little knowledge of physiology, how the above factors favor the development of œdema due to disease of the heart and to venous obstruction. The baneful factors are those which retard the flow of blood, preventing its return to the right heart. Hence it is called the œdema of passive congestion.

3. A third form of œdema, usually slight, is that which is seen in anæmia. Several factors combine to produce it : (*a*) the watery condition of the blood ; (*b*) the condition of the capillaries ; and (*c*) vasomotor paresis on account of imperfect nutrition of the vasomotor centres. It may be diffused, as in the anasarca that attends the anæmia of malaria.

4. Œdema may be of nervous origin. Such is the œdema that occurs in diseases or injuries of nerves. To it possibly belongs the œdema of beri-beri. It may be a trophoneurosis with secondary alterations in the permeability of the vascular walls, or it may be due to vasomotor paralysis.

Mode of Recognition. Whether the accumulation is in local areas or distends the entire subcutaneous tissue, the œdema is not difficult of recognition. The part is swollen and puffy, the surface is pale, smooth, and shiny, the temperature is usually low, and the affected area pits

on pressure. Pitting is more pronounced if the finger is pressed over a part which is seated upon a firm background, as bone. Œdema of the ankle or over the tibia is more readily recognized than œdema in the calves.

The œdema obliterates normal depressions and increases the rotundity of the affected part. It causes deformity, as of the face and neck or of the penis, when the accumulation of serum is considerable. The swelling appears in the most dependent parts if the œdema is diffuse or the cause is general, as in cardiac disease; or in parts made up of loose connective tissue, as the eyelids or scrotum. The temporary *disappearance* of the œdema, either entirely or from one part, to appear in another, is a prominent feature of it. It will disappear between morning and evening, or its position will alter with change in the position of the body. The presence of a previously existing œdema can often be told by the scars or striæ that resulted from overstretching of the skin, as of the abdomen and thighs.

Œdema is to be distinguished from—(1) Inflammatory swellings, by the absence of the classical signs of inflammation: pain, heat, and redness. (2) The enlargement of myxœdema differs from œdema by the absence of pitting on pressure, the occurrence of induration, which resists the pressure of the finger, and by the occurrence of anæsthesia or analgesia. (3) The swellings of connective-tissue dystrophies are hard, localized areas that do not pit on pressure, and are not seated in dependent parts of the body. They are found on the arm, for instance, or on the thigh, or about the flanks and in the axillæ. (4) The swelling of subcutaneous emphysema differs from œdema in that it arises in the course of some disease of the air-passages, and, on palpation, the crackling sensation of air under the finger is distinctly felt, while there is no pitting on pressure. In the cases that the writer has seen the parts were particularly tender, although pain in subcutaneous emphysema is said usually to be absent.

Diagnostic Significance. The value of œdema as a diagnostic sign depends upon its location, its mode of development, and its association with disease of other organs or structures of the body.

Location. The œdema may be limited to small areas, as the eyelids, the face, or the feet, or to an arm or leg; it may involve an arm and leg of the same side; or it may involve the extremities and trunk and even include the face. We therefore have local and general œdema.

Local Œdema. Local œdema occurs when there is pressure on a vein or occlusion of it by a thrombus. Œdema of the arm from pressure on the veins by enlarged lymphatic glands in the axilla, and œdema of the leg from thrombosis of the femoral vein, are examples of this form of local œdema. Dropsy of an arm often occurs when the patient has laid upon it. Local œdema also occurs over the seat of inflammation, and is a valuable diagnostic sign. It is an indication of suppuration. It is known as "inflammatory" or "collateral œdema." It is due to obstruction of the lymph circulation. It is seen over the mastoid when its cells are the seat of inflammation; over the parotid gland under the same circumstances; over parts of the thorax in empyema; over the præcordia in purulent pericarditis; over the surface

of the liver in some cases of hepatic abscess; in the abdominal parietes in purulent peritonitis, but more marked over the primary focus of inflammation, as the gall-bladder region or the region of the appendix.

The Arms and Thorax. Another form of local œdema occurs when there is pressure upon the superior vena cava from aneurism or disease of the mediastinal glands. The œdema is then limited to the arms, head, neck, and thorax. Such œdema is usually associated with cyanosis of the hands and arms. There is also marked distention of the veins of the upper parts of the body. The œdema has been found, in a few instances, to be more marked on one side than on the other. This has occurred in cases of aneurism which communicated with the vena cava. Either the collateral circulation on one side had been established or pressure was greater on the left innominate vein. The œdema is sometimes limited to the *head* and *arms*. If the obstruction of the superior cava is situated below the entrance of the azygos vein the chest shares in the venous congestion and resulting œdema. If, on the other hand, the obstruction is above the azygos vein there is no œdema of the chest-wall. This form of œdema, as a rule, is easily recognized by the presence of the above-mentioned symptoms, with other pressure-symptoms, due to disease of the mediastinum and by the results of physical examination, which reveals the presence of a tumor in the thorax. It usually develops slowly, hand-in-hand with the other symptoms. At times, however, it occurs suddenly. *Sudden œdema* in this situation is always due to an aneurism which has ruptured into the vena cava (see above). The sudden onset is attended by physical signs of aneurism, or, if they are not present, by a murmur characteristic of the communication between an artery and a vein. It must be confessed that often the physical signs are not precise and the murmur is absent. The *suddenness* of the peculiar localized œdema is the chief point of diagnosis in favor of this rare form of aneurism.

The Œdema of Trichinosis. (See Face.) Œdema of the skin over the affected muscles, as well as of the face, occurs in trichinosis. It begins early in the disease, disappears after a few days, to return again later. It is localized over the muscles, and is associated with the growth of trichinæ in them. It is distinguished from cardiac and renal dropsy by its course and situation as well as by the fact that the scrotum and labia majora are never œdematous.

The *cause* of the above forms of œdema is local and in close proximity to or in intimate anatomical relation with the dropsical swelling. But the cause of local œdema may be central, or in a sense general. It then develops gradually and begins in special localities, as in the *feet* or *face*.

The Feet. Œdema of the feet or ankles is usually due to disturbance of the circulation. It arises in heart disease, or in the course of any exhausting and debilitating disease in which the heart has become weakened. The organic change which takes place in the heart-muscle (dilatation) in the course of *obstructive valvular disease* and in *lung disease* is often attended by œdema of the feet. Later a general dropsy may ensue. But œdema of the feet may occur from another cause—*i. e.*, *anæmia*. In all forms of this affection puffiness of the ankles may

be seen. An explanation of the cause has been given. Similar localized œdema in individuals of relaxed fibre occurs in the evening after a day of considerable physical exertion. Œdema of the feet, subsequently becoming diffuse, occurs in beri-beri.

Œdema of the Face. Œdema may begin or remain localized in the face, and is very striking. (See Face and Eyelids.) It may be limited to the eyelids, as a simple puffiness, or may spread over the entire face, causing complete obscuration of the normal outlines. It is the œdema of *renal* disease, and differs from œdema of the feet in

Fig. 19.

Face of a patient with general anasarca due to chronic parenchymatous nephritis. (Hare.)

that it is more marked in the morning on rising and disappears toward night. Of all forms of local œdema it is the most grave, and should at once call attention to the condition of the urine, particularly if the patient has just had an attack of scarlatina, or if it occurs in a woman who is pregnant.

The diagnostic significance of primary local œdema may be summarized as follows: (1) *Eyelids* or eyes ("Bright" eye, "tear that does not fall") in nephritis; (2) *face*, nephritis; (3) *forehead*, trichinosis; (4) *head*, pressure upon superior vena cava above the azygos vein; (5) one side of *head*, pressure upon innominate vein; (6) *head* and *arms*, or *head*, *arms*, and *thorax*, pressure upon superior vena cava; (7) one *arm*, pressure upon axillary veins; (8) one *leg*, pressure upon femoral vein; (9) both *feet* or *legs*, pressure upon inferior vena cava by abdominal tumor, loss of vasomotor tone, heart disease, anæmia, late nephritis; (10) the *loins*, "lumbar cushion," nephritis, cardiac disease if patient is in recumbent posture; (11) the *scrotum*, nephritis and cardiac disease;

(12) local œdemas over inflammations of structures underneath, as bones, the gall-bladder, the appendix, the pleura, peritoneum, or pericardium.

General Œdema. *Anasarca.* General anasarca is due to heart or to kidney disease in most of the cases. Œdema of the face and feet may become general. In cases in which the face is first œdematous its extension may be very rapid, so that twenty-four to forty-eight hours after the swelling is noticed the whole body is in a state of anasarca. *Renal* disease. The extension of œdema, primarily seated in the feet and legs (*cardiac* dropsy), throughout the rest of the body is more gradual, and develops with other signs and symptoms of weakness of the heart. Hence cyanosis gradually appears. This may be seen first in the extremities. Finally the face and lips take on the peculiar hue. On the other hand, in the general anasarca that follows the local œdema of the face in Bright's disease, pallor occurs, and as the œdema increases it becomes more and more of a waxy hue, while the extremities beome glistening or shining in appearance. In the so-called " wet form " of beri-beri general œdema comes on rapidly.

Angioneurotic Œdema. This curious affection is not of frequent occurrence. It may be present in the individuals of several generations of a family. The attack comes on suddenly. The swelling is circumscribed. It may appear on the face, on the brow, the lips, or cheek. The eyelid is a common situation. It may also occur on the backs of the hands, the legs, or in the throat. It remains but a short time and disappears as quickly as it came on. The outbreaks have exhibited distinct periodicity. Local symptoms of itching, heat, or redness, or general urticaria, may precede the swelling. The sudden swelling causes great deformity. If the upper lip is affected, the mouth cannot be opened ; if the hands, the fingers cannot be bent. In the hereditary cases the attack recurs every three or four weeks. The danger to life is from œdema of the larynx, which caused death in two of Osler's cases. The general symptoms that attend the attack are gastro-intestinal. Nausea and vomiting occur, followed by severe colic.

It must not be confounded with simple urticaria, or the giant form of that affection, with which it may, however, have close affinities. It is regarded by Quincke as a vasomotor neurosis, which leads to impairment of the permeability of the vessels.

Recapitulation. From what has been said the student will observe that œdema may be local or general ; that local œdema may be unilateral or bilateral ; that œdema may be further subdivided, in accordance with the cause, into inflammatory dropsy, œdema or dropsy of passive congestion, hydræmic dropsy, and vasomotor dropsy. The forms of passive dropsies just indicated may be subdivided into cardiac dropsy, hepatic dropsy, and renal dropsy, according to anatomical causes.

While the account of œdema just given refers more particularly to the subcutaneous accumulation of serum, the same pathology and etiology apply to accumulations in the large lymph-cavities, and hence, in addition to general œdema, we may have *ascites*, *hydropericardium*, *hydrothorax*, *hydrocele*, and *effusion* in the joints. The methods of

recognition of dropsy of the larger cavities will be deferred until diseases associated with these particular regions are discussed. It must be remembered that œdema or accumulations of serum in cavities may be of local or general origin.

It must not be forgotten that two or more causes may combine to produce a dropsy, or that a dropsy of one cause may for a time be dependent upon a second and even a more pronounced factor later on in the development of the disease. Thus (*a*) the dropsy of hydræmia may be aggravated by that of (*b*) weak heart which arises from anæmia, to which may be added later the dropsy of vasomotor paresis. The dropsy in Bright's disease is due to (*a*) capillary changes produced by a poison circulating in the blood, and (*b*), later, to the condition of the heart if, as is frequently the case, it undergoes dilatation.

Myxœdema.

Enlargement of the surface of the body, local or general, is also seen in myxœdema, a condition which simulates dropsy, as already stated. In myxœdema the swelling is general. The face is involved. The

FIG. 20.

A typical case of myxœdema. (STARR.)

arms are more markedly swollen, however, than the fingers; the legs more than the feet. Usually the swelling of the legs and arms is irregular. In some cases supraclavicular paddings are marked. These

paddings must not be confounded with the *pseudo-lipomata*, described by Verneuil, occurring in these situations. The swelling is due to the infiltration of mucin into the connective tissue, and arises from some affection of the thyroid gland. The gland is absent, functionally or actually. The hard, indurating, non-pitting swelling is associated with striking change in the appearance of the face, particularly the nose and forehead. The nose becomes thickened, the forehead more prominent and overhanging. The outline of the face is rounded, so that the term "full-moon" is applied to it. The skin is thickened, dry, and rough, somewhat translucent in appearance, pale or yellow in color, and of a doughy consistence, but with a moderate degree of elasticity. The perspiration is diminished. The hands change in shape, they become square or spade-shaped, and the fingers clubbed. The appendages of the skin change. The nails become brittle and distorted, the hair dry, harsh, and brittle, and it may fall out. With these remarkable changes in the exterior marked nervous and mental symptoms arise. Speech is thick and hesitating, the memory feeble. The intellect is dull and irresponsive, the temper irritable. Sensibility is impaired, particularly the loss of sensation to pain. Patients have been burned without their knowledge. This happened in one of the writer's cases. Abnormal sensations of heat and chilliness are complained of, as well as other paræsthesias. The patient is anæmic, the temperature is subnormal, the heart's action weak, the respiration sluggish. Breathlessness on slight exertion is pronounced, and exertion itself is very difficult, while there is a greater sense of fatigue than the exertion and the condition of the organs would warrant. The muscularity is enfeebled. There are impairment of appetite, indigestion, and flatulency. The urine may become albuminous, but for a long time is not characteristic save in amount and specific gravity. The former is increased, the latter lowered.

As the case advances mental and physical failure become more pronounced, the patient is subject to hallucination, and is extremely irritable. Stupor sets in; death may take place in coma or from uræmia. It is a disease of mature life, and occurs most frequently in women.

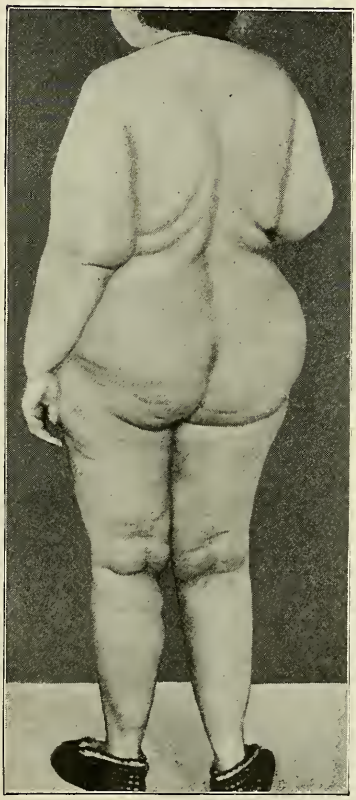
The following varieties are seen: (1) Spontaneous myxœdema of the adult; (2) infantile myxœdema; (3) operative myxœdema; and (4) endemic myxœdema or cretinism. In infantile myxœdema the functions of the thyroid body are suppressed during the period of the development of the individual. Typical cases justify the name of myxœdematous idiocy.

Subcutaneous Emphysema.

Enlargement or swelling of the surface, either local or general, may occur on account of air underneath the skin. The skin is pale and quite distended, and hence depressions are filled up, as the axillary, clavicular, and intercostal spaces. The primary seat of the swelling is in close proximity to the air-passages, and occurs because of communication between them and the subcutaneous connective tissue. It may occur in ulcerations of the upper passages, as the larynx or

trachea; in ulcerations of the œsophagus into the mediastinum; in the ulceration and rupture of phthisical cavities into the chest-wall; and in rupture of the lungs from hard coughing, sharp crying, severe exertions, such as blowing of wind instruments. The air may escape under the pleura to the mediastinum and thence to the neck, or, when the pleura is adherent, air will pass from the lung into the connective tissue. The swelling gradually spreads over the entire body from the seat of rupture or in close proximity to it. In a case of laryngeal phthisis under the writer's care it encircled the neck and spread uniformly over the anterior and posterior portion of the thorax. Thence it extended downward until it met a corresponding infiltration of the lymph-spaces in the thighs, due to serum. The distinction between œdematous swelling and subcutaneous emphysema could thus be made: the latter offered no resistance, did not pit on pressure, crackled under the finger, and was quite tender on pressure. Spontaneous pain was not present; but any position was painful in which the weight of the body pressed upon the part affected.

FIG. 21.



Note accumulations on back and on extremities. See knees and elbows; wrists and ankles unusually small. Patient aged 56. Second attack of insanity. (Original.)

Connective-tissue Dystrophies.

Enlargements of the surface are seen in the so-called dystrophies. The dystrophy is usually due to a localized anomalous overgrowth of connective tissue, probably of trophic origin. It can easily be distinguished from œdema by the absence of the signs of œdema, or from local inflammatory swelling by the absence of pain, heat, and redness. The swelling occurs on the arms and legs, usually on the outer aspects, and may occur in various portions of the trunk. In one of the writer's cases the swellings were periodical; or, rather, the persistent swellings increased in size at irregular intervals.

Dercum and Henry have described cases of dystrophy in which the enlargements had been attributed to accumulations of fat. The patients presented marked subjective nervous phenomena, paræsthesias of all kinds, with flushings and sensations of sinking and depression. There were areas of anæsthesia, pain, and tenderness in the nerve-trunks. Pain preceded the advent of the swellings.

Herpes zoster occurred in Dercum's case, and other symptoms of neuritis were marked. The irregularity in the distribution of the swellings, their character and mode of development, the occurrence of neuritis, and the absence of perspiration, distinguished dystrophy from lipomatosis or excess of fat. The patients were of a neurotic type, and mental impairment usually resulted in the course of the disease. The general nutrition failed, particularly as gastro-intestinal disorders ensued.

Scleroderma.

Scleroderma is a hyperplasia of the subcutaneous connective tissue with swelling and induration. It is brawny. As the tissues are almost immovable, the term "hide-bound" is applied to this condition There are marked stiffness and also pain.

In *localized* scleroderma, or *morphœa*, the skin has a waxy or dead-white appearance, is brawny and inelastic. There may be preliminary hyperæmia of the skin. Subsequently pigmentation of the hyperæmic area takes place, causing changes in color, or the pigment may atrophy, causing *leucoderma*. The secretion of sweat is diminished or entirely abolished. In the *diffused* form the affection begins in the extremities or face, and is accompanied by a sense of stiffness or tension ; the skin is usually hard and firm, and gradually a diffuse, brawny induration develops. The skin cannot be picked up in folds. It may appear normal, but is generally very smooth, glossy, and dryer than usual, rarely pigmented. Scleroderma may be confined to a limb or may become universal. The appearance of the face is characteristic. It is expressionless, and the lips cannot be moved, while mastication is impossible ; the eyes and the nose are deformed ; the hands become fixed and the fingers immobile and contracted, on account of induration about the joints, the deformity being called *sclerodactyle*. It is thought to be due to a trophoneurosis, or to fibrosis of the arteries of the skin, with connective-tissue overgrowth in the adjacent areas.

Brawny Induration.

Œdema must not be confounded with the brawny induration of the calves of the legs in scurvy, probably from deep-seated hemorrhage. It must be remembered, however, that œdema of the ankles is very common in this affection. Brawny induration may also be found in syphilis. In a patient recently under the writer's care, in the Presbyterian Hospital, a brawny induration of the thigh, with painless swelling and stiffness of the leg, appeared to be due to syphilis. It disappeared rapidly under treatment with potassium iodide.

Localized Subcutaneous Nodules.

Sarcomata. The subcutaneous nodules seen in these affections are rarely, if ever, confounded with œdema or other swellings. In sarcoma the subcutaneous tumor becomes attached to the skin and may change its color. It is usually secondary to sarcoma in some other

organ of the body. When primary, or secondary to organs in which there is normal pigmentation, as the eye, they become blue or bluish-black. On palpation the surface is found to be rough and uneven if the tumors are numerous.

Primary *melanotic sarcomata* of the skin can always be distinguished by their color. In both forms of sarcomata the general symptoms of this affection daily become more and more pronounced, and subcutaneous hemorrhages are commonly associated with the local phenomena.

The first external evidence of *lymphosarcoma* may be subcutaneous nodules in unusual situations. Thus, in a case under my observation, a lymphoid nodule was first observed in the third interspace on the right side. Subsequently the glandular involvement followed.

Carcinomata. Subcutaneous lymphatic glands may be the seat of secondary carcinoma, and from their location may indicate the primary source of the disease. The glands above the left clavicle are sometimes secondarily affected in cancer of the stomach. In similar diseases of abdominal organs glands in the abdominal wall are enlarged. The subcutaneous nodules should be removed and examined microscopically. The structures of the umbilicus (skin and subcutaneous tissues) enlarge, become nodulated, and sometimes the seat of fungoid ulceration in abdominal carcinoma, particularly of the stomach. It must not be forgotten that primary sarcoma or carcinoma of the skin, limited to one area, and simulating an intra-abdominal growth, may occur, as in a case under my care in the Philadelphia Hospital, operated on by Horwitz.

Cysticercus Cellulosæ. The nature of the subcutaneous nodules of cysticercus are recognized by microscopic examination. They are usually associated with the larvæ in other tissues, hence the patient complains of great soreness and stiffness, and may become helpless.

Rheumatic Nodules. Subcutaneous nodules are seen in rheumatic patients in the course of the disease, or after the attacks. They are common in the young. They are particularly frequent in cases of rheumatic endocarditis. They may occur independently of the articular symptoms. They may occur in large numbers, and vary in size from a small shot to a large pea. They are of fibrous structure. They are attached to the tendons and fasciæ, particularly on the fingers, hands, and wrists, but may be found over the elbows, knees, the scapulæ, and the spines of the vertebræ.

Syphilitic Nodes. Gummata are observed in the tertiary periods of syphilis. They must not be confounded with the enlarged glands. They are attached to the skin, and may from time to time ulcerate. They may be seen on the back or buttocks; less frequently on other parts.

The Lymphatic Glands.

Information of diagnostic value may be obtained from the condition of the lymphatic glands. (See Chapter VII.) Enlargement may be general or local.

Enlargement of the cervical glands, and of the axillary and inguinal glands attended by fever, occurs in that obscure infection described by

Dawson Williams and others called glandular fever. Similar glandular enlargement is quite characteristic of German measles or rötheln. (See the Infections.)

Enlargement of the *post-cervical* glands, the *epitrochlear* glands, and lymphatic glands in other portions of the body points to syphilis. In the two first-mentioned localities the enlargement is of great diagnostic importance, as it is less likely to be due to any other causes. Suppurating glands do not here concern us.

Inguinal and Axillary Enlargement. With or without suppuration, enlargement always points to an irritation or lymphatic invasion in the area drained by the affected lymphatic gland. When in the groins the feet are affected, and when in the axillæ the hands. Great enlargement in either situation causes œdema of the corresponding extremity if the veins are pressed upon. The axillary glands are early affected and enlarged in mammary cancer. The breast should always be examined in œdema of the arm.

The Supraclavicular Glands. These glands are often enlarged and indurated, and may cause pressure-symptoms. The only local enlargement that is of special diagnostic significance is that which is seen above the clavicle on the left side. They often point to carcinoma of the stomach, as Troisier announced.[1] Indeed, there are cases of this disease in which only the general symptoms of carcinoma are present. Local symptoms are wanting and the locality of the cancer cannot be made out by the symptoms. The enlarged glands above the clavicle are a fair indication that the stomach is the seat of the disease. The enlargement is probably due to transmission of the infection along the thoracic duct and its lodgement in the associated glands.

The Cervical and Submaxillary Glands. Enlargement of the submaxillary and cervical glands points to affections of the mouth and throat or of the jaw and teeth. It is caused particularly by infectious disorders in these localities. They are often the seat of nodular enlargement in *actinomycosis*. (See "collar" in adenitis of leukæmia.)

Scars at the site of former glands point to tuberculous destruction or former bubo, and are suggestive.

The glands are enlarged in simple *adenitis*, *tuberculosis*, *Hodgkin's disease*, *leucocythæmia*, *sarcoma*, and *cancer*. The moderate enlargement of syphilis and the local enlargement from irritation in the area of lymph-drainage have been mentioned. *Adenitis* is usually local. The gland is tender and the connective tissue around it is affected. There are local heat and pain. At first the gland is hard, later it softens in the centre, and finally it exhibits fluctuation. In *tuberculosis* more than one gland is affected. Usually the glandular involvement is bilateral (as in the neck). At first the glands are isolated. Later they become matted. The local symptoms are not marked and the process is very indolent. Thick, cheesy pus is discharged which may contain tubercle bacilli. It causes tuberculosis when inoculated in lower animals—a method of diagnosis necessary to be resorted to frequently. The tuberculin test must be used. Fever and "decline"

[1] Bulletin et Memoires de la Société Médicale des Hôpitaux, January 13, 1888.

occur later, but often not until other structures, as the lungs, are infected. (See Leucocythæmia.)

Lympho Sarcoma is an infection of the glandular structures of obscure origin. A local group of glands may be involved or the glands throughout the body may be the seat of the overgrowth. When the infection is general the deep-seated glands, as the mediastinal and retroperitoneal, may be the first involved. Anæmia, fever, and signs of intrathoracic and abdominal pressure may be present without decisive indications of the nature of the disease. In a short time, however, a superficial gland may enlarge, and from thence rapidly other glands be involved. The occurrence of an enlarged gland in any part of the body may be suggestive of the nature of a deep-seated process. Positive diagnosis can be established, and the method should be resorted to by removal of the gland and its examination microscopically. A case of this character seen with Hare showed the first evidence of glandular infection in the enlargement of a small gland over the third interspace on the right side of the chest in front.

Hodgkin's Disease.

Hodgkin's disease (pseudoleukæmia, lymphadenoma, or lymphatic anæmia) is characterized by enlargement of the lymphatic glands and other adenoid tissue; by progressive oligocythæmia without, in most cases, much increase of leucocytes; and by the development of lymphatic tumors in unusual situations.

FIG. 22.

Hodgkin's disease. Glands in right axilla and neck much enlarged.

The disease is most frequent in the first half of life, three-fourths of the cases being in males.

The first symptom noted is enlargement of the glands of the neck; but sometimes the inguinal, less frequently the axillary glands, are first enlarged; rarely the tonsils are the first to be affected. The enlargement is painless and progressive, appearing first on one side of the neck and extending under the jaw to the opposite side. The tumors at first are distinct and movable under the skin. The swollen glands may remain in this condition indefinitely for months or years; but eventually they begin to enlarge very rapidly, lose their separate identity, and coalesce into large masses. Other glands in remote parts, as the axilla and groin, retroperitoneum, and arm, are affected. They

may be soft and fluctuating, or very dense and hard, but heat, tenderness, suppuration, and other evidences of inflammation are absent.

The spleen becomes very much enlarged, but rarely attains the dimensions common in leucocythæmia.

Other adenoid tissue in the intestine, tonsil, and posterior nares, and even the thymus, may enlarge and give rise to pressure symptoms.

Fever is a very constant symptom, but the type is not constant. The onset of the disease may be marked by fever and constitutional symptoms, and the glandular enlargement appears later. On the other hand, in three cases reported by J. Dreschfeld,[1] all the patients enjoyed good health and were able to follow their work until a few weeks before death. In all symptoms appeared suddenly, and consisted of pain, weakness, pallor, loss of appetite, and pyrexia.

Coincident with the rapid and extensive enlargement of the glands, anæmia becomes pronounced and is accompanied by the usual symptoms. Cough is often associated with anæmic dyspnœa, and in women menstruation may cease.

Along with the general symptoms there are numerous local ones, due to the pressure or impairment of function—cerebral anæmia from pressure on the carotids; cerebral congestion from pressure on the veins of the neck; disturbance of the heart from pressure on the pneumogastric; deafness; difficulty in deglutition and mastication; and pleural, peritoneal, and pericardial effusions.

The most frequent complications are nephritis, fatty degeneration of the heart, pleurisy, and, less frequently, pneumonia and pericarditis.

The *duration* of the disease is from six to eighteen months. Two-thirds of fifty fatal cases referred to by Gowers[2] ended in less than two years. It is difficult to determine accurately the beginning of the disease; sometimes a long period of latency follows the early glandular swelling; sometimes a general anæmia precedes any noticeable swelling of the glands; and sometimes the disease runs an acute course, ending fatally in two or three months.

Death results most frequently from exhaustion; but pressure upon the trachea producing asphyxia is not uncommon, and death has occurred from starvation, the result of occlusion by pressure of the œsophagus. The complications already mentioned are the immediate causes of death in other cases.

The *diagnosis* is not difficult with blood examination. By this means leucocythæmia is excluded. It may be distinguished from tuberculosis in the early stages when local by the site of the enlargement. In the former the submaxillary glands are involved; in the latter the glands in the anterior and posterior cervical triangles. The *tuberculin* test is required, as insisted upon by Otis, to establish tuberculous adenitis.

Lymphangitis or Angioleucitis. The streaked redness over the surface of the skin, with tenderness along the course of the lymphatics and œdema, is characteristic of inflammation of the lymphatic vessels, and need not be further mentioned. The glandular and dermal changes

[1] British Medical Journal, April 30, 1892.
[2] Reynolds' System of Medicine, Philadelphia, 1880, vol. iii. 549.

of *elephantiasis*, with chyluria, with or without lymph scrotum, are unmistakable ; the disease is due to the *filaria sanguinis hominis*.

Lymphatism. Poor physical development has recently been observed with lymphatic overgrowth, or the *constitutio lymphatico*. In this state sudden death is liable to occur. It is believed that one of the causes of death from anæsthesia and from antitoxin of diphtheria is a condition known as *status lymphaticus*. Hyperplasia of the lymphatic glands, the spleen, the thymus, and the bone marrow are rarely found in patients with rhachitis, and in hypoplasia of the heart and aorta. The internal lymphatic glands and the lymphatic structures of the alimentary tract are more frequently involved than the more external glands. With this overgrowth of lymph-tissue the spleen and the thymus gland are enlarged, and red marrow replaces the yellow marrow in young adults. The hypoplasia of the vascular system is not easily recognized. The left ventricle may be dilated and the peripheral arteries diminished in size.

CHAPTER XII.

THE DATA OBTAINED BY OBSERVATION—(*Continued*).

The *muscles*—idiopathic muscular atrophy—pseudohypertrophy—Thomsen's disease—paramyoclonus multiplex. Myositis—myalgia—muscular rheumatism.

THE MUSCLES.

The Nutrition. The nutrition of the *muscles* is observed by the hand of the examiner while the muscles are made to relax and contract alternately. We compare corresponding muscles of the two sides. Measurement of the limbs at corresponding situations makes the observation more accurate. The muscles may *atrophy* or *hypertrophy*. Either condition may be local, unilateral, bilateral, or general.

Myoidema is a local contraction of the muscle which occurs upon striking it with a pleximeter or the finger, as in percussion. It is more particularly seen in thin subjects, usually tuberculous, and elicited by tapping the pectoral muscles. The fasciculi raise in little humps, which persist for a short time and gradually subside. At one time they were thought to be diagnostic of tuberculosis. They are of no special significance.

Atrophy.

There are several varieties of atrophy: 1. The atrophy of disuse. 2. Myopathic atrophy. 3. Myelopathic atrophy, or the atrophy of degeneration. It follows lesions of the motor path, of the cortex, medulla, or spinal cord; and neuritis. (See Nervous Diseases.)

The Atrophy of Disuse. It is also known as the atrophy of inactivity. The muscles are slightly lessened in volume. The atrophy takes place very slowly; it supervenes in cases of paralysis and in the joint-diseases which cause immobility. It occurs also in joint-disease from reflex influences. The electrical reactions of the muscles are qualitative and unchanged. By this reaction atrophy from disuse and atrophy from disease of the muscles can be distinguished from myelopathic atrophy, due to disease of the nerves (neuritis), or to degeneration of motor nerves and ganglia.

Myopathic Atrophy. *Muscular Dystrophy.* In this form of atrophy the muscle is diseased. It diminishes in volume and finally becomes completely shrunken. Complete paralysis rarely ensues, but the reaction of degeneration cannot be determined.

Idiopathic Muscular Atrophy. *Dystrophia muscularis progressiva* (Erb). In this affection muscular wasting takes place with or without initial hypertrophy. Three forms are seen:

1. Atrophy with Pseudohypertrophy. It usually begins in childhood, and is often of congenital origin, being transmitted through

the mother. It is first noticed just as the child is learning to walk. The extensors of the leg, the glutei, the lumbar muscles, the deltoids, and the triceps and infraspinati muscles are involved, but the first change takes place in the muscles of the calves. The muscles of the face, neck, and forearm are not usually affected in this form of the disease; the muscles of the hand are not involved. While hypertrophy progresses in certain muscles others waste. The calves may hypertrophy, for instance, while the extensors of the leg waste away and become weak. Attitude and gait are characteristic. (See page 73.) The patient stands erect, with the legs apart, the shoulders thrown back, the spine curved, and the abdomen prominent. The waddling gait is characteristic, and the method of getting up from the floor is pathognomonic. The course of the disease is slow, wasting follows the hypertrophy, but the weakness is greatest in the muscles first atrophied. Contractures and distortions of the spine and of the bones of the leg take place.

2. Primary Atrophy. This is likewise congenital or manifests itself in early life. It is divided into different types, according to the groups of muscles that are affected. The same process occurs as in the former, except that pseudohypertrophy is not primary. There may be several forms in different members of the same family. Of these we have *the juvenile form of Erb.* The upper arm and shoulder and the thigh muscles are first involved. Later the muscles of the gluteal region and calf may become enlarged and hard. The back muscles are gradually affected, inducing the attitude previously mentioned. The reaction of degeneration is not present. There is also an *infantile type*, first described by Duchenne, or the *fascio-scapulo-humeral type.* Erb's form begins about puberty. The other forms usually begin in childhood, but may be delayed. The face is involved; it is expressionless, and in laughing the muscles move slowly; the child cannot whistle, as the lips are thick and everted. The eyes remain partly open. The muscles of the group waste; later the thighs become involved. Erb has given a useful test to determine the strength of the shoulder and girdle muscles. When the child is lifted by the armpits, if the scapulohumeral groups are weak, the shoulders are forced up to the child's ears without resistance.

3. Peroneal Atrophy. A *peroneal type* of muscular atrophy has been described by Charcot. The extensors of the great toe and afterward the common extensors and peronei muscles are affected; club-foot results. The muscles of the thigh may become involved later. When the disease occurs in childhood it gradually spreads to the upper extremities and affects the muscles of the hand, differing in this respect from other forms of muscular atrophy. The thenar, hypothenar, and interossei muscles are symmetrically involved, producing the claw-hand. Unlike the other forms of atrophy embraced under this heading, the peroneal type is attended by disturbances of sensation, and by pain, fibrillary contractions, and vasomotor changes. The reactions of degeneration may be present. It is thought by competent observers to be simply a form of neuritis; and it is also called *progressive neural muscular atrophy.*

Diagnostic Features of Myopathic Atrophies. The disease is characterized by gradual progression of the wasting and weakness in

various groups of muscles not specially related. We never see wasting of the intrinsic muscles of the hands, as in the spinal forms of muscular atrophy, or of the tongue, pharynx, larynx, and eye. Electrical irritability is lessened and reaction of degeneration is not present. Fibrillary twitching is not seen. Sensation is not affected. The reflexes are diminished and later may be lost. The sphincters are not involved; deformities about the joints or in the spinal column may occur.

The *diagnosis* of idiopathic muscular atrophy is not difficult if the above-mentioned facts are borne in mind. The fact that it occurs in family groups is an important point in the diagnosis. In *cerebral atrophy* there is primary loss of power. In *chronic anterior poliomyelitis* (*spinal atrophy*) wasting begins in the muscles of the hands; in both the simple and spastic form there are reactions of degeneration, fibrillary twitching, and increase in the reflexes, and, in the latter, spastic contraction of the legs. The myopathies occur early in life, and are hereditary.

In *neuritis* the paralysis is proportionately greater than the atrophy. Sensory symptoms are often present. The cause is distinct. There is no family history.

General Atrophy. In cachexias the muscles as well as the tissues undergo atrophy. Even in nervous disease the atrophy of the muscles markedly increases when general wasting takes place.

RAYMOND'S TABLE OF ATROPHIES.

- Circumscribed atrophies
  - Atrophy from compression.
  - Atrophy in inflammatory conditions (pleurisy, joint-disease, etc.).
  - Atrophy from injury or inflammation of individual nerves.
- Progressive atrophies
  - Progressive spinal muscular atrophy; type Aran-Duchenne.
  - Progressive myopathic atrophy
    - Pseudohypertrophic muscular paralysis.
    - Type Leyden-Möbius.
    - Type Zimmerlin.
    - Type Erb.
    - Type Landouzy-Déjérine.
    - Type Charcot-Marie.
- Diffuse atrophies
  - Anterior poliomyelitis
    - Infantile form.
    - Acute of adults: spinal paralysis, with rapid course and curable (Landouzy-Déjérine); subacute and chronic form; chronic mixed form (Erb); diffuse subacute, general spinal paralysis (Duchenne).
  - Syringomyelia.
- Facial hemiatrophy
  - Multiple neuritis (amyotrophic form)
    - Lead paralysis.
    - Leprous neuritis.
    - Alcoholic neuritis.
- Muscular atrophies of cerebral origin
  - With secondary degeneration involving the anterior cornua.
  - Without secondary degeneration involving the anterior cornua.
- Muscular atrophy in hysteria
- Muscular atrophy from systemic disease of the cord
  - Amyotrophic sclerosis.
  - Glosso-labio-laryngeal paralysis.
- Atrophy complicating other disease of the cord
  - Atrophy in myelitis.
  - Atrophy in compression of the cord.
  - Atrophy in multiple sclerosis.
  - Atrophy in tabes dorsalis.

Hypertrophy.

Hypertrophy of individual muscles occurs from overuse, as when an extremity or a portion of the trunk is used in excess. General hypertrophy of muscles occurs in Thomsen's disease. True hypertrophy is recognized by increased volume, great hardness, and increased vigor of the muscle.

Pseudo-hypertrophy (see under Muscular Atrophy) is associated with increased volume of muscle but diminished power.

Thomsen's Disease (*Myotonia congenita*). This is an hereditary disease and may occur in several generations of a family. Tonic cramps take place in the muscles when voluntary movements are attempted. The disease begins in childhood, rarely after puberty. The muscles become rigid and fixed when put in action. The lack of voluntary control of the muscles is shown by the slow contraction and relaxation when voluntary efforts are made. The rigidity may wear off and the limb can then be used. It is particularly noticeable when walking is attempted. As the leg is advanced slowly it may remain stiff for a second or two, but after it becomes limber the patient can walk for hours. If he stops walking the same difficulty is experienced when he starts again. Both arms and legs are affected. Patients are usually well nourished, however. There are no atrophies. The muscles are irritable, so that mechanical stimulus or pressure causes tonic contraction. Movement and cold aggravate it. Sensation and the reflexes are not affected, and there is no evidence of disease of the cerebro-spinal system, save the occurrence of hypochondriasis in some cases. The myotonic reaction described by Erb is induced. (See electrical diagnosis—Diseases of the Nerves.)

Paramyoclonus Multiplex. In this affection there is clonic contraction of the muscles. It is usually confined to the extremities and occurs in paroxysms. It may have been caused by sudden twitching or violent motion. The clonic spasms at first do not interfere with the patient's occupation, but gradually they increase. Both legs are affected, and the number of contractions varies from 50 to 150 a minute. The contractions may be rhythmical. In severe cases the muscles of the back and abdomen contract violently. Tremor of the muscles may be present in the intervals. (For *paralysis, spasm, tremor, contraction,* etc., see Nervous System.)

Myositis. *Inflammation of the muscles.* (See also Trichinosis.) In inflammation of the muscles there is pain, swelling, and loss of power. In universal myositis the inflammation begins in the lower extremities and gradually involves other muscles of the body. They are swollen, hard, and painful on pressure. Atrophy supervenes in groups of muscles. The muscles may become more or less rigid. Local œdema of the skin over the muscles occurs. The progress is gradual, and death ensues when the respiratory muscles are involved.

The three cardinal symptoms that attend the disease as described by

Loenfeld are: (1) Swelling of the extremities due to subcutaneous œdema and swelling of the muscle, causing functional disturbance; (2) extension to the muscles of respiration and deglutition; (3) a more or less extensive eruption. The latter is erythematous, its distribution is usually general but irregular, and may be followed by pigmentation. The disease must not be confounded with trichinosis. In the latter examination of a small portion of muscle reveals the trichinæ.

Progressive ossification of the muscles is rare. The muscle-tissues undergo gradual ossification, either in localized spots or in wide-spread areas. Inflammation of the muscle precedes the ossification. As the inflammatory swelling subsides the muscles become hard and are gradually converted into bony tissue. The disease lasts many years.

Myalgia is an inflammation of the muscles produced by cold or trauma. There is pain on movement and spontaneous pain in the muscle; it is tender on pressure. It may be the seat of spasm.

Muscular Rheumatism. In this variety of rheumatism there is pain in the affected muscles, which often comes on suddenly in the night, or is first noticed when the patient attempts to rise in the morning. The pain when the patient is at rest may be inconsiderable, rarely amounting to more than a dull, aching, sore feeling; on attempting to move, to bend, or twist, or straighten himself, however, the patient catches himself suddenly on account of the agonizing, tearing, or burning pain. When the muscles are relaxed the patient is fairly comfortable. Sudden movement is the most painful. The affected muscles are tender to the touch and to sharp blows. Muscular rheumatism may be acute or chronic. In the latter the symptoms are very much like those of chronic articular rheumatism, except that the muscles and not the joints are affected. There is the same proneness to recur in unfavorable weather and in cold, damp seasons.

The disease receives different names according to the muscle affected. The most common subvarieties are: *lumbago*, in which the muscles of the small of the back are affected; *pleurodynia*, in which the intercostal muscles suffer; and *torticollis*, in which the sternomastoid and trapezius are painfully contracted.

In *lumbago* the patient holds himself rigid and is unwilling to rotate the trunk upon the vertebræ. Often the most comfortable position is that in which he sits and bends slightly forward over another chair. Motion is painful but pressure is not. Fever is absent. There is a history of repeated attacks, or of exposure, such as lying upon damp ground. Lumbago needs to be distinguished from disease of the spinal membranes, from disease of the vertebræ, aneurism, abdominal abscess, and diseases of the uterus and ovaries. The diagnosis of rheumatism is arrived at by exclusion.

In *pleurodynia* there is usually tenderness upon pressure as well as upon motion and deep inspiration. The pain is of the same sore, burning character, aggravated by coughing and sneezing. The patient breathes as little as possible, and often bends over toward the affected side to lessen the motion. Pleurodynia is distinguished from pleurisy

by the absence of fever, cough, and, above all, of friction-sounds. In intercostal neuralgia there are painful points upon pressure, whereas in pleurodynia firm pressure is grateful, though tapping is painful.

In *torticollis* the head is drawn to one side and fixed in that position. The sternomastoid especially is rigid and tender on pinching. In spinal affections the head is retracted, and there are antecedent symptoms, as headache and darting pains with fever.

Fibrous Tissues. Intimately associated with rheumatic affections of the muscles is that of the fibrous tissues or fascia. Pain, fixation, and tenderness are noted, and if with them other rheumatic manifestations are found the diagnosis is established ; especially is the above true of trauma.

CHAPTER XIII.

THE DATA OBTAINED BY OBSERVATION—(*Continued*).

The *bones*—general examination. *Enlargement—acromegaly—osteitis deformans—pulmonary osteo-arthropathy—Diminution—rhachitis osteomalacia.* Local examination—position and shape—nodes—inflammation—osteomyelitis.

THE BONES AND JOINTS.

Method of Examination. When the bones and joints, especially the spinal column, are to be examined, the patient should be stripped, and after the movements and position in the upright or semi-upright position have been noted, he should be made to lie down on a hard, smooth surface, and the trunk and joints examined in that position. Anterior, posterior, and lateral movements of the spinal column must be made to determine its flexibility. In this manner deformities, changes in the length of the bones, and abnormal posture can be carefully observed. In addition we must note muscular wasting, the presence of local tenderness and swelling, changes in the movements of the joints, and loss of other functional activity causing lameness or joint-disability.

To distinguish joint lesions from abnormal flexions or extensions, the result of spasm of muscles, *anæsthesia* must be employed.

The Bones.

The bones are fixed landmarks by which the location of organs is determined. The student should familiarize himself with the shape of the bones and the location of normal tuberosities.

The bones may be the seat of nutritive changes which involve the skeleton in whole or in part, causing enlargement or diminution of the osseous system, and hence of the body. Local changes are traumatic (periostitis) or infectious, giving rise to nodes or to swellings.

General Examination. ENLARGEMENTS. Nutritive changes giving rise to enlargement of the bones occur in acromegalia, osteitis deformans, and pulmonary osteo-arthropathy.

Acromegalia.

Marie first described *acromegaly*, a skeletal change, characterized by hypertrophy of the bones of the hands, feet, and face. The fibro-cartilages of the ear and larynx are also enlarged. The enlargement of the inferior maxillary and frontal bones causes the face to assume a peculiar, elongated, elliptical outline. The nasal bones are enlarged,

and the nose thickened; the temporal fossæ are deepened, on account of enlargement of the malar bones. The forehead retreats because of the enlargement of the frontal sinuses and projection of the superciliary ridges; the chin is prominent and the lower teeth project beyond the plane of the upper; the lips and eyelids may be thickened; the tongue is enlarged and thickened. The hair is coarse and dry; the face dry and pigmented.

The hands are peculiar; they are much broader, the fingers are sausage-shaped, and the hand spade-like in shape; the nails are flat, striated, and too small. There is usually spinal curvature; the abdomen is prominent, and, as before intimated, the height is increased. The muscles become weak and may atrophy; the skin is often pigmented; varicose veins have been observed, and the patient complains of hemorrhoids. The thyroid gland may be atrophied or hypertrophied. It may be well to state, in passing, that with these appearances nervous phenomena are observed and disorder of special senses complained of. Hemianopsia, limitation of the visual field, and blindness or deafness arise.

FIG. 23.

Case of acromegaly. (OSBORNE.)

Osteitis Deformans.

Another remarkable change is seen in the skeleton, and has been described by Sir James Paget; in this there is marked change in the contour of the patient and a peculiarity in the mode of locomotion. It is known as *osteitis deformans*. The head is advanced and lowered, so that the neck is very short, and the chin, when the head is at ease, is more than an inch below the top of the sternum. The chest becomes contracted, narrow, flattened laterally, deep from before backward, and the movements of the ribs and spine are lessened; the arms appear unnaturally long; the shafts of each tibia and femur are bent so that the patient becomes bow-legged. There is some stiffness, but no loss of power and not a great deal of pain. The skull is increased considerably in thickness.

These changes in the bones cause a dwarfed appearance of the trunk in comparison with the legs and arms, and the posterior lateral curvature necessitates a characteristic attitude. The skeletal changes are noted particularly in the long bones. As a result of the enlargement of the cranial bones, the face presents a triangular outline, with the base

above and the apex below (see Fig. 24, outline 3), thus differing in appearance from the outline in acromegaly (Fig. 24, outline 2).

FIG. 24.

1
Outline of face in myxœdema.

2
Outline in acromegaly.

3
Outline in osteitis deformans.

Pulmonary Osteo-arthropathy.

Marie distinguishes acromegaly from another skeletal change in which there is hypertrophy of the bones of the extremities, including enlargement of the shafts. In this form of arthropathy the bones of the head and face are not affected. The hands and feet are enlarged, and the patellæ and other bones of the knee-joints increased in size.

FIG. 25.

Pulmonary osteo-arthropathy. Female, aged eleven. Tuberculous vertebral caries and pulmonary tuberculosis. Enlarged clubbed fingers and thickened ulna and radius. Private patient, 1885.

Curvature of the spine is present. The appearance of the fingers is different from that seen in acromegalia. The ends are enlarged and bulbous, and the nails are too large and are curved in a transverse and

longitudinal direction, like the clubbed fingers of phthisis, although the chief enlargement of the fingers is not terminal, and there is no cyanosis, as in phthisical clubbing. The change seemed to be associated with pulmonary affections, and Marie called it *ostéo-arthropathie pneumonique*.

Diminution. Small development of the bones is seen in idiots and cretins; later in life diminution in size may occur from rhachitis and osteomalacia.

Rhachitis.

In this affection the size of the body is lessened. For its recognition it is important to know how rapidly the osseous deposits in childhood have formed. The fontanelles and the epiphyses must be examined. If the fontanelles are open beyond their period of closure in health, or if the epiphyses are enlarged and lack firmness, the condition points either to simple malnutrition or to rhachitis.

In rhachitis late development of the *teeth* is observed. If the ribs are examined, nodules will be detected at the junction of the bone with

FIG. 26.

Rhachitis; attitude in sitting; one hand raised to exhibit swelling at the wrist. (WILLIAMS.)

FIG. 27.

Rhachitis in moderate degree in a boy aged fifteen months; showing backward excurvation of the spine. (WILLIAMS.)

the cartilage. These may be seen, as well as felt, if the child is thin. They form the so-called *rhachitic rosary*. The thorax also is changed in shape. At the junction of the cartilages and ribs a depression takes place which is continuous with a groove which passes out from the ensiform cartilage toward the axilla. This transverse curve is known as Harrison's groove. It may deepen with inspiration. The sternum projects, forming the so-called "pigeon-breast." (See Thorax.) Such

deformity must not be confounded with a similar one seen in adenoid disease. Changes at the lower end of the radius and ulna, and sometimes at the end of the humerus, are noticed. The parts are enlarged at the junction of the shaft and epiphyses. There may be thickening of the clavicles at the sternal ends. In the legs the lower end of the tibia becomes enlarged, and at times the upper end, or even the shaft, becomes thickened. The child becomes bow-legged, or the tibiæ and femora may arch forward. Knock-knee sometimes occurs. The bones of the vertebral column and of the pelvis are also affected. The spine is usually curved posteriorly, but the lateral curvature may also be produced with it. The contraction of the pelvis is such as to narrow its outlet—a matter of much importance for the future of female children.

The head of the child with rickets is quite characteristic. It has been mentioned that the fontanelles remain open for a long time, and areas of ossification are imperfect, so that the bone yields to the pressure of the finger. This occurs particularly at the side, and the term *craniotabes* is applied to it. The large head is square in shape, not globular, when seen from above downward. It gives the face a peculiar appearance. It is proportionately very small, especially in the lower two-thirds, while the forehead is broad and square.

Rhachitis is usually developed in childhood, and is most common in children with bad hygienic surroundings, who have lived upon a starchy diet and have taken cow's milk for too long a period of time. A child that has been nursed during the mother's pregnancy is liable to have the disease.

In addition to changes in the bones a child presents other evidences of defective nutrition. There is marked pallor ; the muscles are flabby ; the child is feeble ; and the weakness of the muscles results in an inaction which resembles paralysis.

The disease usually progresses slowly, and is eminently chronic. A form is seen, however, in which the progress of the symptoms is more acute. With some gastro-intestinal disturbances there are mild fever, considerable weakness, and great restlessness. Sleep is disturbed, and pain is complained of if the child is of an age to make such complaint. *Soreness* of the body is observed on handling the child ; and of its own accord, on account of the pain and soreness, it avoids all customary movements. The child lies on its back and shrinks from any attempts to disturb it. The pain is not only caused by handling of the muscles, but the bones also are sore and tender. Sometimes the most marked manifestations of the more acute forms are the gastro-intestinal symptoms. It may often happen that vomiting and diarrhœa have as an underlying basis this rhachitic condition.

With the above symptoms, and also in chronic cases, *perspirations* about the head are common. There is usually more heat of the head than is natural, hence in sleep the child rolls the head. This rolling causes the hair on the back of the head to be worn off. This sign is most characteristic of rhachitis when observed along with changes in the skeleton.

In the acute and chronic forms *enlargement* of the *liver* and *spleen* is

observed. The enlargement is not only actual, but also a false enlargement may be seen from distortion of the organs, on account of changes in the vertebræ and ribs. The abdomen is prominent, usually on account of flatulency, although the enlarged organs contribute to the swelling.

Nervous phenomena are common in the course of rhachitis. *Tetany*, limited to the upper extremities, and *laryngismus stridulus* are the most frequent. Either of these complications may occur before the disease is otherwise suspected.

Diagnosis. The possible presence of rhachitis must not be overlooked in cases of chronic vomiting in childhood. The acute form of the disease must not be confounded with scurvy, as often happens in the case of children. It must not be forgotten that scurvy may set in in the course of rhachitis. In scurvy the pain, tenderness, and weakness are limited to the lower extremities. The immobility of the extremities may go on to pseudoparalysis. The tenderness, however, is great; œdema is more pronounced, and local areas of periostitis are more common. In scurvy the gums are swollen and may be spongy, or may be the seat of ecchymoses. The most decisive diagnostic criterion is the therapeutic test, scurvy rapidly yielding to a proper regimen.

Osteomalacia.

Among the general affections of the skeleton which may cause lessened size, *osteomalacia* must not be forgotten. As the lime salts are dissolved the bones become preternaturally soft, break on the slightest provocation, or bend in various directions, depending upon the external pressure and the direction of the muscular force. The ribs are drawn in by inspiratory force until the cavity of the thorax is lessened to a degree incompatible with life. The pelvis is deformed so that labor is impossible. (It occurs frequently in pregnancy.) All sorts of fixed contortions are assumed. If the patient is able to be up the body shortens, the back becomes rounded, the neck flexed, so that the chin is brought close to the sternum. On palpation the bones can be indented with the finger, and crepitate like egg-shells.

Osteomalacia is easily distinguished from *carcinoma* or *sarcoma* of the bones. In the latter spontaneous fracture occurs in various parts of the skeleton, but is generally preceded by pain and swelling at the seat of fracture. Then, in sarcoma, subcutaneous hemorrhages are present. When a single joint is affected in osteosarcoma the same egg-shell crackling is observed.

Local Examination. The Position and Shape of Bones. The peculiar position (falling downward) of the *scapula* in paralysis of the serratus magnus is diagnostic of that affection, and indicates disease of the posterior thoracic nerve. In examination of the *clavicles* fractures must not be mistaken for disease of the bones, such as rickets. The examination of the *spinal column* is of the greatest importance. (See Spinal Joints.) A study of the diseases of the spinal column due to caries from tuberculosis is not within the province of this work; no physical examination, however, is complete without an investigation

of the movability of the spine and the presence or absence of curvature. I refer to the curvature due to weakness of groups of spinal muscles. Functional disorders of the gastro-intestinal tract and of the uterus are undoubtedly intensified by the presence of curvature, which leads to deformity of the body, and hence to the assuming of abnormal positions when sitting or walking. The recognition of lateral or anterior curvature leads to the adoption of lines of treatment which otherwise would not be followed, but without which weak muscles, improper aëration of the blood, and sluggish circulation would persist. Pain in the distribution of nerves, or at their termination, is often due to spinal caries pressing on them as they pass through the foramina. The most noticeable is the pain about the umbilicus in children due to Pott's disease.

The bones and cartilages connected with the thorax will be considered under Diseases of the Lungs.

Inflammation. The discovery of a slight change may lead to the recognition of a grave general process. Simple local inflammation or *periostitis* may be due to syphilis, and is recognized by local pain, swelling, and slight œdema. It may be diffuse. It is seen most frequently on the tibia, sternum, and clavicle. It not infrequently follows typhoid fever.

Nodules or nodes are usually due to syphilis. They form on various portions of the skeleton, but are most frequently seen on the skull, especially on the forehead; they are also found on the shafts of the long bones, preferably the tibia, ulna, and clavicles. They are usually multiple or bilateral. They are painful and tender on pressure, and may be the seat of heat and redness. They are not so hard and dense as *exostoses*. The latter are situated on the outer aspects of the bone and in relation with the strongest tendons or muscles.

As an illustration of the importance of recognizing nodes the writer recalls a case of persistent headache, the true nature of which was only ascertained by finding a small node on the skull. The headache had been of long (five years) duration, and treatment for it had been sought in many countries.

Tenderness of the *sternum* upon pressure is often of diagnostic significance and is usually indicative of syphilis. The pain and tenderness just noted, however, must not be confounded with local tenderness due to necrosis, which often arises in convalescence from fevers, notably those of an infectious nature.

Osteomyelitis. The occurrence of high fever, with or without chills, but usually with pyæmic symptoms, without recognized cause, should lead to an examination of the bones. A spot of tenderness followed by local redness and swelling—on the tibia, for instance—would indicate the seat of suppuration in *osteomyelitis*.

The Joints.

The Data Obtained by Inquiry. Careful observation of the bones enables us largely to discern the nature of the diseased process, as has just been indicated. It is true *osteomyelitis* is less likely of recognition

than any other process, but when the patient has been exposed to an infection, and fever is present, this condition must always be sought for in the absence of any other infectious area.

Such is not true, however, of joint-disease. By observation we determine the joint affected and in part the nature of the morbid process. Other data are needed. Hence we collect the usual data obtained by *inquiry*. The *social history* is not productive of valuable data. Acute rheumatism is more common in early life, rheumatoid arthritis in the middle periods, and chronic rheumatism in late life. Females are more commonly attacked than males in rheumatoid arthritis, and this affection is more common in the poorer classes. Males and the well-to-do are the victims of gout.

In the *family history* one learns of the transmission of gout from generation to generation and of the occurrence of rheumatism or of its various allied processes in members of the same or previous generations. *Previous diseases* elicited are those of an infectious nature or an intoxication, as of lead. Such diseases must be sought for if the true nature of an arthritis is to be discovered. The history of the *present disease* is often that of recent infection or intoxication.

The *subjective symptoms* of joint-affections are worthy of note. *Pain* is the most prominent. This may be spontaneous, or may arise upon pressure, or follow attempts at movement. Spontaneous pain with tenderness is more pronounced in rheumatic and gouty inflammations of the joints. The pain is usually worse at night. This is particularly the case in tuberculous joints, and is due to removal of the apprehensive spasm of the muscles whereby the joints had been protected.

Pain in the joints must not be confounded with that of local or multiple neuritis. I have seen the pains of neuritis attributed to rheumatism of the phalanges, tarsus, and ankle until paralysis of the extensors took place. I have seen the pain of neuritis of the circumflex mistaken for shoulder-joint disease. Multiple neuritis is attended by pains that may be located in the joints by the patient; but neither in local nor in general neuritis are the joints ever swollen, tender, or painful on passive movement.

Inspection. The *size*, *shape*, and *color*, the degree of *movability* and the *position* of the joints are observed.

The Size and Shape. The joints may be *enlarged*. The enlargement may be due to infiltration of the tissues about the joints, to effusion within the joints, serous or purulent, or to inflammation of the ends of the bones.

1. When the enlargement is due to infiltration about the joint the tissues are previously thickened, as shown by palpation, and the outline of the joint is changed. The normal contour is lost entirely, and, instead, there is a globular swelling beginning above and extending below the joint. 2. When the enlargement is due to effusion it may be detected by palpation, as this secures fluctuation. This is particularly so in the large joints. If the joint involved is the knee the patella will float. The effusion changes the normal contour, but, in the earlier stages, may cause local swellings where the synovial sacs are near the surface; hence, at the articulation of the tibia and

fibula with the tarsus, on the inner and outer side, a boggy swelling is observed. At the knee the swelling is on each side above and below the patella. When the effusion is great the joint becomes immobile, and may be flexed from distention of the sac. 3. When enlargement of the joints is due to hypertrophy of the bones the latter are thickened and very hard. There may or may not be, and usually is not, fixation, and movement is but moderately interfered with.

Changes in the *outline* of the joint are also seen in rheumatoid arthritis. The loss of the cartilaginous substance of the joint, with the secondary osteophytic changes, causes deformity, so that in the case of the small joints of the finger subluxation is seen ; similar subluxations are seen in larger joints. The ends of the phalangeal bones are thickened.

THE COLOR Change in the *color* is usually noticed in inflammations. The surface is either bright red or dusky.

THE POSITION. The *position* assumed is of diagnostic importance. Flexion of the limb of the affected joint occurs in over-distention. It must be remembered that the hip-joint is flexed in appendicitis and in psoas abscess or other affections in proximity to the psoas muscles. In rheumatoid arthritis there is subluxation. *Immobility* is observed. (See Palpation.)

Palpation. By palpation we determine the degree of *movability* of the joints, the presence of *fluctuation* and of *crepitation.*

1. The *movability* of the joint is learned. Movement is inhibited in inflammation on account of the pain. A reflex muscular spasm takes place if osteitis and cartilage-destruction are present. The spasm prevents movement. In effusion there is less movability or even none at all. In rheumatoid arthritis movement is prevented by the osteophytic growths which surround the joint.

2. *Fluctuation* is revealed by palpation, pointing to liquid effusion within the joint. Œdema of the surrounding tissues occurs in purulent effusions.

3. A *crepitus* or grating sensation is observed in rheumatoid arthritis and other destructive diseases.

The Morbid Process. The processes which give rise to change in the joints are inflammatory and degenerative, and, curiously, neurotic or neuropathic. When a single joint is the seat of disease the process may be local, as in traumatic *synovitis.* But tuberculosis and other infections, gout and rheumatism or rheumatoid arthritis, may be localized to one joint—the latter rarely, however. Multiple joint-disease, *polyarticular,* is infectious or systemic (intoxication) usually.

Much information, therefore, is learned by noting if the process is limited to one joint, *monarticular ;* or to many joints, *polyarticular ;* if to large joints or to small joints ; if it is fixed, as in synovitis, or fugitive, as in rheumatic fever. Monarticular inflammation of small joints points to gout ; of large joints, to gonorrhœal rheumatism or pyæmia. Polyarticular inflammation of small joints, to rheumatoid arthritis ; of large joints, to rheumatism. Lesions may be unilateral or bilateral, symmetrical or asymmetrical. Bilateral joint lesions are characteristic of rheumatoid arthritis. Asymmetrical and fugacious lesions are seen in rheumatic fever.

It must always be remembered that joint-lesions or processes may be expressions of *general infections*, as septicæmia, influenza, cerebro-spinal meningitis, scarlet fever, and dysentery; or *blood diseases*, like purpura or hæmophilia or scurvy; or of *nervous diseases*, like tabes dorsalis.

We have to consider synovitis or arthritis single and multiple, traumatic, toxic, or infectious, of which gonorrhœal and tuberculous infections are the most common monarticular causes. We will then consider rheumatism and gout, rheumatoid arthritis, and follow with the neuropathic joints.

Synovitis. The inflammation is recognized by pain, heat, redness, and swelling. Effusion is present, and its physical signs are readily elicited. It is both periarticular and intra-articular. It may be due to traumatism, but we are chiefly concerned with inflammations due to internal morbid processes. When single joints are affected the most common causes are tuberculosis, pyæmia, and gonorrhœal infection. A mild degree of inflammation may be limited to one joint in subacute rheumatism. When many joints are affected the cause is an infectious one, as rheumatism, septicæmia, pyæmia, epidemic cerebro-spinal meningitis, scarlet fever, and dysentery, rarely gonorrhœa.

The Tuberculous Joint. In *tuberculosis* the joint is swollen and the neighboring tissue œdematous. Effusion may be detected. There is fever. The hip, the knee, the elbow, the wrist, and the ankle are most frequently affected. Cheesy material may be withdrawn by tapping. Destruction ultimately takes place, with subluxations and subsequent fixation of the joint. With fever, wasting, and local signs of tuberculosis in other portions of the body the true nature of the affection is indicated. The tuberculous process may be limited to the affected joint, extend to the tendinous sheaths, or secondary tuberculosis of internal organs may supervene.

The Joint of Gonorrhœal Rheumatism. The knee-joint is usually affected. Signs of acute or subacute inflammation are present, with œdema and effusion. The patient is a male in whom an acute or chronic urethral discharge is found. The pain is worse at night. The process is of long duration. Metastasis does not take place. Destruction rarely occurs, but anchylosis may. General pyæmic symptoms may ensue, and gonorrhœal endocarditis supervene. The micro-organisms (gonococci) can be found in the blood and in the pus of the affected joint. There is entire absence of heart-symptoms from simple endocarditis. The general and local signs of rheumatism or of a rheumatic diathesis, and changes in the urine, skin eruptions, cardiac lesions, etc., are wanting. In certain cases many joints are affected, but the temperature is not so high or the sweats so profuse as in acute rheumatism. Tendo-synovitis is not infrequent.

Rheumatic Fever.

An acute, general, febrile, non-contagious disease, characterized by specific inflammation of the *joints* and their contiguous structures, hence called *acute articular rheumatism*. It is further characterized by a tendency of the inflammation to involve the larger joints successively, to

skip from one joint to another, and to be associated with endocarditis or pericarditis.

The predisposing causes of rheumatic fever are *heredity*, which is operative in 25 or 30 per cent. of the cases; *age*—81 per cent. of first attacks occur between the eleventh and thirtieth years (Pye-Smith); *sex*—in childhood girls are more frequently affected than boys, but after that period sex appears to have no influence. Polyarticular inflammations, sometimes rheumatic in nature, are met with during convalescence from scarlatina and dysentery. They also occur in association with the puerperal state and gonorrhœa, in which they are probably pyæmic. The nature of the polyarthritis which occurs in connection with dengue and hæmophilia is obscure.

Symptoms. The *onset* of the disease is not characterized by constant symptoms. Sometimes the fever and joint-inflammations are preceded a day or two by debility, wandering pains in the joints or muscles, and loss of appetite. In other cases there is a chill or repeated attacks of chilliness, followed in a day or two by fever and inflammation of the joints. In rare cases the onset may be followed not by inflammation of the joints but by inflammation of the serous membranes, particularly those of the heart and its sac.

The *temperature* may rise a day or two before there are any joint-symptoms, or fever and arthritis may begin almost simultaneously. The temperature rises rapidly to 102°, 103°, or 104° F., and one or more of the larger joints, generally the knee and ankle, become painful, tender, swollen, and hot.

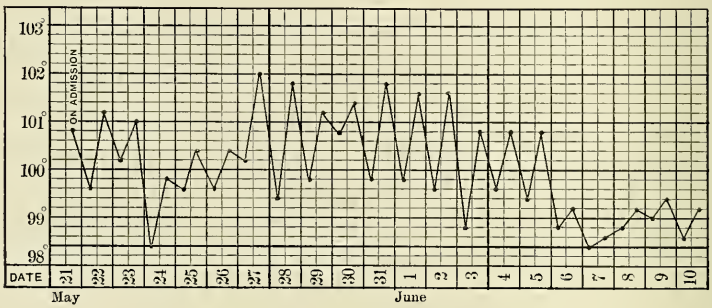
The Joint. There may be great pain on motion before there is evident swelling or much local tenderness. The pain varies from mere discomfort to the most excruciating suffering. It is always aggravated by motion or pressure, and is at times so exquisite that the slightest touch, the weight of the bedclothing, or the jar of the bed from a heavy step in the room makes the patient cry out. It may extend beyond the joint to neighboring tendons and nerves. The swelling likewise varies greatly; sometimes there is only slight puffiness with increased distinctness of the cutaneous veins, increased heat in the part, but no general redness; in other cases there is considerable swelling about the joint, so that the bony prominences are obliterated, the surface being tense, red, and very hot to the touch. There is often effusion into the joint. Swelling is most marked in the wrist and ankle, and less so in the shoulders, hips, elbows, and knees.

Multiplicity of Joints Affected. A characteristic peculiarity of rheumatism is its tendency to involve one joint after another. One or several joints may be affected at first; it is very common for the right ankle to be affected, and then in a short time the opposite ankle, followed by the left knee and right knee, and so on with the other joints. The inflammation usually lasts in each joint from two to four days. The process may subside in one articulation and begin in another with startling rapidity. At one visit of the physician the patient's right ankle may be swollen, hot, and unbearably painful, and on the next day the right ankle may be quite well again and the patient be found suffering acute pain in the right knee or left ankle.

The *pulse* in the early stages of rheumatism is moderately accelerated (99 to 110); it is regular, of good volume, often bounding, and sometimes hard. The *urine* is scanty, high-colored, abnormally acid, and deposits on cooling a copious precipitate of urates, resembling red sand in appearance. The *skin* does not feel so hot as one would expect from the temperature. It is continuously covered with a copious, acid, and somewhat pungent perspiration. *Nervous symptoms* are not marked. There may, however, be slight nocturnal delirium. Sleeplessness from pain is very common.

The *temperature* in rheumatic fever is not usually very high; it is much oftener under than over 103°. In rare cases, however, especially when the fever is complicated with pericarditis, pneumonia, or

FIG. 28.



Rheumatic fever. Admitted fourth day of disease.

some disturbance of the heat-regulating apparatus, the temperature may attain the extraordinary range of 106°–112° F. Such high temperatures may occur suddenly or gradually, and are sometimes attended with marked brain-symptoms (so-called cerebral rheumatism).

Endocarditis and *pericarditis* may occur at any period of rheumatic fever; they may even precede any joint-inflammations. They are most common, however, in the first two weeks of the disease. The younger the patient and the more severe the attack the greater the liability to heart-complications. They occur in about one-fourth of all cases. Endocarditis is most common; often it is the only lesion, but sometimes it is associated with pericarditis and more rarely with myocarditis. These complications usually give rise to no symptoms at first. Hence the heart should be examined daily. A sense of constriction in the præcordia or pit of the stomach, an anxious expression of the face, with pallor, a change in the frequency, but especially in the rhythm of the pulse, and the occurrence of cough or dyspnœa, should attract attention to the heart. The physical signs of the respective lesions have been described fully under Diseases of the Heart.

The setting in of convalescence from rheumatic fever is marked by cleaning of the tongue, which also becomes less red, and increase in the secretion of urine, which remains of high specific gravity. The fever subsides gradually, the joints cease to be red, swollen, and tender,

the acid sweats lessen, and the appetite improves. In proportion to the duration of the case and its severity the patient is left with debility and marked anæmia, both red cells and hæmoglobin being diminished. In anæmic cases a hæmic murmur may be heard over the base of the heart. In some cases acute dilatation has been observed, with a tricuspid murmur.

Complications and Sequelæ. Apart from heart complications which have been mentioned, *pleuritis*, *pneumonia*, and *bronchitis* occur in from 10 to 15 per cent. of the cases. They are frequently bilateral, and are very much more common in rheumatic fever with pericarditis or endocarditis than in simple rheumatic fever. Moreover, the pulmonary complications are frequently latent, and would be overlooked but for the daily physical examination of the chest. On the other hand they may develop with great suddenness, and what appeared to be a full-blown pneumonia may subside suddenly as a fresh joint is affected. They behave more like sudden active congestions than true pneumonias. Rheumatic pleurisies are characterized by the rapidity with which effusion takes place, the persistence of pain in the side during effusion, the tendency to involve both sides in succession, the readiness with which the effusion is absorbed, and their acute course.

Nervous System. The most common complication of the nervous system is *delirium*, which is generally associated with insomnia and hyperpyrexia, but the latter is not constant. These brain-symptoms generally appear in the second week of illness, and about the time of convalescence, or while the joints are still inflamed. The delirium may be low and muttering, accompanied by ataxic symptoms or even by tremors and spasms of muscles ; or it may be furious. In favorable cases a deep sleep ushers in recovery ; or, in unfavorable cases, the delirium persists with adynamia, the patient dying in collapse or coma, preceded or not by convulsions.

Chorea sometimes occurs as a complication, but it is more common as a sequel of mild cases in children. Cerebral meningitis occurs occasionally, especially when there is ulcerative endocarditis. Cerebral embolism is another rare complication.

Various spinal symptoms occur in some cases, at times with, and at times without, demonstrable lesion of the cord or its membranes. Tetanus, myelitis, and spinal meningitis may all be simulated. Perhaps these symptoms are due to high temperature ; but very high temperatures are met with without the occurrence of any cerebral or spinal symptoms.

Nephritis is rare, but sometimes hemorrhage into the kidney occurs with its usual symptoms. Peritonitis is extremely rare.

Various erythematous skin-eruptions are seen from time to time, and occasionally purpura. Subcutaneous nodosities have been described by several writers. They are attached to the tendons, fascia, and periosteum, and are most frequent on the back of the elbow, the ankles, and patella. They are painless, and may occur in any form of rheumatism.

Diagnosis. Rheumatic fever is distinguished from *gout* by the profuse acid and acrid sweating, the tendency to involve a number

of joints, and particularly the larger ones, by the greater intensity of constitutional symptoms, by the great liability to heart-complications, and by the absence of uric acid from the blood.

It is distinguished from *pyæmia* by the wandering character of the inflammation; the acid sweats; the absence of any antecedent condition which would develop purulent foci—such as injuries, abscesses, or specific eruptive fever; the absence of chills, and the fact that in rheumatic fever the sweats are constant, whereas in pyæmia they follow a fall in the temperature. Cutaneous abscesses do not occur in rheumatism, and after its subsidence the joint's usefulness is not impaired.

Acute synovitis resembles rheumatic fever, because in both occur symptoms of pain, tenderness, and swelling in connection with a joint. Usually, however, in synovitis but one joint is involved, and there is a history of exposure to cold or injury. The effusion is limited to the synovial sac of the joint, is frequently abundant, and fluctuation can easily be detected. The constitutional symptoms are much less marked than in rheumatism.

Milk-leg, or *phlegmasia alba dolens*, differs from rheumatism in that it usually occurs in women after confinement, or as a complication or sequel of fever, as typhoid fever. Usually one leg is affected, or part of the leg, especially the calf. This becomes tense, tender, uniformly swollen, and the seat of great pain. The leg is moved with much difficulty. The femoral vein may be found to be knotted and tender. There is almost always evidence of antecedent disease.

Acute periostitis when close to a joint simulates rheumatism. But the tenderness and heat are not in the joint itself; they are superficial, and are associated with less swelling. Pitting on pressure is common; and circumscribed fluctuation usually discloses the presence of suppuration. Pyæmic symptoms are added to the local symptoms, particularly if osteitis or osteomyelitis is present.

The articular symptoms of *glanders* are to be distinguished by the occupation of the patient, the mode of onset, the associated symptoms, especially one or more pustules, and the fact that the painful joints are not so apt to be swollen and red as in rheumatic fever.

In *syphilis* joint-pains frequently occur, but their character is made out by the fact that the joints are not inflamed, and that the pain is much worse, or occurs only at night, and by the history of the patient and the therapeutic test.

In some diseases of the brain and spinal cord joint-inflammations of *trophic* origin occur. They are distinguished by the coexistence of some lesion of brain or cord, with hemiplegia or other palsy, and of other trophic changes, such as bed-sores, atrophied muscles, loss of hair, shiny skin, and defective growth of nails.

Subacute Articular Rheumatism.

In some instances the joint-inflammation is less severe, and is accompanied by only slight fever. One or more joints may be affected. It differs from the ordinary form in being milder in degree and more persistent, lasting sometimes for months. It is generally subacute from

the beginning, but may be the type present in those who have had several attacks of rheumatic fever and have been left in a very sensitive condition. Rheumatic fever is usually subacute in children, and often only one joint is involved. Cardiac complications are more frequent than in adults, and chorea may occur as a sequel. Erythema nodosum and subcutaneous nodosities are more common in children.

Chronic Articular Rheumatism.

In this form the patient has pain and stiffness in one or more joints, or in the contiguous tissues. The joints most frequently affected are the shoulder and knee. The pain is more or less constant, but worse in damp weather or on the approach of a storm, and worse also at night in many cases. Conversely, it is better in warm, dry weather. There is not much if any tenderness, and rarely any swelling or elevation of temperature. The joints very frequently crack and grate on motion. In the interval between the attacks there is no impairment of the usefulness of the joints. In very chronic cases there may be some atrophy of muscles and permanent stiffness, even fibrous anchylosis.

In some cases there are repeated attacks of subacute articular rheumatism, accompanied by the usual symptoms and joint-effusions.

Chronic articular rheumatism is distinguished from *chronic gout* by the fact that there is no special tendency to involve the great toe, by the absence of the deformities resulting from gout, and the absence of deposits of sodium urate in the ears, fingers, and around the joints.

Gout.

A disease characterized by specific arthritis, associated with uric acid in the blood and the deposit of sodium urate in the joints, or manifesting itself as a diathesis in which occur other inflammations of non-articular tissues and various disturbances of functions of organs, the blood also containing uric acid.

Gout is common in Europe, particularly in England, but in its articular form is rare in this country. There is an hereditary predisposition in from 50 to 60 per cent. of the cases. It results from overeating of rich foods and the drinking of malt liquors, associated with insufficient exercise and excretion. Garrod has called attention to its association with lead-poisoning. Paroxysms are induced by indiscretions in eating or drinking, by nervous shock or great mental strain, by exposure to cold or injury, or by overwork and sexual excesses.

The characteristic phenomena of gout are preceded for a variable time by acid flatulent dyspepsia, colicky pains in the stomach and bowel, constipation alternating with diarrhœa, and scanty, heavily loaded urine. Accompanying these dyspeptic symptoms often are impairment of physical and mental vigor, irritability of temper, and hypochondriasis.

In other cases the premonitory symptoms are palpitation of the heart, or dyspnœa resembling asthma, or various nervous symptoms, as drowsiness, insomnia, or headache.

In *acute articular gout* the onset is often sudden, especially in the first attack. The patient may go to bed in apparent health, but wake up early in the morning with a feeling of discomfort or uneasiness, usually in the great toe. In some cases the pain is agonizing from the first. The patient finds he is unable to step upon the foot without torturing pain. The ball of the great toe is hot, swollen, red, and exquisitely resentful of the slightest touch or jar of the bed. The veins are swollen and the joint stiff. There is slight fever, perhaps chilliness, thirst, coated tongue, constipation ; scanty, high-colored urine, depositing urates on cooling ; the skin is warmer than normal, and there is slight perspiration. The pain usually abates during the day and increases at night. It is aggravated by motion and attended by painful muscular cramps. By the end of the first day or two the swelling increases and the pain lessens, owing to diminished tension of the part. Pain on motion is still great, however, and without treatment may continue for a week or two ; under treatment the paroxysm subsides in four or five days.

Both great toes may be attacked in the first seizure, more often alternately than simultaneously, and sometimes other joints than those of the toes are affected.

After the subsidence of an attack the urine contains a larger quantity of uric acid, and the patient feels better in health and spirits than for some time. A second attack may be postponed for several years, but usually after that the intervals between them steadily diminish, until an attack recurs every few weeks or months, and the patient may be scarcely ever free from it. Other joints than the toes, particularly those of the fingers, become involved in subsequent attacks.

The Blood. Neusser has attributed to gout and the uric-acid diathesis the presence of granules, observed after staining, in the white corpuscles, but they have been found in other affections, and are not diagnostic. The nature of many otherwise obscure gouty manifestations or arthritic changes may be determined by an examination of the serum of the blood. Collect the serum which accumulates in a blister and examine for uric acid. (See Blood.)

Chronic gout results from repeated acute attacks. It is characterized by deformity of the affected joints, around which are deposited chalk-stones (tophi) of sodium urate. Similar deposits occur in the helix of the ear. The first appearance is that of a clear vesicle under the skin, which subsequently becomes chalky-white and solid. The deposits of sodium urate occur not only in the cartilages of the joints but in the ligaments and bursæ also, resulting in great impairment of motion and deformity. "In extreme cases an appearance is presented by the hand very closely resembling a bundle of French carrots with their heads forward, the nails appearing to take the place of the stalks" (Garrod).

Gouty abscesses consist of collections of liquid and solid sodium urate, which discharge, with or without pus, through the skin. A patient may have a number of them with but very little impairment of the general health. They may even act as a helpful vent to the system.

In so-called *retrocedent gout* the external joint-manifestation is sup-

pressed or replaced by an internal inflammation, as one of the serous membranes.

Gout attacks the *nervous system*, causing headache, delirium, and sometimes apoplexy, apoplectiform seizures, epilepsy, mania, various neuralgias, and spinal symptoms.

It also affects the *heart* and *bloodvessels*, causing valvulitis and chronic arteritis.

The symptoms presented by the *digestive organs* have been mentioned. They are often premonitory of an attack.

The *kidneys* may be affected, causing typical contracted kidney, or there may be chronic *cystitis* and *urethritis*.

Rheumatoid Arthritis.

Rheumatoid arthritis, or rheumatic gout, is an affection characterized by acute or chronic inflammation of the joints, of progressive character, and resulting in deformities. It is attended with very little fever, and occurs apart from any known systemic disease.

It may be acute or chronic. The *acute* form differs but little in its manifestations from acute rheumatic fever. Several joints are enlarged, tender, and painful. Constitutional symptoms, such as fever, loss of appetite, frequent pulse, thirst, and furred tongue, occur as in rheumatism. Profuse acid sweats, however, are absent, and so is the tendency to serous inflammations. Moreover, while the larger joints, as in rheumatism, may be affected, the smaller ones also, especially of the fingers and toes, are inflamed and often the seat of serous effusions. Furthermore, the inflammation persists in the affected joints and does not jump from one to another. Instead of disappearing in a few weeks, it drags on for a much longer time. The pain subsides, but the swelling persists, and permanent deformity results in at least some of the joints. The muscles of the arms and legs waste and are affected with painful spasms.

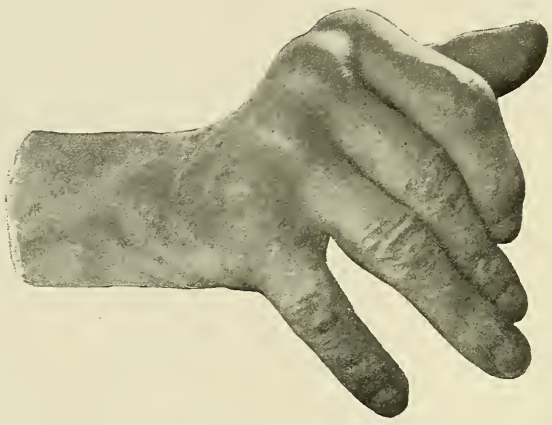
The disease is most common in young women exhausted by repeated pregnancies or prolonged lactation, and is favored by poverty, privation, and cold.

The *chronic* form is much more common. It also attacks most frequently young women who are exhausted or are subjected to great fatigue. There is pain, numbness, or formication in a joint, as the knee. The joint becomes tender, painful, and may be slightly swollen.

This subsides after a while, but sooner or later the same joint or another one becomes affected, the process is persistent, one joint after another is attacked, and gradually all the joints may become greatly distorted, enlarged, and the seat of contractions. There may be no impairment of general health, or, at most, only dyspeptic symptoms. The progress is interrupted by remissions from time to time. Pain may be severe and subject to nocturnal exacerbations. The shape of the joints is altered by the effusion into the joints and adjacent bursæ, by thickening of the tissues around the joints, growths of new bone on the joint-extremity of the bones, absorption of the articular cartilages, and growths of new cartilage in the synovial sheaths, relaxation of

ligaments, muscular contractures, and luxation of the joints. The joints crack and creak like rusty hinges, are sore and stiff, and the attached muscles are affected with painful cramps. (See Fig. 29.)

Fig. 29.



Rheumatoid arthritis.

Great enlargement of the joints at times occurs from the causes already mentioned and from infiltration of the overlying tissues. The enlargement is rendered more conspicuous by the atrophy of adjacent muscles. (See Fig. 14.)

In addition to the articular symptoms other phenomena attend the process. One of the more common is increased *frequency* of the *pulse*. Although the patient is afebrile, the average pulse-rate is 100 to 120, or even more. Moreover, the pulse is soft and compressible, in contradistinction to the pulse of gout and rheumatism. It is worth noting that a return to the normal frequency of pulse is a sign that the process of the disease is arrested, although the joint-lesions remain.

The *skin* is characteristic. It is soft and often much freckled, while the complexion is fair. C. T. Griffiths has observed the pigmentary cutaneous changes, along with neural symptoms, prior to the joint-manifestations, and describes two forms : a diffuse melasmic discoloration, and dark-brown spots resembling moles, but not raised. Moisture of the skin with clamminess is common. It is limited to the palms of the hands, or may occur in the distribution of certain nerves. The sweats are not acid ; they are usually local, but may be profuse. *Pain* independent of the joint-lesion is due to neuritis, and may precede the joint-trouble. It is not merely confined to the nerve-trunks, but affects the smaller branches which are distributed to muscles, as the base of the thumb. *Numbness* and tingling are often present.

The progress of the disease is pretty steadily worse. In extreme cases not only are the limbs crippled, deformed, and helpless, but there is fixation of the cervical spine and of the articulations of the jaw, so that the patient cannot move the head or masticate food.

The following describes the characteristic deformity of the hand: The first phalanx of the fingers is either flexed upon the metacarpus or extended, and the terminal phalanx in like manner is either markedly flexed or extended upon the second, or these two phalanges are kept at a straight line, while the first phalanx is, as usual, decidedly flexed upon the metacarpus. The hand is pronated and the fingers turn toward the ulnar side (Palmer Howard and Charcot). (See Fig. 29.)

The foot is abducted and flattened, and the great toe abducted across and above the other toes. Rarely it may be beneath the other toes. The metatarso-phalangeal joint is enlarged.

A variety of the disease is sometimes met with, chiefly in old persons (senile arthritis), in which the tendency is to involve one or two joints, particularly the hip, or hip and knee. It is of slow progress, and is otherwise attended with the same deformities as the usual polyarticular form.

Rheumatoid arthritis is distinguished from *gout* by the absence of heredity and by its development under the exhausting influences of repeated pregnancies, lactation, poverty, and malnutrition. Rheumatoid arthritis is progressive, with occasional remissions; gout occurs in successive attacks, with intermissions. Uric acid is absent from the blood in the former and is present in gout. Rheumatoid arthritis in the vast majority of cases is subacute or chronic. The acute form is distinguished from acute gout by the duration of the paroxysm and the absence of intermissions; by there being less heat, swelling, and redness of the joints, and less infiltration of the soft parts; by the fact that large and small joints are involved, and that there is no special tendency to inflammation of the great toe.

From *chronic gout* rheumatoid arthritis is distinguished by the absence of hereditary predisposition, of repeated acute attacks, and of the causes of gouty paroxysms—indulgence in sugars, acids, malt liquors, etc. Moreover, rheumatoid arthritis most frequently begins in the hands, and is symmetrical and bilateral. Gout has a predilection for the great toe, and is unilateral. Again, gout attacks well-fed males most frequently after the age of thirty years, while rheumatoid arthritis tends to attack women under the depressing influences already mentioned. It may, however, occur in both sexes, and even be associated with gout.

Rheumatic fever is distinguished from acute rheumatoid arthritis by its tendency to involve the larger joints, its erratic course, acid sweats, and heavy deposits of urates from the urine, its shorter course, its tendency to heart-complications, and its subsidence without impairment of the usefulness of the joints.

Chronic articular rheumatism is distinguished by the preceding history, the tendency to seasonal exacerbations, by its involving fewer joints, and not being so symmetrical in the joints affected. It does not produce so great deformity as is common in rheumatoid arthritis, nor is it so likely to affect the vertebræ and jaws. The existence of valvular heart disease or a history of antecedent chorea is in favor of rheumatism.

The joint-affections of *locomotor ataxia* are distinguished by the associated symptoms of incoördination and absent knee-jerk, by their

sudden onset without pain or fever, by the occurrence of large effusion into the joint, with subsequent disorganization, fractures, and dislocations.

Gonorrhœal arthritis is distinguished by the history of gonorrhœa or the existence of a discharge from the urethra, by the tendency of the disease to attack the larger joints, particularly the knee or shoulder, and to become fixed in one, not wandering from one to another. The affected joint suffers effusion, and the synovial membranes and bursæ are inflamed. The process is very chronic but indolent, and the heart rarely becomes affected.

Scurvy.

The *joints* are swollen, painful, and tender in about one-third of all cases of scurvy. When to these joint-symptoms the spongy gums, the hemorrhages, the anæmia, and cachexia are added, scurvy may be suspected.

Scorbutus, or scurvy, is a constitutional condition brought about by a long-continued diet deficient in fresh vegetables. It is characterized by pallor, great physical weakness and mental sluggishness, dyspnœa, subcutaneous and submucous hemorrhages, a swollen, spongy condition of the gums, and a brawny induration, especially of the calves and hams.

The onset of the disease is gradual, and is marked by a peculiar dirty-yellow or greenish *pallor* of the face, associated soon with an apathetic expression of the face, physical *weakness*, and decided lack of customary energy. The appearance is so characteristic that patients are said to detect it readily in others, though unaware of it themselves. Sleep and digestion are good, but rheumatoid pains may be complained of. Other prominent subjective symptoms are fatigue on slight exertion, dyspnœa, faintness, and despondency. In the course of a week or two petechiæ appear upon the lower extremities, especially around a hair as the centre. (See page 128.) Depending upon the severity of the case there are also bullæ, vibices, and ecchymoses. Brawny induration, due to deep effusion of blood, occurs, especially in the calves and hams, producing considerable pain on flexure of the knees.

There is no fever apart from complications. The pulse is frequent, weak, and small, and the first sound of the heart and the impulse may be very faint.

The face is swollen and of a dirty, possibly greenish-yellow color, according to Bird, Buzzard, and others; in some cases the eye and its surroundings are the only parts exhibiting signs of scurvy at this time. "The integument around one or both orbits is puffed up into a bruise-colored swelling. The conjunctivæ covering the sclerotic is tumid and of a brilliant red color throughout, and about an eighth of an inch in thickness or elevation above the cornea, leaving the cornea at the bottom of a circular trench or well."[1] The condition is not inflammatory. These cases often terminate fatally.

[1] Buzzard: Reynolds' System of Medicine, 1880, vol. i. p. 451.

The gums swell almost always, become spongy, and bleed upon the slightest irritation. They are dark cherry-red in color and look not unlike a split cherry. Sometimes they swell, so as almost to hide the teeth completely and even to protrude the lips. The breath has a heavy, sickening odor, and the teeth sometimes drop out of their sockets.

In addition to the cutaneous and gingival hemorrhages, hemorrhages occur from the nose and other mucous surfaces, and effusions take place into the lungs, intestines, pericardium, and pleura, associated with inflammatory products. There may be no physical signs on the part of the lungs to account for the dyspnœa, or some dulness and bronchial breathing, or a few râles, may be detected.

A very peculiar symptom, and sometimes the earliest, is hemeralopia, nyctalopia, or night-blindness, in which the patient can see during the day but not by moonlight, and apart from artificial light is totally blind at night.

So-called *scurvy-rickets* is more or less common in infants fed on artificial food exclusively or on sterilized milk. It is therefore limited to the first four or five years. The symptoms of scurvy are added to those of rhachitis. In the eight cases I have seen, the most pronounced features were those of weakness, anæmia, polyuria, restlessness, the scorbutic gums, local periostitis, particularly of the tibia, sometimes periarticular inflammation, and always a general tenderness of the body, as in rhachitis.

The Tabetic Joint. In forms of nervous diseases, particularly in sclerosis of the posterior columns, secondary joint-involvement sometimes occurs. The change in the large joints is preceded by pain, stiffness, and inability to use them. Gradually nutritive changes take place. At first there is boggy swelling. The cartilages become eroded, the heads of the bone waste, the ligaments ossify, and irregular bony growths project. Wasting of the head of the femur is followed by dislocation. Sometimes an effusion takes place in the joints, and there may be periarticular œdema. The large joints are most commonly affected—the knee, hip, ankle, and elbow. Injury excites the abnormal atrophic process. When the tarsal bones and the articulations are affected the foot becomes flat, and the tarsal and metatarsal articulation and the tarsal bones project forward or backward. This is called the tabetic foot.

The Joint of Hysteria. Symptoms of joint-disease are seen in hysteria. Pain and fixation of the joint are sometimes complained of. The joint rarely undergoes organic changes, but sometimes a plastic infiltration of the connective tissue outside of the capsule does occur. The hysterical nature of the pain and immobility are recognized by the absence of a cause for joint-lesion, the absence of fluctuation, or of signs due to erosion, by the association of the local symptoms with the phenomena of hysteria, but, more particularly, by the fact that contraction and even wasting precede the joint-symptoms. In true affections of the joint both occur *after* the joint has become diseased; in hysteria muscular contraction will take place *first*.

The knee is the joint usually affected. Care must be taken not to be deceived by local vasomotor changes of hysterical origin which may

be observed under the surface of the joint. This local increased temperature is not associated with general fever, however, while the vasomotor changes indicated by the swelling of the skin, increased tension, and the shining appearance, with increased sensibility, are not persistent, but occur once or twice in the twenty-four hours. In a remarkable case of Mitchell's the local vasomotor change took place at night. The temperature of the knee which was affected increased three or four degrees, while the pulse remained at 80. The local symptoms of heat, redness, swelling, tension, and increased pain passed away by three o'clock in the morning. The fact that the same symptoms could be brought on by handling the knee, or by pressure upon the patella, pointed to its vasomotor origin.

In joint-cases of hysterical origin the reflexes must be studied. They do not change, and the electrical reactions are normal, although there may be atrophy from disuse, but not to the degree that occurs in organic disease. The muscles may be contracted, but, as previously noted, the contracture is primarily a relaxation, which takes place if the tension is removed. Concerning these vasomotor changes, Sir James Paget's expression, "A joint which is cold by day and hot by night is not an inflamed joint," is a safe guide to the recognition of an hysterical joint. When the joint becomes hysterical after injury it is most difficult to ascertain its true nature.

Special Joints. The three joints that should concern the student more particularly are the shoulder, hip, and knee. When symptoms are referred to either of these joints they should not be passed over lightly. Grave consequences have followed the attributing of hip-joint inflammation to rheumatism when it was of tuberculous origin. Not only has hip-joint disease been mistaken for rheumatism, but the mistake has even been made of considering the process to be going on in the knee instead of in the hip. This is because there is often flexion of the leg, and because pain is so often referred to the knee-joint.

On the other hand, cases of hip-joint disease have been mistaken for suppuration in the pelvis or in the iliac fossa. Typhlitis or appendicitis has frequently been mistaken for hip-joint disease.

In the case of the shoulder-joint there is danger of confounding neuritis of the circumflex nerve, and consequent paralysis of the deltoid, with affections of the joint. Although the patient is unable to move the joint, it is still readily moved by the physician, and the physical signs of joint-inflammation are wanting.

CHAPTER XIV.

THE DATA OBTAINED BY OBSERVATION—(*Continued*).

Chills; fever; subnormal temperature.

THE TEMPERATURE.

Before discussing the subject of fever, it is not illogical to consider *chills*.

Chills.

"Chills" vary from a passing "creep" or cold sensation, extending up and down the spine, to the "shake" or true rigor of one-half hour or even longer. In infectious diseases the milder form is of as much significance as the more severe. The rigor may be so violent and prolonged as to terminate fatally. It must be distinguished from the algid stage of cholera and the coldness of collapse. The chill is attended by general tremor or shaking, chattering teeth, cold extremities, pallid face, often parched blue lips and finger-tips. Notwithstanding the peripheral coldness and the extreme sensation of cold, the internal temperature rises, and may be 104° to 107°.

Clinically, a chill or rigor marks the onset of severe infection, as pneumonia. "Chills" are symptoms of some affections, as malaria. They are seen in the course of many diseases, as typhoid fever, tuberculosis, and septicæmia. In typhoid fever they disclose the occurrence of a secondary infection or a mixed infection; they may be due to antipyretic treatment by coal-tar remedies (Osler) or result from constipation. Endocarditis is attended by daily chills or they occur at irregular intervals. Pyæmia and septicæmia, purulent inflammations (infections), inflammations of the biliary or renal passages, stone in the biliary canal, or the pelvis of the kidney (see Intermitting Fever) are frequently attended by chills. The morphine habit gives rise to chills, with some fever.

Fever.

In conditions of health the body-temperature is maintained constantly at about 98.6° F. (37° C.). This stability of temperature is due to the central regulating apparatus called the thermotaxic mechanism, which controls the production and the dissipation of heat. Fever is a condition characterized by an increase of temperature, with usually increased disintegration of nitrogenous tissue. The muscles and large glands, as is well known, are the chief seat of heat-production. Both heat-production and heat-dissipation are believed to be under the control of the nervous system, either through the motor nerves or

special nerves which pass with them to and from definite centres in the brain, called heat-centres. In conditions of disease this thermotaxic mechanism may be altered, so that the normal temperature is increased or lessened. (1) There may be elevation of temperature from diminished dissipation of heat, though not necessarily increased nitrogenous disintegration and disordered function. Or (2) there may be increased production of heat with diminished dissipation, hence the temperature will naturally be higher than if increased heat-production were accompanied by normal heat-dissipation. (3) There may be increased heat-production and at the same time increased heat-dissipation, in which case there would be the increased waste of fever with or without any elevation of temperature. (4) It is possible that heat-dissipation may be greater than heat-production, or that the thermotaxic mechanism may be disturbed, so as to promote loss, in which case there will be subnormal temperature.

Mode of Determination of Fever. The temperature of the body can be roughly estimated by the hand of the physician, but this method is open to many sources of error. The skin is at times hot, and gives a deceptive sensation of considerable elevation of temperature, whereas when tested by the thermometer the temperature is found to be but slightly or not at all above normal. So, too, when the skin feels cold and clammy in phthisis and during a chill from any cause, the actual temperature of the body is decidedly above normal, and may be as high as 103° or 104°. To insure accuracy, therefore, it is now almost the universal custom to employ clinical thermometers. They are of a convenient size and shape for insertion under the arm or into the mouth, rectum, or vagina. The better ones are provided with an indestructible index, so that the mercury in the capillary tube remains stationary at the highest level to which it rose when the thermometer was in the mouth or axilla. When not provided with such an index the reading must be made when the thermometer is still in position.

Thermometers vary in the accuracy with which they register temperature. The best ones are compared with an acknowledged standard, and sold with a slip of paper which gives their fractional variations from the standard. When the exact temperature is a matter of great importance, it should be taken in the *rectum* or *vagina*, as their temperature is more nearly that of the body. It is of advantage to take the temperature in the rectum of children or in patients who are comatose. This situation is also a good one to select when a bath is being administered. If possible, scybalous masses should be removed from the rectum. At least an incorrect reading may be obtained if the thermometer should happen to be plunged into the fæces; this must be guarded against. From motives of delicacy, however, the axilla is to be preferred to the rectum and vagina on all ordinary occasions. The temperature it records is somewhat less than a degree below that of the rectum. The temperature of the *mouth* is above that of the axilla and below that of the rectum. It has some advantages over that of the axilla, being more accessible and recording the temperature more quickly and more accurately. Nevertheless, as the physician's thermometer is carried from patient to patient, some place should be

selected which is less capable of absorbing disease-germs than the mouth. The *axilla* is, therefore, by common consent the usual place for taking the temperature. Observe two precautions: (1) Before introducing the thermometer see that there is no undue moisture; if there is, the axilla should be wiped dry, otherwise a lower than a true reading will be obtained. (2) See that the instrument is inserted into the armpit and does not project beyond the posterior fold, and that it is not caught in a fold of the undershirt or night-dress. After the thermometer is in position the arm should be brought gently across the chest and kept in that position until the instrument is withdrawn. The arm should not be held rigidly, as such muscular action increases the hollow of the armpit and may keep the sides apart, instead of in contact, as they should be to make a correct reading. The length of time required to take the axillary temperature will depend upon the instrument used; generally from five to eight minutes are required. Some very delicate thermometers register in one minute, but they are too fragile for ordinary use. If the index is in such a position that it can be seen, it is proper to withdraw the thermometer when the mercury has ceased to rise for two minutes.

The index, of course, must be shaken down to normal, or slightly below normal, before the thermometer is again ready for use; and the instrument must be carefully cleansed after use.

In children who are restless the temperature may be taken in the groin, as the folds of fat readily admit of completely enveloping the bulb of the thermometer. The height to which the mercury rises will correspond to the temperature of the axilla. The temperature of the *urine* corresponds exactly with that of the body, if taken when freshly passed and during the act, a method only applicable in the case of males. Sometimes this method of securing the temperature is resorted to, particularly in patients who may act as malingerers, when it is desirable to have the temperature taken in the physician's presence.

If the *mouth* is selected as the place in which the temperature is to be taken, care should be exercised that the thermometer is placed under the tongue, or along its side between it and the lower jaw, and retained in position by the lips of the patient. If the teeth are set firmly on the thermometer, it may be broken, or, what is of still greater importance, it will be tilted out of position and a correct reading will not be obtained. The lips should be closed and breathing be carried on through the nostrils. Four to seven minutes is sufficient time to allow it to remain in position. The patient should not have taken ice or anything cold prior to the observation.

Observations of the temperature should be made at least twice a day, in the morning and evening, and, as far as possible, at the same hour on successive days. It is frequently desirable to have the temperature taken every two or three hours, and sometimes at more frequent intervals. This is particularly the case if observations of the indications for, and the effect of, antipyretic treatment are to be made.

In obscure cases the observations should be repeated at night as well as during the day. In this manner the presence of unsuspected tuber-

culosis may be revealed, or the occurrence of suppuration in some portion of the body definitely determined. It should not be forgotten, however, that the temperature may be taken too frequently for the patient's good, the disturbance of his needed rest being distinctly harmful.

As the general range of temperature and its diurnal variations are of more importance than the absolute temperature at any one time, thermometers not perfectly accurate in their reading are still good enough for clinical and therapeutic purposes.

Physiological Variations of Temperature. The temperature is subject to *physiological variations*. 1. It rises from seven or eight in the morning until seven or eight in the evening, at which time it reaches its maximum. It then begins slowly to fall, reaching its lowest point in the early hours of morning, between two and four. This *diurnal fluctuation* does not usually amount to more than a degree. 2. *Exercise*, etc. Violent exertion raises the temperature, and so does a heated atmosphere, cold having a contrary effect. 3. *Age*. In infants and young children, up to puberty, the temperature has a somewhat higher range, and is subject to greater variations than at a later period. In very old persons the temperature may be subnormal. The *normal* axillary temperature of adults is 98.6° F. The period in the twenty-four hours in which the temperature is at its lowest ebb is from 12 P.M. to 4 A.M. It may then be subnormal. The writer has known an over-cautious parent to make this physiological fall the subject of meddlesome observation and ill-judged treatment.

Pathological Variations of Temperature. An elevation of temperature above the normal, not to be accounted for by external heat or severe exhaustion, may be considered febrile, and is pathological. The range of febrile temperature varies from above normal to 105° or 106° in ordinary cases. A range above 106° may occur, but is not usually compatible with life. Certain terms have been applied to various degrees of temperature, to indicate in a general way the degree of fever :

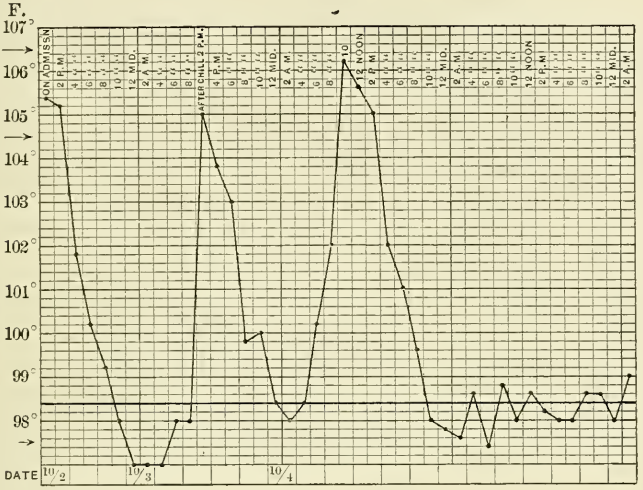
Below	35° Cent. = 95.0° Fah. 36 " = 96.8 "		Very low or collapse temperature.
About	36½ " = 97.7 "		Subnormal temperature.
Normal	37 " = 98.6 "		Normal temperature.
About	37½ " = 99.5 " 38 " = 100.4 " 38½ " = 101.3 "		Slightly above normal or sub-febrile temperatures.
About	39 " = 102.2 " 39½ " = 103.1 "		Moderately febrile temperature.
About	40 " = 104.0 " 40½ " = 104.9 "		Highly febrile temperature.
Above	41 " = 105.8 "		Hyperpyretic temperature.

(From FINLAYSON.)

The Degree of Danger. In general the degree of danger to the patient increases with the height of the fever, but the duration of the high fever modifies this greatly. A temperature of 106° on the second or third day of an acute lobar pneumonia is not rare, such cases fre-

quently ending in recovery, while a temperature of 105° in the second or third week of typhoid fever is of much graver significance. Da Costa has reported a case of cerebral rheumatism in which the axillary temperature reached 110°, yet the patient recovered. In the case of injury of the spine, reported by Teale, the extraordinary temperature of 122° was recorded, and the temperature-range for days was between 112° and 114°. The patient recovered.

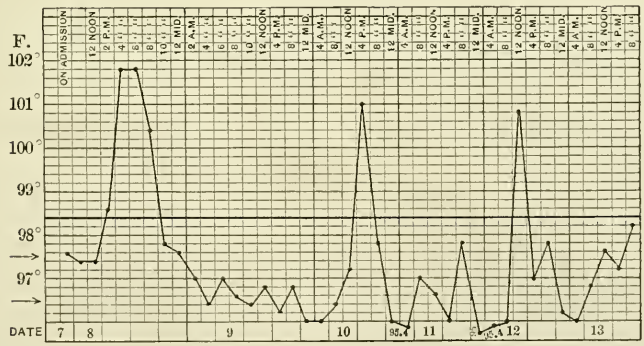
FIG. 30.



Malarial intermittent fever. Quotidian type.

The Types of Fever. Fevers are divided, in accordance with the character of their range, into certain definite types. The types may

FIG. 31.

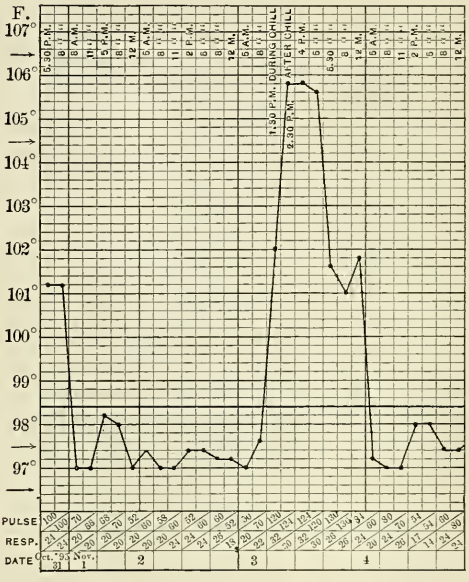


Malarial intermittent fever. Tertian type.

be indicative of special processes. It is certain that the recognition of a peculiar type forms a positive aid to diagnosis. The fever that con-

tinues for more than two days, in which the difference between the daily maximum and minimum of temperature is less than 2°, is known as *continued* fever. (See Fig. 36.) The fever existing more than two days, in which the daily difference is greater than 2°, is known as *remittent* fever. Further, a fever in which there is a rise of temperature followed by a fall to or below the normal, occurring periodically,

FIG. 32.



Malarial intermittent fever. Quartan type.

is known as *intermittent* fever. The paroxysms may occur daily, every second or third day, or once a week. When the paroxysms occur daily, the intermittent fever is of quotidian type (see Figs. 30 and 33); every second day, tertian type, one day intervening without fever (see Fig. 37); every third day, quartan type, two apyretic days intervening (Fig. 32).

The Course of the Fever. Fevers frequently have a definite course, known as (1) the initial stage; (2) the fastigium; (3) the period of defervescence. During the *initial* stage the temperature rises higher each hour (or if extended over days, each day) than the preceding hour or day—in this latter instance interrupted by the daily fluctuations. The stage may last from a few hours, as in a paroxysm of intermittent fever, to four or five days, as in typhoid fever. In this stage we have a chill such as characterizes the onset of an intermittent fever, or the recurrent chills or chilliness with headache and backache that attend the first four or five days of typhoid fever. During this stage, also, the heat-dissipation from the cutaneous surface is diminished and the total heat-dissipation is less. When the hand is placed upon the patient the surface will be found to be cool, whereas

the temperature in the mouth or rectum will be found to be far above the normal. The patient complains of the coldness or chilliness, and the low temperature of the surface is indicated by the shrunken hand, the pallid, pinched face. The peripheral arteries are contracted, and hence cause diminution in the amount of blood to warm the skin and to compensate for the loss by radiation and conduction. This peripheral contraction is the cause of the chilliness and the fall in the temperature of the skin.

During the second period of the course of pyrexia—the *fastigium*—the temperature of the body attains the highest point, and remains almost stationary, or may vary but a degree or two between maximum and minimum. It may last a few hours or from two days to three or more weeks, during which time it may oscillate to the maximum point of the first day. The temperature of the surface of the body is about the same as that of the deep parts, particularly in cases of pneumonia, measles, and scarlet fever. In typhoid fever, acute rheumatism, and phthisis, during this period, there may be a difference in the external temperature and the temperature taken in the cavities, as the mouth or rectum. More or less antagonism between heat-production and heat-loss exists under these circumstances. The latter may be greater than the former, if the skin perspires freely, as in rheumatism. The temperature then remaining high indicates that the production of heat must be proportionately increased, and hence far greater than in the cases in which the external and internal temperature are nearly the same. (See Fig. 34: the fastigium here occurs in the first three days. In Fig. 37 the fastigium lasts until the crisis.)

In the period of *defervescence* the temperature falls to the normal. In this period an attempt is made by the economy to return to a physiological state, in which heat-production and heat-loss are evenly balanced. The state of pathological pyrexia has come to an end. The termination may be by *crisis*. (See Figs. 31 and 37.) When this takes place the perturbation of the thermotaxic mechanism must be very great, but the normal state is at once resumed. In other cases the termination is by *lysis*—the temperature falls a degree or two each day until the normal is reached. (See chart of Typhoid Fever.) It seems that the thermotaxic mechanism of health is restored with difficulty. In some cases, in the period of defervescence, the aberrations are very remarkable. It seems as if the thermotaxic mechanism which controls heat-loss was in a convulsive state. The temperature rises and falls irregularly, gradually resuming the normal only as the strength of the patient increases.

The Mode of Onset; Initial Stage. The onset may be sudden or gradual. 1. The sudden onset occurs in acute diseases, as tonsillitis, pneumonia, and gastro-intestinal disorders of children, in erysipelas, and in intermittent fever. Within a few hours the maximum of temperature is reached. (See Fig. 37.) 2. The mode of onset may be gradual. The initial stage is prolonged under these circumstances, as in cases of typhoid fever. (See chart of Typhoid Fever.)

The Mode of Decline; the Defervescence. A sudden fall of temperature at the termination of a disease is known as *crisis*, which is

also attended by copious perspiration, a "critical sweat," or by the passage of a large quantity of urine, and sometimes by several large liquid stools. The pulse-rate and respirations fall correspondingly with the temperature. (See Fig. 37.)

The defervescence may, however, occupy several days, in which case it is called *lysis*. In this case the sweating is less marked, but may recur for several days. The slowing of the pulse and respiration likewise take place gradually. (See chart of Typhoid Fever.)

Diseases of sudden onset usually terminate with sudden decline, and conversely in diseases with a prolonged onset the decline is also prolonged. Many cases which naturally terminate by crisis may end by lysis. This irregular termination is usually due to a complication. (See Fig. 34.) For instance, in measles, pneumonia is usually the causal complication, while in pneumonia it is empyema or endocarditis.

The Daily Range of the Prolonged Initial Stage and the Fastigium. The daily range of the temperature in fever generally corresponds to the normal variations. That is, the temperature is higher in the evening than in the morning. The difference in the daily range varies in the different types of fever—generally, as previously noted, the continued fevers show a smaller, the intermitting fevers a larger, difference between morning and evening temperature.

Sometimes there is *inversion* of the normal range. The evening temperature is lower than the morning; although a rare condition, this is of serious import. It is seen in the more severe cases of typhoid fever and occasionally in tuberculosis.

Recrudescence. In many cases the fever returns after the temperature has fallen to the normal. This may occur from a number of causes. It may be from perturbation of the nervous system, on account of excitement, over-exertion, loss of sleep, or from indigestion. Slight aberrations, which in health would not modify the temperature, cause pronounced oscillations in illness. Recrudescence, further, may be produced by a relapse. After the afebrile period following typhoid fever, for instance, the temperature may rise and a full recurrence of the disease take place.

The Symptoms of Fever. Pyrexia, or increased temperature, is not the only evidence of fever. The production of heat within the body is not due to increased tissue-change alone. It may be due, for instance, to increased oxidation of sugar, which is part of the substance of the body. Physiologists have found that a high temperature may take place, and yet the quantity of urea and of carbonic acid discharged may not be as great as that of a healthy person who is taking active exercise or who has eaten a large meal. It must be remembered, therefore, that it is not heat-production alone but *alterations of heat-regulation* which cause pyrexia and its phenomena.

Wasting. Wasting of the body is a striking symptom of fever. There is no doubt that even in fever of moderate duration great wasting of the solid structures takes place. At the same time the blood wastes (see observations of Thayer) and the various fluids of the body are also diminished, hence the disorders due to diminished secretion of glands are prominent in the course of fever. Diminution of secretion

in the gastro-intestinal tract, causing thirst, loss of appetite, indigestion, and constipation, indicates the wasting of the fluids. Scanty urine of high color and specific gravity is due to the same cause.

The Pulse-rate. Acceleration of the pulse is one of the phenomena that attend pyrexia. While increased pulse-frequency is the rule, and is, in all probability, a result of the increase in temperature, other circumstances may cause a change in the pulse-rate in pyrexia. Thus, in basilar meningitis, although there may be a high fever, the pulse is not more frequent. On the other hand, some diseases, usually accompanied by fever, as diphtheria and peritonitis, may be afebrile, and yet the pulse be very much accelerated.

Arterial Tension. The rapidity with which the blood flows in fever and the arterial tension do not bear a due proportion to the acceleration of the pulse. The true febrile pulse is not dicrotic. In the early stages of fever the pulse is large and hard, the arterial tension is high, and the vessels full. In the later stages arterial relaxation takes place, and the pulse becomes soft and feeble, and often small, with low pressure. The pulse is rapid, and dicrotism, or even hyperdicrotism, now becomes a prominent feature. The heart beating rapidly empties itself incompletely and discharges less rather than more blood into the arteries. The impairment of the cardiac beat is no doubt due to the degenerations on account of the high temperature, and is not dependent upon any special febrile affection. Such changes also take place in the glands, particularly the liver and kidneys, and are known as parenchymatous degenerations, or cloudy swelling. These changes in the cardiac muscle may induce, in the later stages of fever, thrombi, and cause death from heart-clot.

The Respiration. The respirations are increased in fever, probably because of the close dependence of the regulating centre of respiration on that of the heart. The heated blood acts as a stimulant to the respiratory centre. As proof of this, the hurried respiration of pneumonia ceases as soon as the temperature falls, notwithstanding the fact that the affected part of the lung remains hepatized.

Cerebral Symptoms. Delirium and other nervous symptoms may attend fever. They are not dependent upon the increased temperature of the blood alone. No relation appears to exist between the intensity of the fever and the severity of the delirium. In relapsing fever a temperature of 106° occurs with the mind clear. In certain cases of typhoid fever a temperature of 103° is attended with marked delirium. If fever persists for a short time a low asthenic state, so-called *adynamia*, may develop. Because the symptoms resemble those of typhus fever, the term typhoid is also applied to them, and the condition about to be described is known as the *typhoid state*. The expression is dull and heavy, the capillaries of the face are congested. There are stupor and sluggishness of mental processes, so that the patient is slow in answering questions. The stupor is attended with low muttering delirium, and may be followed by complete unconsciousness. The pupils are contracted, the eye heavy and dull. The patient is so prostrated that he slips down into the bed from the pillow. There is marked subsultus tendinum. The tongue, if protruded, comes out

slowly and is tremulous. It is dry and brown, and the mouth and teeth are covered with sordes. The sensibilities are blunted, so that food and drink are not asked for, or particularly relished if given. Involuntary discharges take place from the rectum and bladder, and the incontinence of retention of the urine arises. The pulse is small, feeble, and dicrotic, the heart-sounds are weak and feeble. The first sound becomes short and snappy like the second, or may be absent entirely. Venous stases take place in the dependent portions, particularly in the back of the lungs.

As œdema or hypostatic congestion advances the breathing becomes shorter and labored. More or less cyanosis then creeps over the general surface. The urine becomes more and more scanty and high-colored, contains albumin, and sometimes blood.

The typhoid state may continue for many days, or even last two or three weeks, although not in so advanced a degree as has been described. It is more likely to supervene when there is excessively high temperature, but it also occurs in the course of a prolonged illness with a temperature of moderate degree—that is, 103° F. Although it is in all probability due to the direct effects of heat upon the nerve-centres and the organs of the body, yet there are cases in which the temperature is not high, and yet all the symptoms of the typhoid state supervene. While the typhoid state is common to *typhoid fever*, it occurs also in *pneumonia* and *septicæmia*, and may even be seen in its most typical form in other conditions in which fever is not a pronounced symptom; thus in *uræmia*, in the later stages of *softening of the brain*, in *paresis*, or in allied nervous diseases the symptoms of the typhoid state are most striking. In this class of cases it certainly cannot be attributed to the fever, but is, in all probability, due to the depressing effect on the nervous system of material which should be excreted from the body, a view which has been advocated by Murchison, Flint, and others.

Ataxia, or the *ataxic state*, in fever is a condition the opposite of the adynamic, or typhoid state. In the latter there is weakness, while in the former there is exhibition of strength. In the latter the nerve-centres and the vital processes are depressed; in the former they are stimulated. Ataxia as an exhibition of strength is characterized by a strong pulse and by active, violent delirium, so that it is almost impossible to keep the patient in bed; by evidence of great muscular strength. The face is flushed, bright-red in color; the eyes injected, bright, and active. The tongue is furred, but is not necessarily dry or brown. The delirium may be constant or paroxysmal, and is often maniacal in character. The temperature of the body is high, and a sensation of intense heat is imparted to the hand when placed on the surface of the trunk. The patient may complain of a bursting, intense headache. If the ataxic state is not controlled after a few days, or at the most a week, the patient becomes exhausted and lapses into stupor, which may proceed to coma. In some forms, particularly in children, convulsions may accompany the excessively high temperature and be followed by coma. The so-called coma vigil may supervene. The same exhibition of strength is shown. Ataxia is seen notably in scarlet

fever, "cerebral" pneumonia, and in forms of typhoid fever. The peculiar behavior of the temperature and nervous systems in this affection and in apex pneumonia, or so-called pneumonia of the cerebral type, have led observers to mistake such cases for actual cerebral disease. Frequently they have been admitted into insane asylums for supposed mania. The true nature of such cases is often mistaken, and, because of lack of attendants, the patients have jumped from the window or done violence to themselves in other ways.

It is as difficult to determine the exact cause of the extreme perturbation of the nervous system in *febrile* ataxia as in adynamia. It may be due to a high temperature, acting on nerve-centres; or to a poison, as the special toxin of the infection which has caused the fever.

The presence of fever may be suggested by *flushing* of the face. This may be general or local. The local flush of phthisis and of pneumonia has previously been referred to. *Dryness* and pungency of the skin occur in fever. In former times the sense of heat was given different attributes, said to be distinctive of various affections. Hence the terms calor mordex, etc. Thus the sensation to the hand of the heat in typhus fever was said to be peculiar and characteristic. The degree of fever was determined by the sense of touch. The thermometer has displaced this method of reckoning temperature. *Sweating* is a condition habitual in some fevers. It may occur throughout the course of the disease, or at certain stages only as instanced by the early morning or night-sweats of tuberculosis. In such cases it is cold and clammy. The same sweatings are common in the fever of deep-seated suppuration and in disease of the bones. Sweating in defervescence marks the occurrence of crisis.

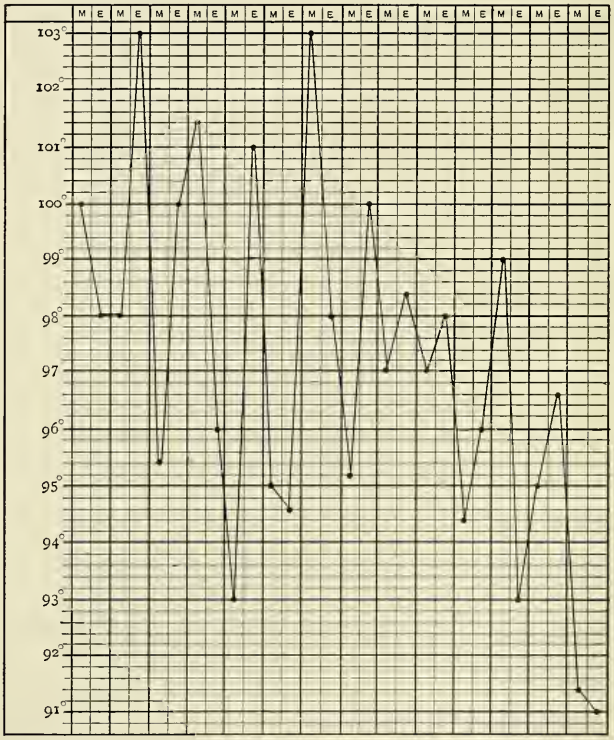
HEADACHE AND PAIN IN THE BACK occur in the acute specific fevers in the initial stage. One or both are nearly always present, but in different affections they have diagnostic significance. Thus severe pain in the back is more pronounced in tonsillitis and smallpox, severe headache in cerebro-spinal meningitis, and protracted throbbing headache in typhoid fever.

Subnormal Temperature. A temperature below the normal may occur independently of fever. It may follow as a sequelæ of the diseases with more or less prolonged pyrexia. It occurs in the course of wasting diseases, as in cancer, in starvation, at times in anæmia. It is seen habitually in myxœdema, and occasionally in diabetes. In certain forms of tuberculosis it may extend over a long period of time, as in tuberculous peritonitis. (See chart under Tuberculous Peritonitis.) In cases of cerebral abscess the temperature is often subnormal.

Sometimes the drop to subnormal temperature may occur suddenly, to be followed by a return to normal or even a rise above normal. The sudden fall may occur in shock, or in hemorrhage from any cause. It may take place from disturbance of the nerve-centres, as from apoplexy, thrombosis, or embolism of the brain, causing shock or other disturbance of the thermotaxic mechanism. It is characteristic of cholera. In the course of organic heart disease pulmonary embolism is also attended by subnormal temperature. In many of these in-

stances the temperature will rise (reaction) after the shock if the latter is not too profound. This is notably the case in apoplexy and in embolus or thrombus, because of local irritation or a secondary softening. In apoplexy the rise in temperature will occur either from central disturbance of the thermic mechanism or from secondary inflammation about the clot. A subnormal temperature in the course of

FIG. 33.



Subnormal temperature. Oscillations in hepatic intermittent fever with jaundice. Catarrh of ducts, with diffused hepatitis. G. W., aged 60. Philadelphia Hospital, 1877.

fever may be due to an accident or complication, as hemorrhage in disease of the lungs, or in typhoid fever, or perforation of the intestine in the latter condition. It may attend the crisis of acute disease. More or less collapse usually attends the pathological fall of temperature below the normal. While such fall is the result of accident in many of the diseases mentioned, in others it is a part of the process.

The chart (Fig. 33) represents the effect of a local process in the largest gland of the body upon the general temperature. It is possibly a septic temperature, although the observation was made before the days of bacteriological research. The extreme low temperature is remarkable.

The Diagnostic Significance of Fever. Its Clinical Causes. The presence of fever is itself of diagnostic importance.

A. It usually excludes *hysteria* and *malingering* disease.

B. It indicates that one of several morbid processes is present. The morbid processes which give rise to fever are:

First, an *infection*, general or local, as seen in any one of the infectious diseases and in the local inflammations induced by micro-organisms, especially those known as pus-producing. When local, the inflammation is known as purulent, suppurative, or septic. The micro-organism, a product of its growth, or the poisons or ferments resulting from the tissue change, disturbs the thermotaxic mechanism and causes fever. Any tissue, membrane, or organ of the body may be the seat of an infectious process.

Second, an *intoxication*, or *toxæmia*, as caused by albumoses, ferments, toxins, or ptomaïnes, generated within the system, the result of impaired functional activity of organs or structures, or of cell metabolism, as seen in tissue waste; and by food products, medicines, or toxic substances introduced from without. Catarrhal inflammations cause a toxic fever. The fever of gout, of anæmia, of starvation is toxic.

Third, fever may be of *central origin*, from disease of the brain involving the centres controlling heat, or from disease in proximity to the heat-centres. It may arise in cases of brain-tumor, in cases of apoplexy, and of thrombosis. The centres may also be irritated by direct exposure to external heat alone, or possibly by poisons generated within the system on account of the heat (an intoxication), as in sunstroke.

Fourth, a pronounced *peripheral irritation* or sensation of *pain*, reflexly altering the thermotaxic mechanism, will produce fever. Hence, in iritis or orchitis a fever arises out of all proportion to the local inflammation.

Finally, cases of *continued fever* exist that have not thus far been classified. One of the nurses of the Presbyterian Hospital with a continued temperature from 100° to 103° was under my care for two months. No general or local condition could account for it. The patient was emaciated. She had had two years of very hard work. Although fever kept up, the appetite was good. Careful and abundant feeding, with rest for many weeks, caused the temperature to fall to normal, with complete recovery. I looked upon it as a nervous fever; an expression of exhaustion. Fagge refers to such cases.

Practically, we must in all cases of fever decide between one of *infectious* and one of *toxic* origin. Discussion of the mode of determining the occurrence of an *infection* will be considered shortly. In the meantime we may observe that the poisons which are generated in the gastro-intestinal tract are likely to disturb the cardiac and respiratory as well as the thermotaxic mechanism. Hence we often see *irregularity* and *intermittency* of the heart—so often as to look upon it as of diagnostic value in favor of toxic fever.

Certain clinical features of a febrile course belong, in the main, to special affections, and thus far are diagnostic of them. Hence the mode of onset or *initial stage*, the course or *fastigium*, the decline and the type should be carefully studied. They are a most important indication of the nature of the disease.

The Initial Stage. 1. In the initial stage of fever *sudden*, excessive rise of temperature from a condition of apparent health argues against any of the acute specific fevers except scarlet fever. It is of more frequent occurrence in acute gastric or gastro-intestinal catarrh in children than in any other ailment. It may be due to pneumonia, and is significant of this infection in adults if attended by a rigor. In children convulsions may replace the chill. The sudden rise may be due to certain types of malaria, when it is also preceded by a chill and followed by free sweating. It may also be due to affections of the throat, to follicular or phlegmonous inflammation of the tonsils. The throat must always be examined in cases of sudden high temperature.

In children, if pain attends any inflammatory affection, the temperature will rise to a greater height than the local process alone would warrant. This is the case with suppurative inflammation of the middle ear. This must always be borne in mind in sudden rise of temperature. The same active febrile reaction will take place in osteomyelitis and in mastoid abscess. The associate signs point to the true nature of the affection, although it must be confessed that in both the symptoms are often obscure in the beginning.

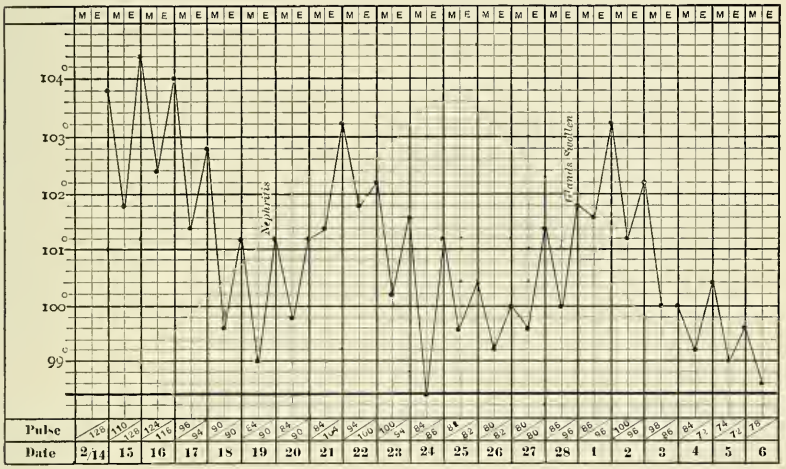
2. In typhoid fever the temperature rises in a characteristic way. It ascends by successive evening rises, followed by morning remissions, until it reaches the maximum at about the end of the first week.

The Fastigium. In typhoid fever the course of the fastigium is of characteristic significance. From the end of the first, throughout the second week, and sometimes longer, the fever is of the continued type. Subsequently during the third week, or later, morning remissions set in, the temperature for a time still rising to the former height in the evening. Then the morning remissions become more decided, the temperature not rising as high in the evening, and so gradually the temperature sinks to and below normal. This course of the temperature in typhoid fever is very far from being invariable ; it is modified by indiscretions on the part of the patient or his attendants, and by the necessities of antipyretic or other treatment ; nevertheless, the gradual onset of the fever and its long duration are sufficiently common to make them of great value in diagnosis, as, with the exception of tuberculosis, there is hardly any other disease in which a continued fever exists for two or three weeks apart from local inflammation or suppuration.

The Decline. *Defervescence.* In the self-limited diseases there is a period when defervescence should take place. A continuance of the fever, the persistence of the fastigium beyond the usual period, indicates that the case is one of a greater degree of gravity than usual, or that there is a complication. It is usually significant of a complication. In measles the complication is usually pneumonia. This may take place after the disease has developed, and may be the cause of the unusual rise in temperature. In scarlatina it may indicate acute nephritis, or inflammation of any of the serous membranes, particularly the pericardium or endocardium. Persistence of the fastigium of typhoid fever after the period at which it should decline,

if the patient is well nursed and properly fed, usually indicates the occurrence of a reinfection, a secondary infection, or the development of tuberculosis. If the latter, the fever is more likely to develop during convalescence. Of the inflammatory complications, phlebitis and glandular and bone infections are likely to cause persistence of fever.

FIG. 34.



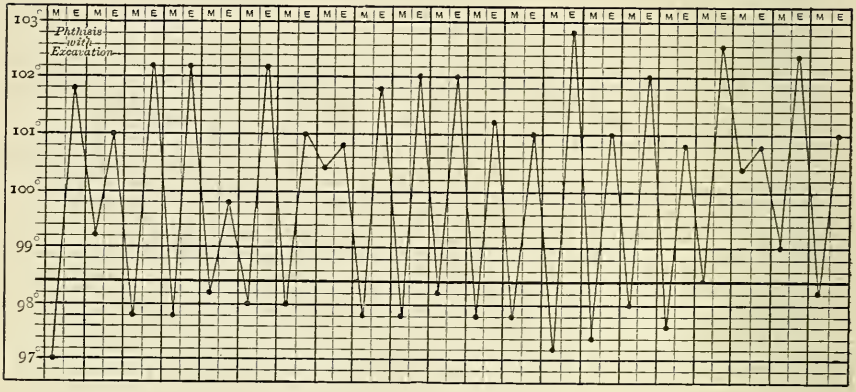
Scarlet fever. Modification of temperature by complications. Nephritis on the ninth day.

A Sudden Fall; Subnormal Temperature. A sudden fall of temperature in a person who has previously had high fever signifies the crisis if the time for that event has arrived, as in pneumonia; or of a grave complication, which induces shock. In typhoid fever this unusual drop in the temperature will take place if there has been hemorrhage from the bowels, or perforation, or if peritonitis has developed. It must not be confounded with the sudden falls of temperature that occur in the typhoid fever of children, corresponding to the onset of convalescence. They occur earlier in the period of the disease than with adults.

THE TYPE OF THE FEVER. *Intermittent Fever.* The representative of the type is seen in *malaria*, but it is simulated by a number of conditions: (1) In certain cases of *typhoid* fever and of *relapsing* fever the type is intermitting or paroxysmal. The same type of fever is seen (2) in *suppuration*, particularly if the pus is confined, although in brain abscess the temperature may be normal or subnormal; (3) in *infectious endocarditis;* (4) in *tuberculosis*. *a*. It may occur in the earlier stages of tuberculosis. The primary seat of the lesion may be in the lungs, in the bones, or in the glands. *b*. In pulmonary tuberculosis, after the formation of a cavity, intermitting fever is of common occurrence. It is then of septic origin due to the septic influence of the necrosed tissue and products of putrefaction in the cavity. (See Fig. 35.) (5) In *lymphadenoma* and *anæmia* the fever is at times paroxysmal.

(6) In *syphilis* the same type is often seen. It may be noted (*a*) in the initial fever; (*b*) in the tertiary periods of the disease where gummata have formed, or other forms of visceral syphilis have developed. (7)

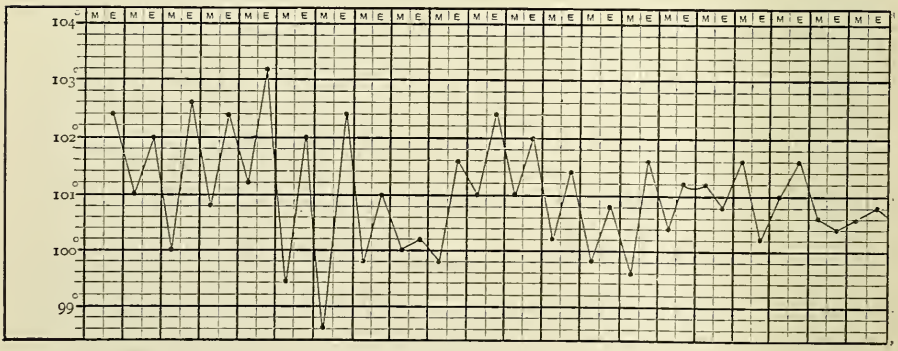
FIG. 35.



Intermitting fever of tuberculosis.

Urinary intermitting fever is the form which usually occurs after the passage of a catheter or sound, but it may also occur when there is suppuration in the genito-urinary tract. (8) *Hepatic intermitting* fever is a form of frequent occurrence and of great diagnostic importance.

FIG. 36.

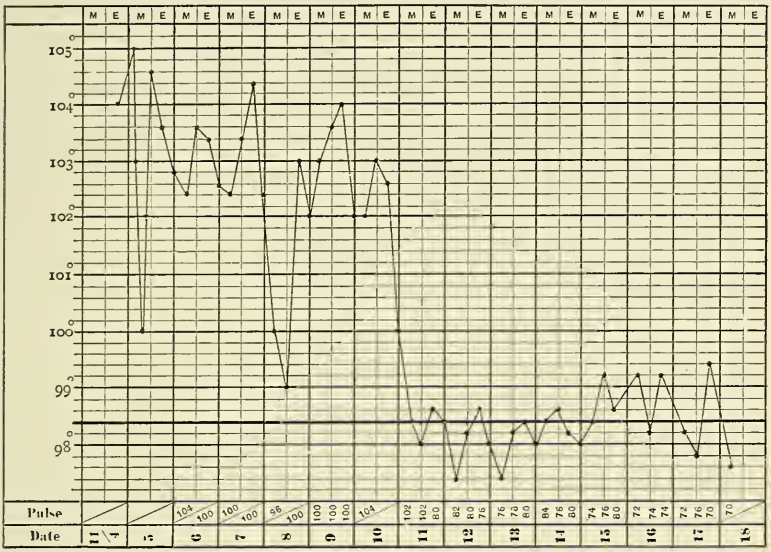


Continued fever of tuberculosis.

It may be due to (*a*) gallstones somewhere in the biliary ducts, usually with obstruction; (*b*) suppuration in the canal, with or without obstruction; (*c*) obstruction of the biliary passages by external pressure without suppuration; (*d*) inflammatory affections of the liver, as abscess, and forms of cirrhosis. It occurs rarely in rapidly growing cancer. (See Fig. 33.) (9) Intermittent fever may also attend the prolonged use of *morphine*.

Of the above-mentioned varieties of paroxysmal or intermitting fever, those of the most common occurrence are due to suppuration, pyæmia, to infectious endocarditis, to tuberculosis, and to hepatic disorder. In addition to the paroxysmal temperature, rigors precede and sweating follows the paroxysm, as in cases of malarial intermittent fever.

Fig. 37.



Pneumonia. Sudden rise. Termination by crisis. Pseudocrisis also seen.

The diagnosis from malarial intermittent fever can be established at once by an examination of the blood, which reveals in the latter the plasmodia of Laveran.

Remittent Fever. Fever of a remittent type occurs in many of the conditions in which intermittent fever is present. It is characteristic of one of the forms of malaria. It is most frequently encountered in tuberculosis of the lungs. The remissions usually occur in the mornings, but the order may be reversed. The same type of fever is met with in puerperal fever, pyæmia, and septicæmia, and in local suppurations, such as abscess of the liver and empyema. A continued fever may be made to resemble a remittent by antipyretic treatment, which may cause abnormal remissions. Remissions characterize the decline of the continued fevers, particularly typhoid, during the period of lysis.

Continued Fever. Continued fever is met with in lobar pneumonia, typhoid fever, typhus fever, erysipelas, and tuberculosis. In acute lobar pneumonia the temperature rises rapidly, and in a few hours from the initial chill reaches 103° or 105°. The morning and evening temperatures vary but little, usually not more than one or two degrees, until a crisis occurs in from four to eight days. The temperature then falls to or slightly below normal, and does not rise again. (See Fig. 37.)

A marked remission in the fever sometimes occurs on the fourth day, before the actual crisis; the temperature falls to 100°, and rises again to 103° or 104°, remaining at that level for twenty-four or forty-eight hours, when the true *crisis* occurs. The first fall is known as the *pseudocrisis*. The fall of temperature of defervescence (crisis) may be completed within a few hours.

The Influence of Age and Sex. The significance of a high febrile change is not so great in children as in adults. That is, the high temperature is not so important, inasmuch as children are liable to have sudden, excessive increase of temperature; and a higher temperature may persist in children without deleterious effects upon the tissues which are noticed in adults. In women of nervous temperament the temperature is also likely to rise to a great height without adequate cause or serious result.

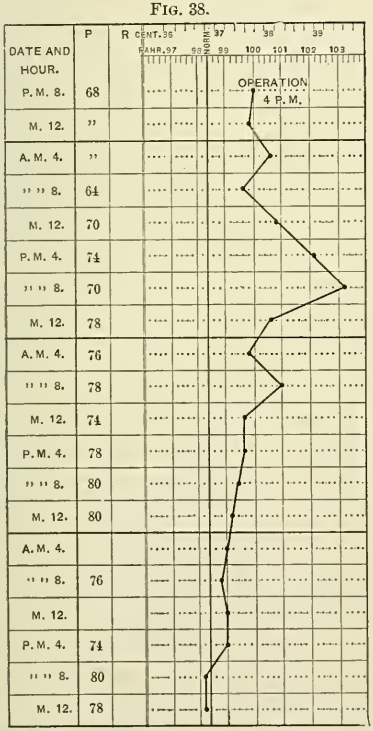
CHAPTER XV.

THE DATA OBTAINED BY OBSERVATION—(*Continued*).

FEVER. THE INTOXICATIONS.

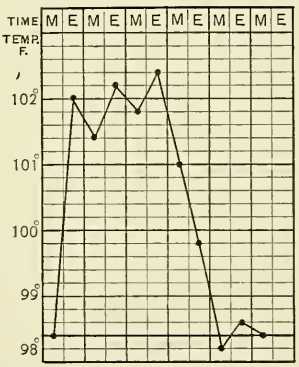
PRACTICALLY, it may be said that the symptom *fever* may be due to an intoxication, an infection, or a central cerebral lesion. In this chapter a word may be said of the fever of an intoxication. The substance which produces fever of this type may be a toxic material, the product of local or general disturbance of tissue metabolism. Thus in a local catarrhal inflammation, as of the bronchi, the result of the direct action of an irritant vapor, toxic substances are generated which

FIG. 38.



Temperature curve after amputation of the forearm.

FIG. 39.



Aseptic or fermentation fever.

disturb the heat mechanism and produce fever. Now, an intoxication or simple inflammation, therefore, is attended by fever, which may be styled *catarrhal fever*. (See Fig. 41.) In anæmia, on the other hand, if all infections can be excluded, it may be said the general disturbance of tissue metabolism possibly gives rise to the formation of a toxin which causes the fever well known to attend this process—*anæmic fever*.

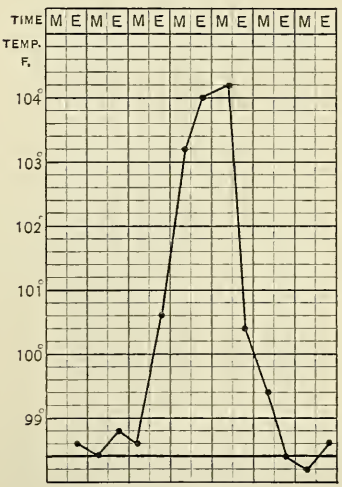
A better example of fever due to a poison is that which Collins Warren terms *aseptic fever*. It is also known as *absorption* or *fermen-*

tation fever. The fever follows a perfectly aseptic operation, and no causal factor is present. It is due to the absorption of ferments, from blood clot, or coagulated serum, or tissue débris. The temperature rises to 102°, and may remain above normal from three days to two weeks. (See Figs. 38 and 39.) There is a striking absence of constitutional symptoms, however. Another peculiarity is that the fever begins immediately after the operation. The urine is not lessened, the body-weight remains normal, and the pulse-rate corresponds to the temperature rise. In some instances an eruption like that of scarlet fever—*surgical scarlet fever*—breaks out.

Should it happen that the retained fluids undergo decomposition and are absorbed, a more intense type of fever is seen, attended by marked constitutional symptoms. We then have *traumatic fever*—a fever which subsides as soon as the poison is liberated from the wound. In the meantime the temperature has been as high as 102° to 103°—the pulse very rapid, delirium has been marked, and there has been furred tongue, thirst, anorexia, restlessness, and malaise.

It may happen that septic infection of a wound takes place. Thus, one of my patients, while dressing a suppurating vaccine wound, inoculated or infected her finger. The tender spot was followed by redness along the lymphatics, and enlargement of the glands—a lymphangitis. She had fever. A deep cut in the infected spot released a serous discharge, the fever disappeared, and the lymphatic inflammation subsided at once. Such accidents happen frequently to surgeons. Another patient was infected by a surgeon who had just operated on an osteomyelitis. The temperature rose to 106.5° in twenty-four hours, and the constitutional symptoms were extreme. The wound in the abdominal walls was opened and cleansed, and the peritoneum was not reopened; no peritonitis resulted. The temperature fell four degrees at once. The muscles and other tissues of the wound became gravish and almost putrid. Recovery was slow. Such cases are known as septic cases, the ailment *septicæmia*, and the intoxication *sapræmia*. (See Fig. 40.) No bacterial invasion of the body takes place, and there is no local suppuration. No doubt, in each instance micro-organisms infected the wound, but the symptoms arose from the chemical product resulting from their growth.

FIG. 40.



Septic intoxication.

In obstetric practice the retained putrefying placental fragment will cause such symptoms. In medicine we see such intoxication take place in infections. Thus in diphtheria, systemic intoxication with fever results from the absorption of a toxin from the local point of bacterial growth. In tetanus the same toxic fever and symptoms occur. It is

impossible to draw hard-and-fast lines between the toxic fever and the infective, pyogenic or suppurative fever, and, indeed, such cases properly belong and will be considered under the next prominent causes of fever to be considered—the infections.

But "fever" may be due to other intoxications. It is well known that pepsin and other digestive ferments injected into the body cause fever. It is supposed products of imperfect assimilation or digestion absorbed into the system from the gastro-intestinal tract give rise to fever. Ptomaïnes or leucomaines, albumoses or peptones, absorbed from the intestinal tract may thus cause fever. The retention of excretory products, as those of the renal organs, cause a systemic intoxication, with the frequent occurrence of fever. Gout, too, may be considered as an intoxication giving rise to fever.

The fever of auto-intoxication (gastro-intestinal or glandular), so called, therefore, is an entity. The clinician, at least, without proof by the bacteriologist, sweeps the intestinal tract with his mercurials and salines, and thereby administers the causal antipyretic.

Poisoning by food products, as of cheese, meats, sausages, milk, etc., appear to cause fever, although it is possible intestinal bacteria may play some part in the process.

Varieties of Febrile Intoxications. It is assumed that the student is investigating a case of fever. In keeping in mind an intoxication as a cause of fever, he must first consider all causes of intoxication from within; second, all causes from without the organism.

To the first belong gout, uræmia, cholesteræmia, and the auto-intoxications from the intestinal tract, as well as those from modification or suppression of internal secretions, as of the thyroid and other glands.

To the second belong the following: *Sunstroke, morphinism,* and *food-poisoning.* The fever due to an intoxication, as in the so-called *febricula* and in the *simple continued* or *catarrhal ferer,* is of doubtful origin.

Diagnosis. *The Action of the Heart.* Increased frequency of cardiac action is a symptom common to all forms of fever. It is more common to see irregularity and intermittency in the fever of intoxication, and especially of auto-intoxication, than in that of infections. Indeed, I should call a fever which is attended by a cardiac neurosis, cardiac mural disease and cerebral disease excluded, one of intoxication.

Increased Respiration. The same may be said of the breathing. When a respiratory neurosis prevails in the course of fever, it and the fever attending are due to a common cause, an intoxication. Of course, pulmonary and central brain and medulla disease are excluded. It seems both the above observations aid in the diagnosis of an intoxication from an infection.

Febrile Intoxications.

Sunstroke (siriasis, thermic fever, insolation, heatstroke). Whether the cause is the direct action of heat upon the brain centres, or whether a toxic substance is generated and becomes operative, in this affection we have the most pronounced expression of *fever* outside of the infectious disorders. The flushed face, the pungent skin, the dyspnœa,

and the rapid pulse forebode the high body temperature which in the axilla may reach 108° to 112°. This is reached very rapidly, and death takes place in coma hyperpyrexia. If recovery takes place, the temperature may be moderately continuous a few days. The picture is added to by the nervous and cardio-respiratory phenomena. In some instances dyspnœa, heart-failure, and coma may follow on rapidly, and death ensue in one or two hours. In other cases pain in the head, dizziness, and languor precede the stupor. Nausea and vomiting, perhaps diarrhœa, chest oppression, frequent micturition, and convulsions may precede the insensibility. Unconsciousness is lost quickly or gradually, and it may be transient or pass into deep coma. Relaxation of the muscles with twitching is seen, and the pupils, at first dilated, become contracted. As the coma deepens, the heart's action becomes more rapid and feeble, the respirations hurried, shallow, and irregular, and death ensues, preceded or not by convulsions.

The diagnosis is based on the history, the mode of onset, and the hyperpyrexia. It must be distinguished from uræmia and apoplexy.

Heat exhaustion is readily recognized. The moist, pale, and cool skin, the soft, feeble pulse, the quiet but hurried breathing, are unattended by fever. The collapse, for such it is, is not attended by coma, and it usually responds to treatment.

Morphinism. Lewin showed that morphinism is attended by fever. The fever may be continued or intermittent. When the latter, *chills* are of frequent occurrence. The diagnosis is based on the history, on the evidence of poor nutrition without cause, on the general depression and lassitude, and upon the temperament of the patient, to which is added poor sleep, restlessness, and itching of the skin. The peculiar sallowness of the complexion and the prematurely aged appearance are well known. Pseudo-neuralgic pains are common, tabetic symptoms may be present, and notably gastro-intestinal symptoms, as gastralgia, vomiting, diarrhœa, especially if the drug is withheld. Fever, it must be remembered, may be absent.

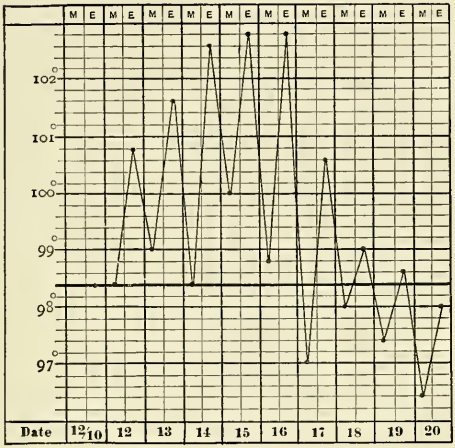
Simple Continued Fever. A non-contagious fever, lasting from one to twelve days, not dependent upon any known specific cause, and not attended with any definite local lesions. Its chief characteristic is the continued elevation of temperature.

It occurs especially in children and in those prone to ready disturbance of the heat-regulating apparatus. Great mental and physical exhaustion, prolonged bathing in the hot sun, and disturbances in digestion may cause it. Perhaps, as suggested by Guitéras, some of the cases occurring in the tropics and in very hot weather should be regarded as very mild forms of thermic fever.

The onset of the disease is abrupt. There may be a chill, or in nervous children a convulsion ; but these are rare. The temperature rises rapidly from 102° to 104°, accompanied by headache, thirst, restlessness or drowsiness, loss of appetite, a coated tongue, constipation, and occasionally nausea. The urine is scanty, and sometimes there is a heavy deposit of urates. There may also be more or less muscular soreness. Sometimes within twenty-four or forty-eight hours free perspiration takes place, with rapid subsidence of the fever. This is *ephemeral fever.*

In other cases the fever continues for a week or ten days longer. During this time the symptoms already noted continue. Sleep is disturbed and mild delirium is at times present. Respiration and pulse are not much accelerated. Sudamina upon the abdomen and herpes on the lips are common. Pale-bluish maculæ are sometimes seen.

FIG. 41.



Simple continued fever.

The spleen is not enlarged except in very rare cases, and there are no local evidences of disease. The fever subsides more gradually than in ephemeral fever, the defervescence being marked at times by perspiration, a few loose stools, a copious deposit of urates in the urine, or by hemorrhages from the nose, rectum, uterus, or urethra.

The *diagnosis* from other fevers and febrile affections is made by the absence of any characteristic eruption, of enlargement of the spleen and liver, and of any lesion, such as endocarditis, bronchitis, or pneumonia.

Food Intoxications. Among the intoxications which give rise to fever are those due to food-poisoning. Meat, milk products, and shellfish cause an intoxication of the system which in the instance of the first three forms often threatens life, and, from the suddenness of the attack and the severity of the symptoms, points to an infection rather than an intoxication.

The history of the case is often the first clue to its nature. The symptoms are those of acute gastro-intestinal irritation, to which are added, with or without afebrile periods, the symptoms of collapse.

Meat-poisoning. In the intoxication arising from poisoning by meat, the temperature rises from 101° to 104°, preceded usually by a brief period of chilliness. The occurrence of fever may be preceded by a period of incubation lasting from twelve to forty-eight hours. During the period of *incubation* there is malaise, loss of appetite, nausea, and colicky pains. As they increase chilliness ensues, and in some instances there is a marked rigor. Prostration occurs almost

immediately, with giddiness and faintness, and the occurrence of cold perspiration. Headache and backache are liable to occur. Following the chilliness the symptoms of gastro-intestinal irritation arise, diarrhœa being more frequent than vomiting. The abdominal pain increases and the perspiration and clammy sweats become more pronounced. As further evidence of the intoxication, there is an extreme degree of muscular weakness. The pulse becomes rapid, and later, thready. In addition to muscular weakness, cramps in the legs and arms, followed by convulsive movements, occur, and the patient complains of paræsthesia of various forms. In milder cases the symptoms of gastro-intestinal irritation and of muscular weakness attend the fever. In the more severe cases fever is replaced by collapse.

Poisoning by Milk Products. Symptoms of gastro-intestinal irritation and choleraic symptoms ensue. The diarrhœa of infants and cholera infantum are types of this intoxication. The high degree of fever that occurs is well known. In cheese-poisoning the fever is not continuous as in the other forms, the temperature becoming subnormal with the onset of collapse.

Poisoning by Shell-fish. In mussel-poisoning the symptoms are those of an acute mineral poisoning with profound nervous symptoms. Fever does not attend this condition, but collapse follows quickly. There are no gastro-intestinal symptoms.

Fish-poisoning is also unattended by fever, collapse occurring early.

Afebrile Intoxications.

For convenience, and by contrast, the afebrile intoxications will be considered. Herein will not be considered those important *afebrile intoxications* due to disease of the *ductless glands*. They include some diseases of the suprarenal bodies (Addison's disease), the thyroid gland (exophthalmic goitre and myxœdema), the lymphatic glands (status lymphaticus), and the spleen.

Alcoholism. In *acute alcoholism* the reeling gait, the incoherent speech, followed by narcosis, are well known. The temperature is afebrile. Often, indeed, it is subnormal, and when equal on both sides of the body is very suggestive. The flushed face, possibly slightly dusky, and the injected eye, would lead us to suspect the presence of fever. The odor of the breath furnishes a clue. The heavy breathing, the full pulse, the dilated pupils, the stuporous rather than comatose state, are accompaniments of this intoxication. The flaccid limb of one side would point to hemiplegia from hemorrhage, especially if the coma is deeper than usual and the stupor more marked. But *uræmia* and *apoplexy*, and either of the two in a drunken subject, must be borne in mind.

Chronic Alcoholism. When the poison is taken for a long time it acts as a tissue poison and a check upon waste. Epithelial and nerve degeneration and fibrous overgrowth follow the first or poisonous irritative action; and fatty change the second. In the alcoholic, tremor of the hands and tongue is seen. The action of the muscles is unsteady. The mind is dull, the temper irritable, forgetfulness is most

common, and later a dementia and epilepsy may ensue. Alcoholic neuritis, to be described later, is of frequent occurrence.

Gastro-intestinal catarrh with poor appetite and constipation is most liable to ensue, and later cirrhosis of the liver and kidneys. Endarteritis and cardiac dilatation develop in some independently ; in others with the nervous affections, delirium tremens.

Grain-poisoning. Three forms are seen. When the grain is contaminated by ergot, symptoms known as *ergotism* occur. Chronic ergotism may cause gangrene or a train of nervous symptoms in which convulsive movements are most prominent. In the gangrenous form the toes and fingers are the seat of mortification. The process is preceded by anæsthesia, paræsthesia, and pain. In the convulsive form there is slight fever with some weakness and tingling sensations in the body. Cramps and contractures occur in the extremities, continuing for hours or days, and relapsing frequently. A mild delirium or the development of melancholia or dementia attends the convulsive form.

In other intoxications fever is not so pronounced. In *lathyrism* the symptoms are those of spastic paralysis, which may proceed to paraplegia. In *pellagra*, a disturbance due to maize, there are disorders of digestion, loss of sleep, general pain, and debility. The digestive symptoms are those of salivation, dyspepsia, and diarrhœa. A peculiar erythema arises. Subsequently, desiccation and desquamation of the epidermis occur, and often small boils develop. Headache, backache, spasms, and paralysis of the legs occur in the severe and chronic forms. The nervous symptoms may give way to melancholia.

Lead-poisoning. Intoxication due to lead or plumbism may be acute or chronic. In the *acute* form we have symptoms of gastro-intestinal irritation with constipation and extreme colicky pains. Anæmia may develop rapidly, and pronounced nervous symptoms arise. Among the latter we have neuritis, convulsions, epilepsy, and delirium. Hemorrhages from mucous membranes may be seen, and a form of nephritis develops rapidly. The urine contains albumin and tube-casts. Fever is not a pronounced symptom.

The characteristic symptoms of *chronic* poisoning are (*a*) saturnine *cachexia*, in which anæmia is most pronounced ; (*b*) *colic ;* (*c*) *paralysis*, which may be acute, subacute, or chronic, and which usually develops without fever. The paralysis may be anti-brachial, causing characteristic wrist-drop ; brachial, in which the scapulo-humoral form of paralysis is seen, and an Aran-Duchenne class, resembling chronic anterior poliomyelitis. Another is the peroneal type, in which the lateral peroneal muscles, the extensor communis of the toes and the extensor proprius of the big toe are paralyzed, causing the steppage gait. Finally, paralysis of the adductor muscles of the larynx occurs in lead-poisoning. The paralysis often extends from a local group of muscles throughout the body, presenting symptoms like those of an ascending paralysis with rapid wasting. In other instances the general paralysis occurs primarily, the wasting and loss of power going hand in hand. Fever sometimes attends a general paralysis in lead-poisoning. (*d*) The *cerebral* symptoms of the acute form have been mentioned. In the chronic cases they may also occur. *Optic neuritis*, or neuro-retinitis, is

common. *Delirium*, with hallucination, may occur. *Tremor* is a common symptom. It must not be forgotten that headache, convulsions, epilepsy, and delirium may be manifestations of *lead encephalopathy*, even in cases in which the history of exposure to lead is not direct; (*e*) chronic lead-poison leads to *arterial sclerosis* and *contracted kidneys* with hypertrophy of the heart; (*f*) *gout* is very common, and may be seen in both acute and chronic forms, particularly in the big toe; (*g*) as described in the section in which the mouth and gums are discussed, the *blue line* is the specific symptom of lead-poisoning. The reader is referred to that chapter for a description of the line. It must be remembered that in all forms of obscure nervous disease, in gastro-intestinal irritation, in arterio-sclerosis, and gouty arthritis, this line must be looked for.

Arsenic-poisoning. *Acute* arsenical poisoning is attended by severe symptoms of gastro-intestinal irritation followed by the rapid development of collapse. Fever is not a prominent symptom unless recovery is about to take place. The temperature is subnormal, but as the collapse symptoms disappear fever due to gastro-intestinal ulceration develops.

In *chronic* arsenical poisoning the fever occurs only if there is great irritation of the mucous membranes, as of the conjunctiva, mouth, or pharnyx. In this form, in addition to the irritation of these mucous membranes, there may be subacute gastro-intestinal catarrh, with diarrhœa. In other instances there is profound anæmia and debility, with paræsthesia and neuralgia. In others, again, paralysis like that of lead palsy may occur. It must not be forgotten that puffiness under the eyelids may be due to this cause.

CHAPTER XVI.

THE DATA OBTAINED BY OBSERVATION—(*Continued*).

Causal relation of bacteria to disease, Koch's laws, value in diagnosis. *Bacteria:* Saprophytes, parasites, pathogenic, non-pathogenic, aërobic, anaërobic, facultative anaërobic. Morphology: Micrococci, bacilli, spirilla—*Micrococci.* Morphology: Form and size. Reproduction, fission; grouping. Biological characters: Non-motile. Pigment production. Liquefaction of gelatin. Production of acids. Toxic ptomaïnes and toxalbumins—*Bacilli.* Morphology: Form and size. Reproduction, fission, spores; grouping. Biological characters: Motility. Pigment production. Liquefaction of gelatin. Production of acids. Putrefaction, fermentation. *Spirilla.* Morphology: Form and size. Reproduction, fission; grouping. Biological characters. Motility. Pigment-production. Liquefaction of gelatin. Production of acids and fermentation wanting.

FEVER. THE INFECTIONS.

We have already indicated the diagnostic significance of the type of the fever (Chapter XIV.). Following the lead in part of the subjective symptoms, we next examine every organ and structure of the body when the symptom—*fever*—is present. By this examination we will find either (1) a functional disturbance of some organ of the body; (2) an inflammation; (3) or we will find a general process, or *infection*, any local inflammation being secondary, brain disease and intoxications having been excluded.

1. Any *functional disturbance* of one or more organs—glandular—attended by fever must be looked upon as an *intoxication*. Fevers due to such causes have been discussed in the preceding chapter, so we pass on to inflammations, toxic and infectious, which cause fever.

2. Suppose we find local *inflammation* of some part, as an inflammation of the nares, a bronchitis, or an apparent gastritis or enteritis. The inflammation may be toxic or it may be infectious. As another example, let us take the kidneys. Blood, albumin, and renal casts would show that they are the seat of inflammation. This inflammation may be toxic, as from cantharides, or the toxin of an infection, or it may be infectious. In either instance the fever is caused by the local process. To determine whether the inflammation is *toxic* (generally catarrhal) or *infectious*, we must rely upon the data obtained by inquiry, the clinical course, and the result of the examination described in Chapter XVII., which discloses the method of determining the presence of an infection.

3. If the above are excluded we proceed with the bacteriological diagnosis. By this means we find if a general infection prevails. Such diagnosis may be necessary also to recognize pyæmia and septicæmia.

The Infections.

It had long been surmised that micro-organisms had much to do with morbid processes, and that the relationship was that of cause and effect. It was known, for instance, that suppuration, surgical fever, erysipelas, hospital gangrene, and puerperal fever were associated with conditions which favored the multiplication of the lower forms of life. What relationship the micro-organisms bore to the various affections was not known. Least of all were the specific micro-organisms which were the causes of particular specific morbid processes known. I have said that it was surmised; but there was groping about, a difference of opinion, and a maximum of theory, a minimum of fact. It is true that in relapsing fever the spirillum had been found, and that none had been found in any other disease. Moreover, it is true that monkeys had been inoculated and the disease reproduced in them. It is true that the bacillus of anthrax had been seen in the blood in the early sixties. It is true that the great genius Pasteur had prosecuted studies of bacteria in animal and vegetable pathology to most brilliant and practical conclusions. Nevertheless, there were confusion and doubt; scientists were not satisfied with the demonstrations which undertook to prove the causal relationship of micro-organisms to disease.

Laws to Establish Causal Relationship. By the genius of Robert Koch theories and objections were set at naught. The scientific world was fully prepared by the labors of early investigators to accept Koch's conclusions. They were based upon an array of well-authenticated facts, which anyone could prove for himself. The postulates formulated by Koch, the fulfilment of which he considered as necessary in order to identify an organism as the etiological factor in a given disease, are as follows: The constant presence of the organism in the affected tissue of the diseased animal; its isolation from the pathological lesions, and its continuous cultivation in pure cultures under artificial conditions through many generations; the power of such pure cultures to reproduce the disease when inoculated into susceptible animals; and the detection of the organism in pure culture in the lesion found in the animal thus inoculated. The experimental circle was then repeated. In this manner the causal relationship of micro-organisms to special diseases had been proved by the distinguished investigator in the case of anthrax, tuberculosis, and other affections. In a certain number of cases particular species of bacteria and other micro-organisms have been isolated from definite diseases and reasonably believed to stand in causal relation to them, but which have, nevertheless, not fulfilled all the requirements of the above-cited postulates. The difficulties often encountered are: The impossibility of reproduction in animals of the clinical and pathological features that the diseases present in human beings, as is the case with typhoid fever, influenza, gonorrhœa, and fibrinous or lobar pneumonia; and the impossibility of satisfactorily cultivating certain other organisms that are the constant accompaniment of particular diseases of man, as, for instance, the plasmodium malariæ, the bacillus of syphilis, and the amœba coli.

The infectious diseases, then, are those that are produced by a living contagion or micro-organism. The organism is introduced into the body through the skin, if the latter is the seat of some lesion, as in syphilis, tuberculosis, and anthrax; through the air-passages, as in diphtheria, scarlet fever, and other specific fevers; or through the digestive tract, as in typhoid fever, dysentery, and cholera. The virus, as the living cause is named, in many instances produces certain changes at the point of entrance—the *initial phenomena*. It is then conveyed by the lymphatics or bloodvessels to near-by organs in the related lymph-stream or blood-stream, or transmitted to the whole body. When the whole body is affected an eruption is sometimes produced (eruptive fever), or the blood is changed in quality (diphtheria), or many tissues are affected simultaneously, or the nervous system is notably disturbed. The above are the *phenomena of general distribution* of the virus, or of *infectiveness*. The virus or poison thus distributed may be the living organism, as in tuberculosis or anthrax, or it may be a poison generated by the organism, a toxin or ptomaïne, as in diphtheria.

Phenomena of secondary local distribution are due to local changes in organs affected secondarily. The poison has a special affinity for certain organs, as in whooping-cough, parotitis, pneumonia, or leprosy.

In some instances the local phenomena are so marked as to give to the disease a corresponding distinctive feature. They are the granulomata. Bearing in mind the above distinctions, specific infectious diseases are divided into six classes.

First Class. *Acute Specific Fevers.* The initial phenomena are slight. The phenomena of infectiveness are marked; an eruption is one of the most characteristic. The secondary local phenomena are variable. The following are included in this class: Typhoid fever, typhus fever, variola, varicella, scarlet fever, measles, relapsing fever, rubella, influenza, dengue, the plague, and cholera.

Second Class. *Specific Inflammation.* Initial phenomena indefinite. General phenomena (infectiveness) variable, but no eruption. Specific affinity of poison for one particular structure. Whooping-cough, mumps, diphtheria, dysentery, erysipelas, tetanus, hydrophobia, cerebro-spinal meningitis, rheumatic fever, and pneumonia belong to this class.

Third Class. *Contagious or Infectious Suppuration.* Initial phenomena marked (suppuration); generalization not marked unless the virus enters the blood; secondary local phenomena decisive. Gonorrhœa is one type, pyæmia, or any infection from pus-producing micro-organisms, as abscess, carbuncle, etc., a second, in which the blood is infected.

Fourth Class. *Infective Granulomata.* Distinct initial phenomena. Phenomena of generalization not marked, or like specific fevers. Secondary local phenomena prominent. Examples: Tuberculosis, syphilis, leprosy, and glanders.

Fifth Class. *Miasmatic Diseases.* No initial phenomena.

Sixth Class. *Vegetable Parasitic Diseases.*

It is readily seen that when the definite cause of an infectious disease is isolated, and the morphological and biological properties of the

causal micro-organism determined, the clinician has acquired a valuable aid to diagnosis. Indeed, in such affections the bacteriological diagnosis has become an absolute certainty.

Bacteria.

To determine the micro-organism which causes the infection the student must be familiar with the morphology and biological properties of the various forms of bacteria. (By means of this knowledge a bacteriological diagnosis is made.) *The morphology:* The shape, the size, the mode of reproduction and grouping are to be studied. Bacteria or fungi are divided morphologically into *micrococci* or spherical bacteria, *bacilli* or rod-shaped bacteria, and *spirilla* or twisted forms. Bacteria procreate by simple fission, and are therefore known as *fission-fungi* or *schizomycetes*. Some forms also produce spores. *The biological properties* include motility, color, the growth on various culture-media and under various temperatures, and the product of vital activity.

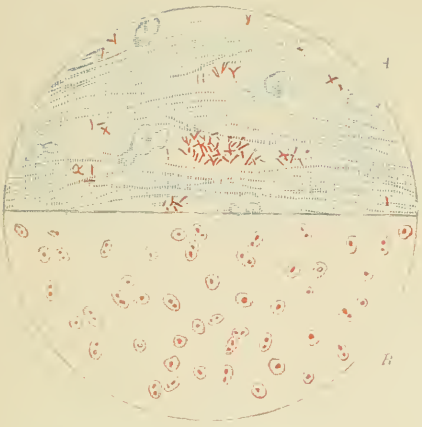
Micrococci.

Morphology. To this group belong the spherical bacteria. Each coccus is of nearly equal diameter in all directions. They vary in size from 0.1μ to 1 or 2μ. A micromillimetre (μ) is one twenty-five thousandths of an inch. The various micrococci resemble each other so much in form and size that they cannot be distinguished by their microscopic appearances. To distinguish them we depend on the color and character of their growth in various culture-media, on their pathogenic power, and on other biological differences. The mode of grouping, after fission or reproduction, is an important characteristic by which varieties are differentiated. Just before dividing they are not perfectly spherical, but short or long, oval. After division (for they divide indefinitely when growing) the *staphylococci* are solitary or in pairs, or, occasionally, in groups of four or in clusters, roughly likened to a bunch of grapes, from which latter grouping they derive their name. The organism is called a *diplococcus* when associated in pairs. Sometimes two or four are included in a capsule. *Zo̤oglœa* are groups of cocci held together by a transparent glutinous substance. *Streptococci* are characterized by a grouping in chains, known as *chaplets* or *torula* chains, because division takes place in one direction only. When division takes place in two directions, groups of fours, or *tetrads*, are formed; and when in three directions, groups or packets of eight are formed, of which the *sarcinæ* are the most familiar examples. These names, significant of the grouping, refer to the *predominating* groups as seen in microscopic preparations. In some of such groups, for instance, are seen only diplococci or streptococci; but in all, transitional, irregular, and accidental groupings may be observed.

Biological Characteristics. Micrococci are not *motile* and do not form spores. *Products of vital activity:* The various forms of bacteria are also distinguished by noting the difference in the products of vital activity. Of these, *pigment-production* is one of the most apparent. The *staphylococcus pyogenes aureus* and *citreus* are chromogenic or pig-

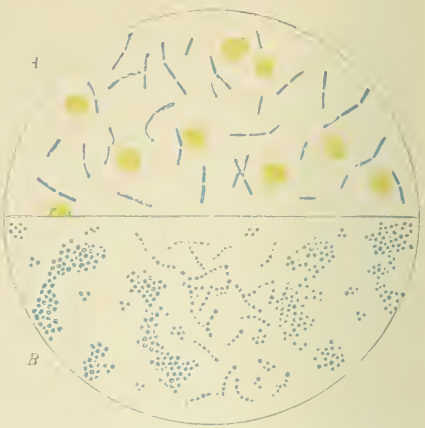
PLATE III

Fig 1



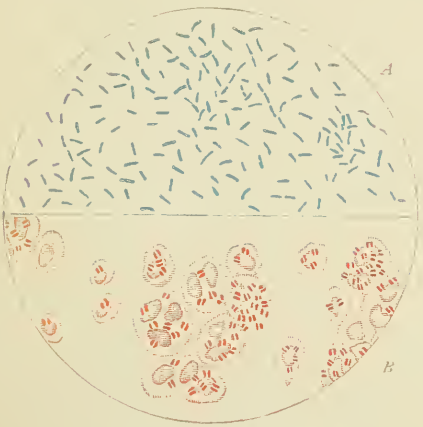
A Tubercle-bacilli. *B.* Pneumococcus.

Fig 2



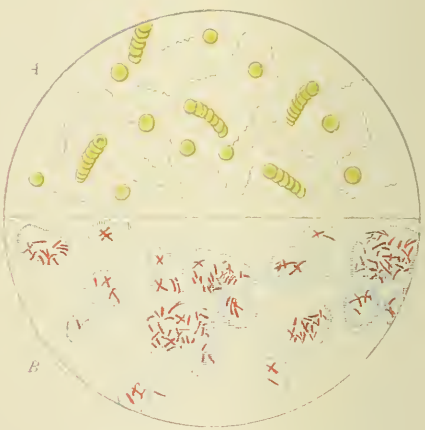
A Anthrax *B.* Streptococcus and Staphylococcus.

Fig. 3.



A Comma-bacillus. *B* Gonococcus.

Fig. 4.

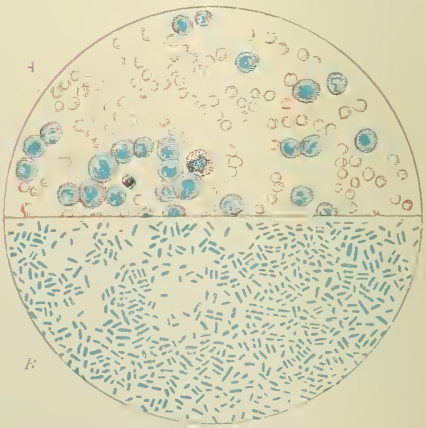


A Recurrent Spirilla *B.* Leprosy.

Fig 5

A Normal Blood. *B.* Normal Blood

Fig. 6.



A Leukæmia *B* Eberth's Bacillus

ment-producing bacteria. The *liquefaction of gelatin*, when cultures are made, is a biological characteristic which assists in the diagnosis of the various species. Some pathogenic as well as non-pathogenic germs have this effect on the nutrient medium; others of both classes do not affect it. A peptonizing ferment is formed during the growth of cells which acts upon and dissolves the gelatin. The amount, degree, and character of the liquefaction serve to distinguish various species. The *staphylococcus pyogenes aureus* and *albus* (as well as some others) are liquefying micrococci. *Production of acids:* Many bacteria produce an acid—lactic acid, acetic acid, butyric acid—which gives an acid reaction to the culture-media. This may be seen if a neutral litmus solution has been added to the gelatin. The pink color produced indicates the presence of an acid. Culture-media, it must be remembered, are alkaline or neutral. The pathogenic micrococci which produce an acid are the staphylococci of pus—lactic acid.

Putrefactive fermentation is set up by bacilli and not by micrococci. Other products of vital activity need not concern us, as they are produced by non-pathogenic forms.

Toxic ptomaïnes and *toxalbumins* are products of many forms of pathogenic bacteria, and cause the symptoms of infective diseases in many instances; thus in diphtheria the local infective inflammation represents the seat of activity of the bacillus, the point at which its poisons are being manufactured at the expense of the tissues in and on which it is growing; the general symptoms are due to the toxalbumin that has been absorbed by the circulating fluids from this local seat of action. The isolation and detection of the toxalbumins are not sufficiently easy to warrant such a mode of investigation for diagnostic purposes. Often the results of inoculation, by which the lethal effect is produced, aid in the diagnosis of the suspected ailment. (See Plate III., Fig. 2, *b*.)

The Bacilli.

Morphology. The bacilli, or rod-shaped bacteria, differ widely in form, in size, and in modes of grouping after fission. *Form* and *size:* The longitudinal diameter is greater than the transverse, and the forms vary from short oval or slender rods to long filaments; sometimes short rods and long filaments are seen in pure cultures of the same bacillus, as in the typhoid bacillus. The transverse diameter of a given species does not vary, as a rule. The form of the extremities of the rods must be observed. They may be square, slightly rounded, round, oval, or lance-shaped or spindle-shaped. *Reproduction* and *grouping:* Fission or reproduction takes place by binary division, transverse to the longitudinal axis. They group in long chains, or are solitary or united in pairs. They may be surrounded by a capsule or collect in zoöglœa masses.

Spores. When conditions unfavorable to continuous multiplication by transverse division arise certain bacilli possess the property of entering into a permanent or resting stage. In this case there develops within the body of the bacillus an oval, egg-shaped structure—an endogenous spore. The spore represents the inactive stage, and lies

dormant until circumstances favorable to growth reappear, when it develops into a bacillus identical with that from which it was formed. Spores do not develop into spores but into bacilli. The spores retain their vitality for months or years, and resist desiccation. They are spherical or oval, and highly reproductive. They are formed by condensation of protoplasm at the centre or at one end of the bacillus, where they are retained in a linear position until set free. Some bacilli grow into long filaments during spore-formation ; others change their shape, swelling at the centre, becoming spindle-shaped or club-shaped, according to the location of the spore within it. Many bacilli do not change their shape at this stage. The spores are free or collected in masses with the bacilli as well as located in the parent bacillus.

Motility. The bacilli are often actively motile, because of the presence of flagella. The movement is one of progression in different directions. It may be slow and deliberate, in a to-and-fro motion, or serpentine, or a quick, darting forward motion.

Biological Characters. *Products of vital activity.* They may be ascertained in the same manner as in the study of micrococci. *Pigment-production* is seen in cultures of the bacillus pyocyaneus or bacillus of green pus, of which there are several varieties producing various shades of blue or fluorescent green. *Liquefaction of gelatin:* This is produced by the bacillus anthracis and the bacillus pyocyaneus, the "comma" bacillus of cholera and many other species. *Production of acids:* The bacillus coli communis produces lactic acid. *Fermentation:* The latter bacillus sets up fermentation of carbohydrates, as of glucose, lactose, and saccharose. (See Plate III.)

The Spirilla.

Morphology. They are seen in the form of curved rods or spiral filaments. The shorter ones are curved, the longer are spiral, like a corkscrew. The curved filaments may be short and rigid, or long and flexible.

Reproduction. They reproduce by binary division (fission).

Biological Characters. *Motility.* They are motile ; the movement is rotary, as well as progressive in the direction of the long axis of the filament. The presence of flagella is determined by Löffler's method of staining. They are single at the ends of rods, or several are seen at one extremity, or one or more may occur at both ends. *Pigment-production:* Pathogenic spirilla do not produce pigment. *Liquefaction of gelatin:* The spirillum of cholera Asiatica (comma bacillus) and the spirillum of cholera nostras (Finkler and Prior) both liquefy gelatin in a peculiar manner. (See Plate III., Fig. 4, *a*.)

CHAPTER XVII.

THE DATA OBTAINED BY OBSERVATION—(*Continued*).

Data obtained by inquiry—By observation. Local infection—General infection. Pyæmia; septicæmia. Terminal infections. Fever in carcinoma. Afebrile infections. Infections of certain bacteriology; of uncertain bacteriology. Bacteriological diagnosis. Method of research: Microscopical examination, cultivation, inoculation. Essentials in technique.—*Method of research:* Blood, discharges, exudations; mode of collection. Apparatus. Preparation of apparatus. Sterilization. Microscopical examination: Technique, cover-glass preparations. Methods of staining; spores. "Hanging drop"—Cultivation of micro-organisms. Culture-media. Tube- and plate cultures. Smear- and stab-cultures—Inoculation of animals—Special bacteriological diagnosis.

FEVER. THE INFECTIONS.

Unfortunately, the cause of many of the infectious diseases has not been definitely isolated. This group is largely the infectious disorders which are epidemic and contagious. In order to diagnosticate them it is necessary to associate with the mode of onset and clinical course of the disease the facts and laws pertaining to epidemics and to contagion. Data, therefore, obtained by *inquiry* are quite necessary to establish the diagnosis. Such data are useful in confirming the results of an objective or bacteriological examination of the patient, even though the diagnosis be at once established by the latter method.

Data Obtained by Inquiry. In the first place, we note the *social history*, learning this while preparing for the objective examination. It should be personal and general. The age, the sex, the habits, the occupation, are looked into. The nature of the prevailing diseases in the community are known or sought for, and all possible unusual circumstances in food, drink, clothing, are inquired for. In short, a history of exposure to influences which attend an intoxication or those which permit infection are to be zealously sought for.

An inquiry for *previous diseases* does not imply a history alone of a previous infectious disease, but a history of such diseases as are often followed by infection. Thus, a history of a previous attack of gallstones or of renal calculus may be a clue to the localization of an infectious process. Too much stress cannot be laid upon the diagnostic value of the data obtained in this manner.

The next data obtained by inquiry is the history of the *present disease.* The mode of onset is of itself suggestive. Sudden onset points more closely to an intoxication, though not necessarily, although more likely in children. Otherwise sudden onset usually indicates one of the short infections, of which scarlatina and pneumonia are representa-

tive types; while gradual onset, a long infection, of which typhoid fever is a type.

The *subjective symptoms* are then inquired for and their site affords a clue as to the steps to be taken in the objective examination. Thus, pain in the throat with difficulty in swallowing calls for an examination of the fauces; pain in the chest, of the lungs; in the præcordia, of the heart, etc. Any functional disturbance of an organ should also lead us to a study of it.

Data Obtained by Observation. The appearance of the inflammatory process may be sufficient to decide its nature, however—a boil, an abscess, a carbuncle, which gives rise to more or less fever, because they are local infections, are readily recognized.

Local Infection. When not preceded or accompanied by any process elsewhere the infection is said to be *local*. An appendicitis, a cholangitis, an inflammation of a serous membrane, as well as a boil or carbuncle, may be a *local infection*. In like manner the accidental wound of a surgeon by which he is inoculated or infected by the micro-organism of the pus may be an infection. The natural or acquired wounds of the puerperium may also be infections. A *local infection* here arises. It must be borne in mind that any local inflammation may be infectious. It is not our purpose to consider here local infections. Some, indeed nearly all, of the streptococcus and staphylococcus infections are local. The general symptoms are produced by a toxæmia, the toxin alone passing into the blood.

General or Systemic Infections. General infections alone, and those which may have more pronounced local expression, as pneumonia or the pneumococcus infection, are discussed. It is of importance, however, to remember that in determining whether a local inflammation is infectious or not, we use the same methods that are employed to determine the nature of a general infection.

It is also important to remember that a local infection may be circumscribed and cause a toxic fever. On the other hand, a small portion of the purulent exudate from the infection may get into the circulation and be carried to distant parts, as the brain, the lungs, the kidney, the joints, the spleen. Distant foci of inflammation are set up, giving rise to multiple small abscesses in the organs affected. *Pyæmia* is the name of this form of systemic infection. Finally, such local infection may become general and the case terminate in *septicæmia*.

Pyæmia is characterized by rigors, fever, usually intermittent, and sweats. There is exhaustion; the skin is slightly icterode. The odor of the breath is sweet. There is anorexia, nausea, perhaps vomiting, frequently diarrhœa. Erythematous eruptions are seen. With these general symptoms there are present the physical signs of abscess in the lungs or the spleen or other organs of the body, or we may have an endocarditis. When the affection is limited to the portal area, and multiple abscesses of the liver succeed a purulent process in the area of the portal vein, the general symptoms are combined with enlargement of the liver, which is tender and painful, and perhaps with deeper jaundice. The micro-organisms which invade the system and cause areas of suppuration are the streptococcus and staphylococcus pyo-

genes, the micrococcus lanceolatus, the gonococcus, the bacillus coli communis, the bacillus typhi abdominalis, the bacillus proteus, the bacillus pyocyaneus, the bacillus influenzæ, and the bacillus aërogenes capsulatus.

Diagnosis. Pyæmia resembles in many respects tuberculosis of the kidneys and calculous pyelitis, in both of which recurring rigors and sweats are common. In gross aspects it resembles malaria. (See Intermittent Fever.) In prolonged cases of pyæmia the symptoms may resemble typhoid fever, but leucocytosis is present in the former condition. Ulcerative endocarditis and acute miliary tuberculosis usually resemble septicæmia, but may be confounded with pyæmia. Any febrile process associated with chills may be taken for pyæmia. These phenomena are seen in grave anæmias, in Hodgkin's disease, in hepatic intermittent fever, and in the intermittent fever of carcinomatosis. (See Chills, Chapter XIV.) Post-febrile arthritis, after scarlet fever and gonorrhœa, is in all probability pyæmic. Of course, we rely in the diagnosis of pyæmia upon the data obtained by bacteriological methods when their employment is practical.

Septicæmia. Again, we may find with the above-described wound, or without any apparent local inflammation, *fever*, which is more or less continuous. In addition there may be an occasional rigor. The pulse is rapid, exhaustion, anæmia, and some emaciation are present. Secondary infection of other structures may or may not be present. Microbic infection of the blood usually takes place. The process is a *septicæmia*. If it originates from a local infection it is known as *progressive septicæmia*. If independently of any apparent local infection it is a *cryptogenetic septicæmia*. The former is easily recognized, particularly if there is a history of a primary local infectious process. The micro-organisms which may give rise to the latter are the staphylococcus pyogenes, the streptococcus pyogenes, the bacillus proteus, the bacillus pyocyaneus, and the micrococcus lanceolatus. It is recognized by a bacteriological diagnosis.

The accompanying chart (Fig. 42) represents the course of an infection and various areas of secondary infection in a general septicæmia. The illness extended over a period of thirty-five days. The first five days, as indicated by the chart, there was pneumonia at the base of the left lung. The crisis only is represented. From the tenth to the twenty-first day, to save space, the chart does not give the temperature range. During this time the fever was continuous. On the twelfth the right pleura was infected; on the nineteenth the femoral vein of the right leg, the temperature not rising above 101°. On the twenty-first, as the chart indicates, a patch of pneumonia was found in the right lung posteriorly. On the twenty-fourth pseudocrisis, and on the twenty-fifth and twenty-sixth the true crisis took place. On the twenty-ninth and thirtieth there was reinfection of the pleura of the left side. On April 3d phlebitis of the femoral vein of the left leg developed. During the course of the disease there was a low-pitched endocardial murmur, which in all probability was anæmic. Sweats, attacks of collapse, and irregular rigors took place. Life was imperiled at the time of the collapse. The spleen was enlarged; the sputa con-

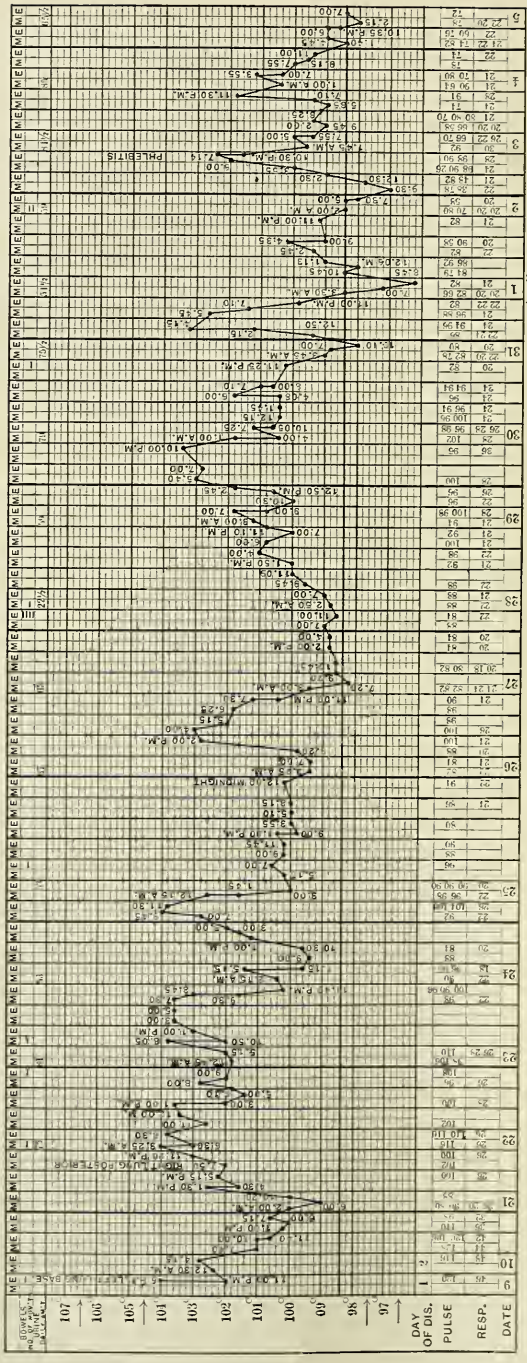


Fig. 42.

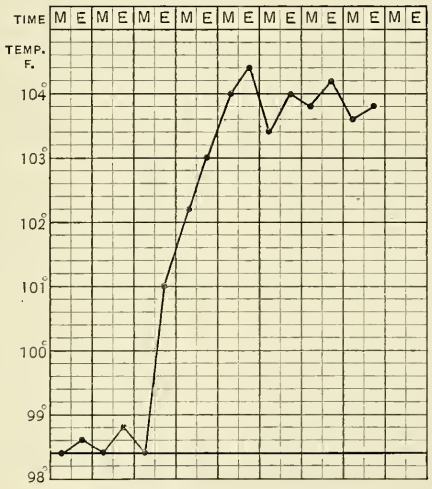
General infection or septicæmia, probably pneumococcus.

tained pneumococci. The blood examination was negative. The patient recovered.

Fever, varying in type, sweats, emaciation, anæmia, and exhaustion are the common general symptoms. The pulse is increased in frequency, and is dicrotic and compressible. The heart sounds grow weak, the breathing hurried. There is slight delirium at times. The urine contains albumin and casts. It is scanty, high colored, and of high specific gravity. In some forms there is leucocytosis. There is anorexia, nausea, and vomiting, often diarrhœa. As the case advances the symptoms of the typhoid state develop. (See Chapter XIV.)

Objective Signs of Septicæmia. In other instances there is marked evidence of a septic process in the structures which carried the poisoned or infected blood from the primary point of entrance of the infecting material—the *infection atrium*. Hence in this infectious pro-

Fig. 43.



Temperature record of septic infection.

cess—in septicæmia—we may see *lymphangitis* and *adenitis*. The *spleen* is enlarged. There may be *phlebitis*, especially of the femoral, inflammation of which is always infectious in character. Other *veins* may be affected. The *endocardium* is infected, and indeed *endocarditis* may be the chief symptom-complex of the septic process. The serous membranes may be involved, so that septic *pleurisy* or *meningitis* or *pericarditis* or *peritonitis* or *arthritis*, singly or combined, may be local expressions of the sepsis. *Hemorrhages* from the mucous membranes, or subcutaneously, either because the blood is destroyed (toxic) or because of multiple small infarcts, frequently attend septicæmia Hemorrhages may be the most pronounced symptom of certain forms of infection, as that due to capsulated bacilli. A slight *jaundice* of toxic origin may prevail.

Toxic Symptoms. In some instances there is a profound *toxæmia* indicated by delirium, stupor, and later coma and convulsions. The

typhoid state may predominate. The intoxication may overwhelm the cardio-vascular centres. The pulse grows rapid and feeble, the respirations hurried and shallow. The urine is diminished in amount and contains albumin.

The clinical course varies with the infective agent. Streptococcus infections are characterized by chills, high fever, and an extreme septic state. Infection by the capsulated bacilli (Howard) gives rise to a hemorrhagic septicæmia. In other infections the greater part of the clinical course may be afebrile. Toxic symptoms, and especially increased frequency of pulse-rate with collapse phenomena, are present, as in forms of infectious peritonitis.

A general infection, or this general expression of septicæmia, occurs in the course of diseases in which the clinical course of the infectious process is usually a definite one. Hence we speak of typhoid septicæmia or a pneumococcus septicæmia when the intoxication or general infection is paramount to the local process. Then in tuberculosis and other prolonged infections septicæmic symptoms arise, so that the terminal phenomena of the disease are usually due to a *mixed infection*. It must then be understood that *pyæmia* or *septicæmia* or septico-pyæmia are not due to special micro-organisms in the sense that typhoid fever is due to the bacillus typhosus, malaria to the plasmodium, or pneumonia to the pneumococcus.

Terminal Infections. At the termination of many chronic diseases, as the various fibroid affections—cirrhosis of the liver, the kidneys, endarteritis, or spinal cord disease, and in carcinoma—there is *fever*. This is generally due to an infection which the weakly resisting organism invited.

Flexner has studied the terminal infections. In 255 cases of renal and cardiac disease he found 213 infections, excluding tuberculosis. They were local and general. Infections of the serous membranes are the most common. The old clinical fact that serous inflammations were complications of Bright's disease has been proven by bacteriological methods to be due to an infection, and not as formerly thought to a chemical change in the blood. The following micro-organisms are met with: the streptococcus pyogenes, the pneumococcus, the staphylococcus aureus, the bacillus proteus, the gonococcus, the bacillus pyocyaneus, and the gas bacillus.

Tuberculous infection is also a terminal process in many diseases. Frequently an acute tuberculosis of serous membranes is found in the course of chronic heart or kidney disease.

Fever in Carcinoma Fever occurs in the course of carcinoma under two circumstances. If it is proven that carcinoma is an infection this process is one cause of the fever. It is well known, however, that in rapidly growing cancer of the liver, fever, often intermitting in type, is present. It may also be present in general carcinoma, and in all probability in carcinoma of the lungs and of the bones. But fever in the course of cancer may be due to a secondary infection. It can be readily understood that the process is likely to take place if the malignant disease occurs in the course of any of the tubes or channels. The *infection atrium* is the inflammation or ulceration found so often in

carcinoma, and in consequence local suppuration occurs. From this local infection a general septicæmia may arise.

Afebrile Infection. Although most infections cause such reaction of the system as to produce fever, some few are afebrile. Such is the case with tuberculosis—when it is local—and of syphilis in certain stages. The writer is of the belief that when the syphilitic poison is active—*i. e.*, productive of lesions—fever is present some time during the twenty-four hours. He is fully persuaded that mistakes are made because fever is not considered a part of the syphilitic infection. He has seen it in all of the arbitrarily called stages of infection, and presenting all types of fever—intermittent, remittent, and continuous. The rise may be moderate or very pronounced. For its detection the thermometer should be employed every two hours.

Typhoid fever is an example of an infection which sometimes runs its course without rise in temperature. This is very rare, but nevertheless does at times occur.

Infections of Certain Bacteriology. In our investigation of the cause of the *fever* in a suspected case we have found evidence of an infection as shown by (1) the phenomena of a local inflammation, (2) by the presence of pyæmia or (3) of septicæmia. The clinical course alone enables one to make a diagnosis generally. At times we may have to resort to more positive methods. The nature of the process, however, is usually proven ; the nature of the infection must be decided by bacteriological examination. We must, therefore, follow this mode of recognition of an infection.

Infections of Uncertain Bacteriology. The presence of those infections, the bacterial cause of which is not known, must be determined somewhat differently. In one group we must be content with the data obtained by inquiry and by observation, comparing the symptoms with the known course of a similar disease. Scarlet fever can only be recognized in this way. In subsequent chapters, therefore, the infections are divided into those recognized by inquiry and observation, and those recognized by supplementary observation with bacteriological methods.

The classification of the infectious diseases is based upon the fact that a specific micro-organism is known which gives rise to phenomena similar in the respective infections. In other words, the infection of malaria or of tuberculosis or of diphtheria follows a recognized clinical course. The period of invasion, the mode of onset, the symptoms throughout the course of the disease, are with notable exceptions practically the same.

Bacteriological Diagnosis.

Bacteriological examination includes (1) the finding of the specific micro-organism in the blood or tissues (of the subject) or in the pathological secretions or excretions ; (2) the isolation and cultivation of the micro-organism ; (3) the inoculation and the reproduction thereby of the disease in animals. In many infections the morphological peculiarities of the micro-organism are so characteristic that a diagnosis may be established by finding it in the blood or the secretions. Thus an

examination of the blood, with or without staining, will disclose the presence of the micro-organism of relapsing fever and of anthrax and the protozoa of malaria. The examination of inflammatory products of an infection, as the sputa in pneumonia or tuberculosis, are sufficient to determine the nature of the infectious inflammation of the lungs. On the other hand, in some infections, the absence, or rather failure of detection, of the micro-organism in the fluids or discharges is not proof that the disease is not present in the suspected individual. The infection tuberculosis well illustrates the propositions in the last two sentences. If the bacillus is found in the sputum of a suspected case the diagnosis is established definitely, and no further procedures for diagnostic purposes are necessary. In other clinical forms, as tuberculous pleurisy, or empyema, or glandular or joint tuberculosis, the micro-organisms are few and difficult to find. Cultures, or, more conclusive still, inoculations, must frequently be resorted to before a final conclusion can be arrived at. It is possible that spores alone exist—morphological elements difficult to detect by staining and microscopical methods, but which may rapidly multiply under favorable culture conditions or inoculation conditions. Again, micro-organisms have been found in certain infections, and although thus far their causal relationship to them has not been fully proved, nevertheless their constant occurrence in the special affection, and in it alone, renders their presence of high diagnostic value. Thus the amœba of dysentery and the plasmodium malariæ of Laveran are diagnostic of their respective affections.

For diagnostic purposes bacteriological investigations must be conducted in accordance with the methods of bacteriology. Such researches are possible at this time, because of (1) the high degree of development and mode of use of optical apparatus, including oil-immersion lenses, Abbe's condenser and diaphragms ; (2) the discoveries by Weigert of the effects of aniline dyes on protoplasm, and the property of micro-organisms of taking different stainings ; (3) of the principles of sterilization by heat, by which foreign micro-organisms are excluded ; (4) of the use of solid culture-media, and the plate-method of obtaining pure cultures suggested by Koch.

Method of Procedure.

To determine the presence of moist infections it is necessary to proceed as follows :

A. Examination of the blood.

B. Examination of the pathological secretions and excretions.

C. Examination of products of infectious inflammation secured by exploratory puncture or evacuation of abscesses. (See Chapter XXI.)

D. Inoculations of animals with pure cultures of the organism or with the products of inflammation, as cheesy matter from a tuberculous abscess.

E. The use of products of bacterial growth to secure reaction, as tuberculin in tuberculosis, and mallein in glanders. (See Tuberculosis.)

When there is no distinctive pathological fluid all the fluids of the body must be examined. In other cases the pathological discharge

(pus), or perhaps the diseased tissue, must be studied. We get a clue to the direction which the examination is to take from the nature of the symptoms. In cases of pulmonary disease, the sputum; of faucial disease, the membrane, pus, or other secretions from the fauces; in intestinal disease, the discharge from the bowels; and in genito-urinary disease, the urine. It must not be forgotten that in many, even highly fatal diseases, the blood is not invaded by micro-organisms. Death is due to the development of toxic substances. Hence, as in cholera and diphtheria, the presence of the micro-organism is not sought for in the blood, but in the specific excretion or exudation. (See Tuberculosis.)

The Apparatus. The *apparatus* necessary for the simplest bacteriological research comprises the following: Sterilizers, incubators, glass flasks, covered dishes, test-tubes and plates, platinum needles fixed in glass handles, raw cotton, materials for culture-media, microscope, with slides and cover-glasses, and, in addition to lenses of lower powers, a one-twelfth oil-immersion lens, and finally the various stains used.

Preparation of Apparatus. Boil all glassware for half an hour in a solution of common soda (2 to 3 per cent.), then scrub thoroughly; rinse in warm solution of HCl (1 per cent.) and then in pure water; drain with tops down; plug tubes and flasks with raw cotton, fitting firmly and evenly, so that the cotton can hold the weight of the test-tube; sterilize in dry oven. The test-tubes (plugged) are placed in a rack for further use.

The tubes and flasks are best filled with the culture-media through a spherical funnel that can be plugged with cotton. Then they are to be sterilized in the steam sterilizer, as heretofore described.

The cover-glasses must be thoroughly cleansed by immersion in strong nitric acid for a few hours, then rinsed in water, then in alcohol and ether. They are then kept in alcohol.

Sterilization. It should be understood that the first requisite for the prosecution of these studies is to secure absolute cleanliness and to prevent the invasion of extraneous micro-organisms. The first step is thorough sterilization of all appliances required for work and of all the media, to destroy previously existing bacteria.

The sterilization is best accomplished with steam, where the objects to be sterilized admit of it. With dry heat a temperature of at least 150° C. must be applied for at least an hour, and, of course, can only be used for glassware and metal instruments. All media (see page 159), whether solid or fluid, are sterilized by steam. Media which cannot withstand long exposure to the necessary heat are sterilized by the intermittent application of steam. The reason that this is effective is that fully developed bacteria are destroyed at a much lower temperature and with shorter exposure than are the spores. One application kills the developed bacteria, then the material is kept for a time in an incubator; spores develop into bacteria and are easily killed by a second application. By repeating this process from three to five times the substance is effectually sterilized. If the exposure is made longer a much lower degree of heat may be used, so that in the case of blood

serum it may be sterilized without coagulating the albumin. Usually an exposure of fifteen minutes to steam on each of three successive days is used for stable media, and an exposure of an hour on six successive days to a temperature of 70° C. for more delicate media, as blood serum. In the intervals the material must be kept at a temperature of 25° to 30° C. A single application of steam under one to one and one-half atmosphere pressure is now often used.

The ordinary "Arnold steam sterilizer" is as good as any. The dry sterilizer is merely a metal box with copper bottom and ventilating holes. It is well to have an asbestos casing.

Metallic articles, as forceps, platinum probes, etc., are best sterilized in the flame of a Bunsen burner.

Collection of Material. A definite, careful method must be observed when the pathological product is removed from the patient or collected for investigation. (See Chapter V., Exploratory Puncture.) Pus and fluids should be placed in sterilized glass bottles or tubes, care having been taken that instruments for the removal of the fluid were previously sterilized. Exposure to air should be as brief as possible. The fluid should not be contaminated with blood or antiseptic fluids used for flushing or other surgical procedure. If an abscess is opened or purulent peritonitis cut down upon, for instance, tube-inoculations can be made at the bedside. The previously sterilized platinum point should be kept before use in a test-tube closed with sterilized cotton. It is dipped into the pus before it flows over the skin, and the pus should be free from the blood of the incision. It is at once transferred to the medium in the test-tube. Sputum should be collected in a previously sterilized bottle, or one thoroughly cleansed by boiling. The bottle should have a wide mouth. Care must be taken to secure sputum from the lungs, and not the secretion from the mouth and fauces. Purulent portions, rather than mucoid, are to be sent for examination. Intestinal discharges may be collected in sterilized glass jars and examined as soon as practicable. It may be necessary to keep the discharge at the temperature of the body. (See Fæces—amœba dysenterica.)

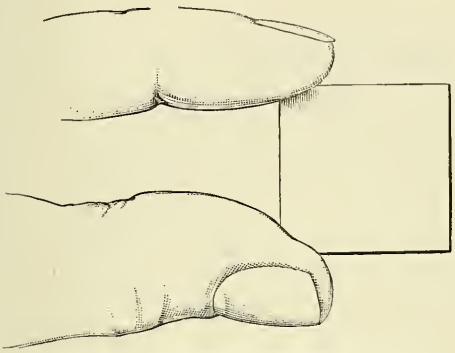
Examination of Blood.

To secure *blood* for microscopical study the finger must be thoroughly cleansed with alcohol and puncture made with a sterilized lancet or needle. After the blood flows a few seconds it is removed and the cover-slip, previously cleansed in nitric acid solution, is gently pressed upon the second overflow. Another cover is placed over the blood-stained surface of the first slip, the two rubbed together and separated by sliding them apart. (See Fig. 45.) Sternberg prefers to spread the blood, which was collected at the edge of the cover-slip, by drawing a polished glass slide, held at an acute angle, over the cover-slip. In either case this thin film of blood is allowed to dry, and can be examined later. Sternberg mounts the blood on a glass slide at once.

1. Microscopical examination is made of the fresh blood. 2. Smear preparations on cover-glass or slide are made for staining. 3. A drop

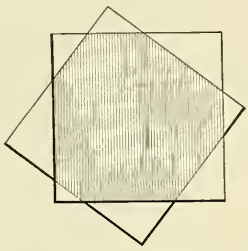
of the blood is examined to observe the biological properties known as agglutination, or the Widal reaction—the *serum diagnosis*. 4. The number of white corpuscles is counted, to show the presence or ab-

FIG. 44



Proper method of holding a cover-glass. (CABOT.)

FIG. 45.



Illustrating the position of cover-glass during the spreading of blood films. (CABOT.)

sence of leucocytosis (see Blood), and a differential count of these corpuscles is also made. 5. The fresh blood is inoculated on media for cultures.

I. Fresh blood is examined with the oil-immersion objective and the diaphragm of the sub-stage condensing apparatus (Abbe's) nearly closed. The protozoæ of malaria, the bacillus of anthrax, and the spirillum of relapsing fever may be detected.

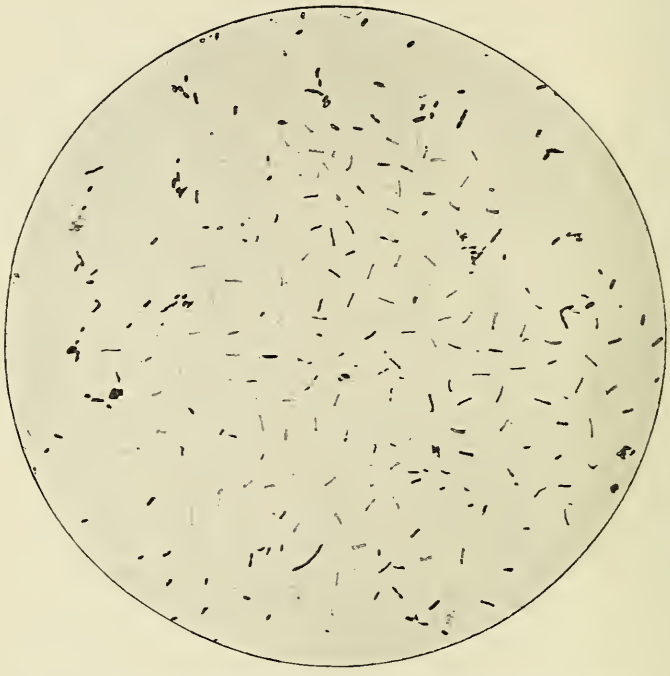
II. Cover-glass preparations are examined with the diaphragm open. The micro-organisms above mentioned and those of yellow fever and typhoid fever may be found in this manner. The method of staining the blood is described below. The following solutions are used: 1. Basic aniline dyes. 2. Löffler's alkaline methyl-blue. 3. Gram's.

III. SERUM DIAGNOSIS. The phenomena of agglutination consists in the gradual approximation, clumping, and loss of motility in the micro-organisms of some infectious disease when the blood of a patient suffering from that disease is brought in contact with it. This is known as the serum, or Widal reaction, and by means of it a number of infectious diseases can now be recognized. If a drop of bouillon culture is examined with a high-power lens the organisms are seen darting about and across the field with great rapidity in various directions. If to ten drops of a pure culture of certain varieties of infectious micro-organisms one drop of the blood of a patient suffering from that infection be added the motility of the organisms is checked and clumps appear in the field. The clumps enlarge rapidly, so as to be easily visible under a magnifying power of 500 diameters.

Serum from patients suffering from other diseases or from healthy patients does not produce agglutination if the proportion of serum to culture in the mixture is 1 to 10 or less. The reaction is specific. Thus typhoid bacilli are not clumped by any serum other than that of a typhoid patient or a patient immunized against typhoid fever by a more or less recent attack of the disease. Typhoid serum clumps no

organism except the typhoid bacillus when used with a certain degree of dilution and examined within a certain period of time.

Fig. 46.



Bouillon culture of typhoid bacilli before the addition of diluted typhoid serum. (Magnified 500 diameters.) After Cabot—serum diagnosis.

Serum diagnosis has become a valuable mode of recognition of typhoid fever, Malta fever, yellow fever, and glanders. It may be of use in other infections, as cholera and the pneumococcus infections. They are more accurately diagnosticated by other bacteriological methods, however, and need not be considered here.

Method. Three methods of securing the serum reaction are employed: microscopic, or quick test of the fluid serum or blood; the microscopic, or quick test of the dried blood; and the macroscopic, or slow test. Each of these methods is of value. The observer should select one and make it his object to become thoroughly familiar with that selected.

First, the *quick test with fluid serum.* The steps are: first, to collect the blood; second, to add it in certain proportion to the fluid culture; third, to examine the slide and cover-slip.

1. Collecting the Blood. The blood is secured by puncture as in the method described in diseases of the blood. If the ear is selected it can be bled freely or blood squeezed out by the milking process until about fifteen drops are collected in a small test-tube. It is not necessary to observe strict antiseptic precautions as in other instances. The

instruments and test-tube should be thoroughly cleansed. The blood thus collected is allowed to coagulate in the tube, which may occupy several hours. It is to be remembered that the clot collects on the

FIG. 47.

The same, five minutes after the addition of typhoid serum (dilution 1 : 10), showing typical clump reaction. (Magnified 400 diameters.) (CABOT.)

sides of the tube and over the surface of the blood. To secure the serum this clot must be removed with a bit of wire.

2. DILUTION. One drop of the serum is added to forty drops of a bouillon culture. The same dropper must be used for each fluid, in order that the size of the drops will be equal. The fluids are to be mixed intimately in a small test-tube. A drop of this mixture of culture and serum is placed upon a cover-glass, which is then inverted over a hollow ground slide and examined under the microscope with the immersion lens. Within twenty minutes clumping should take place. If the reaction does not take place a new mixture should be made, in the proportion of 1 to 20 or 1 to 10. If there is no reaction with this dilution the test is negative. Instead of making successive mixtures three tubes can be prepared at once, containing ten, twenty, and forty drops each of the culture. A drop of serum can be added and the test conducted as above.

3. EXAMINATION OF SLIDE. A No. 7 Leitz dry lens or oil-immersion lens can be used with a No. 3 or No. 4 eye-piece. Artificial light is preferable to daylight; if the latter is used a small aperture diaphragm is the best. It is very necessary that the slide and cover-slip should be thoroughly cleansed.

The Reaction. In a complete or typical reaction the field shows the presence of large clumps of bacilli isolated and motionless. (See Fig. 47.) No motile bacilli can be seen. The clumping may occur instantaneously or gradually. If the reaction is very marked, Greene states a mottling can be seen with the naked eye. Clumping and cessation of motion are the essentials of the reaction, providing they take place within a certain time, and notwithstanding a certain degree of dilution of the serum. When the reaction is feeble small clumps appear, or, as Widal calls them, agglutination centres. As the field is studied bacilli are seen moving toward the centres and gradually ranging themselves in loose masses, sometimes like the spokes of a wheel. Durham has called attention to a peculiar spinning motion of the bacilli around one of its own ends, which is seen in some of the fields in which a few isolated bacilli remain. Such movements occur at the margin of the clump.

It is very necessary to examine a drop of the pure culture before the addition of any serum, to make sure that clumping has not already taken place, particularly if the culture is old or has undergone sedimentation. It is desirable that the bacilli should be isolated and actively motile.

Time Limit and Dilution. As Cabot forcibly states, only when clumping occurs within a certain time and in a certain degree of dilution is it of diagnostic importance. The test is quantitative and not qualitative. The degree of dilution of 1 to 10 is quite sufficient if the time-limit for the reaction is at least fifteen minutes. Any clumping of typhoid bacilli which takes place fifteen minutes after one part of serum has been added to ten of the culture gives a probable typhoid reaction. Various observers select different dilutions. Thus, Wilson and Westbrooke make a dilution of 1 to 50 with a two-hour time-limit. Durham uses a dilution of 1 to 17 or 1 to 20.

Instead of the serum from the blood the serum of a blister may be used, or the serum from blood which has been drawn directly from a vein with antiseptic precautions.

The whole blood can also be used in a fluid state. A drop of the blood can be drawn directly into ten drops of the culture previously measured. This method is of great advantage for rapid work. The same dropper should be used for measuring the culture and subsequently the blood. With the microscope at the bedside the test can be made rapidly with but little risk of failure.

A still more convenient method consists in the employment of the pipette, used for diluting the blood in counting leucocytes. The blood from the finger is drawn up to the 0.5 or 1.0 mark on the stem, and the bulb then filled with distilled water. The mixture is then blown into a small test-tube. As the dilution has already been made, a drop of bouillon culture or a small portion of an agar culture may be added to it directly and examined as above.

The Reaction with Dried Blood. We owe to Wyatt Johnston, of Montreal, the great credit of working out this simple but accurate method of performing the reaction. It is of special value for sanitary work where blood has to be sent by mail for examination. The method

is simple. The blood is collected on glass or *glazed paper*. In this manner it can be preserved for an indefinite time and transported easily. If the drop of blood is dried on a glass slide it can be dissolved by the addition of a little water and then the culture added in the way previously described. If the drop is dried on paper it can be cut out with a pair of scissors and rubbed up in a watch-glass with one drop of water. When the blood is dissolved ten drops of culture are added, and the examination is carried on as in the previous method.

Some operators collect the blood in the eye of a wire loop of a given size, and after placing it on a glass, dilute with water in the proportions desired, ten loopfuls of water being the amount usually selected to mix with the drop in the wire loop. Wilson and Westbrooke have modified Johnston's technique as follows: They use a bit of platinum wire, number 19 gauge, one end of which is bent into a loop, the inside diameter of which is 2 mm. The loop is used to collect the blood, several drops of which are deposited on a bit of aluminium foil, number 40 gauge, 5 cm. square. After the blood is dried the foil is rolled up. At the laboratory the bit of foil is then cleared of blood, which flakes off easily. One mgm. of dried blood and 200 mgm. of distilled water are weighed out and mixed. This gives an exact dilution of 1 to 200 by weight; 1 to 50 dilution by volume. A hanging drop of the dilution is inoculated with the bouillon culture and examined. The time limit is two hours.

It is essential for the success of the reaction that a pure culture of the typhoid bacillus should be employed. The most suitable culture for diagnostic work is that which is the most actively motile. It is true, however, that many observers recommend the attenuated cultures. They hold that an actively motile culture is too sensitive, and may cause clumping even with normal serum. If a fresh culture is kept at room temperature and transplanted every two or three days the culture maintains its motility and sensitiveness for a long period. The incubator bouillon cultures of twelve hours' growth are probably the most available. Johnston, whose experience is worth following, thinks the motility must not be excessive. He reduces the motility of the bacilli by transplanting his agar cultures once a month, growing them at room temperature. The bacilli from this culture, grown for twenty-four hours on bouillon, show a slight gliding motion, which differs from the darting motion seen in an active culture. The bouillon, Johnston holds, should be slightly acid, contrary to the general rule, which states that it should be neutral. It is quite necessary that the bouillon culture should be young—that is, twelve to twenty-four hours' duration in the incubator or two days at room temperature. When a culture is made under these circumstances, before it is used it should be free from sediment and only slightly turbid. It should also be free from any spontaneous clumping and from non-motile or sluggish forms.

Value. The question may well be asked, What is the value of the serum reaction? Let us answer by referring to typhoid fever chiefly. When it is recalled that this reaction takes place in about 98 per cent. of all cases of typhoid fever, it can readily be seen what a constant phenomenon it is in the course of continued fever. As a symp-

tom, therefore, it is one of the most constant. Its presence, however, cannot be determined in a large number of cases before the eighth or tenth day. It has been found as early as the third day, and, on the other hand, may be absent until after convalescence has set in. In a large majority of cases the reaction appears, however, before the fourteenth day. In a few instances, as Widal pointed out, the reaction disappears as soon as the temperature remains normal. In other instances it may continue several months, and in rare cases has been found as long as ten years after the disease.

It is thus seen that the presence of the serum reaction is a valuable diagnostic symptom of some diseases, and notably of typhoid fever. Its absence, however, does not disprove the presence of the disease. Sometimes the blood of a patient ill with some other disease, who has previously had typhoid fever, may give a positive reaction, and thus lead to a false diagnosis. Absence of reaction in a supposed case of typhoid fever implies, in 98 per cent. of all cases, that this infection is not present, providing, of course, that the technique is correct and that repeated examinations have been made. In the following diseases the serum diagnosis is employed: (1) Glanders; (2) Malta fever; (3) yellow fever; (4) cholera; (5) relapsing fever; (6) typhoid fever.

4. Leucocytosis. The presence of leucocytosis is characteristic of many infections, and, on the other hand, is against not a few of the most common of the infectious disorders. Accurate study of the number of white cells has led to fairly definite conclusions as to the diagnostic value of their increase or their diminution. The method of determining the number is described in the chapter on Diseases of the Blood, which may be referred to in order that the student may also learn the circumstances under which leucocytosis occurs physiologically. Pathologically we find inflammatory leucocytosis or the leucocytosis of infectious disease occurring with such frequency as to be diagnostic. A classification of the degree can be roughly made only. 1. In Asiatic cholera, relapsing fever, scarlet fever, diphtheria, syphilis, and erysipelas, leucocytosis occurs to a *moderate degree*. 2. In pneumonia, smallpox in the stage of suppuration, septicæmia, actinomycosis, trichinosis, glanders, beri-beri, acute rheumatism, cerebro-spinal meningitis, and gonorrhœa it is also found, but more *constant* and more *marked*. 3. In all pyogenic infections, especially abscesses, in inflammations of serous membranes and in gangrenous inflammation usually due to streptococci or staphylococci infection, leucocytosis is *great*.

The *significance* of leucocytosis depends not alone upon the number of the white cells, but also upon their rise and fall in the course of the disease. The amount of local inflammation attending the infection is not a measure of the amount of leucocytosis. Moreover, the degree of fever does not affect the leucocytosis. Fever may occur without increase in the white cells, and the opposite condition may also obtain. When leucocytosis and fever are due to the same infection they may rise and fall together, as we often see in cases of pneumonia.

Absence of Leucocytosis. While the presence of leucocytosis is significant of various infections, its absence is likewise of great significance. Hence if there is no leucocytosis it is possible either typhoid fever,

malaria, influenza, measles, rötheln, or tuberculosis are present. The blood-count can in this manner be employed to distinguish typhoid fever, in which there is an absence of leucocytosis from a pyogenic infection, as appendicitis in which the other signs and symptoms may be quite similar. Pneumonia, on the other hand—an infection characterized by great leucocytosis—may in this manner be distinguished from tuberculosis, in which there is an absence of leucocytosis.

When leucocytosis occurs in the course of any disease in which it is normally absent it is an indication of a complication. In typhoid fever it is an indication of intestinal perforation and peritonitis, because of a mixed infection. On the other hand, a fall of leucocytes in a disease in which they are increased is suggestive of localization of the infection, as the "walling off" of the abscess in appendicitis. Such fall in pneumonia is of grave prognostic omen.

5. For direct bacteriological examination of the blood culture methods are resorted to. After the skin has been cleansed and made aseptic either a considerable portion of blood is withdrawn from a vein with a sterilized hypodermatic needle or blood is directly drawn with the instrument described by Ewing. After the blood is thus removed it is transferred to the various media, and its further treatment is carried on in accordance with bacteriological methods. (See Cultivation of Micro-organisms.)

Examination of Pathological Secretions and Excretions.

Microscopical examination, with and without staining, and *culture methods* are employed, as detailed in the sections to follow:

In *nasal discharges* the bacillus of diphtheria, of glanders, of tuberculosis, and of the pneumococci, as well as pyogenic micro-organisms, are found.

In the *mouth* the micro-organisms peculiar to that cavity and the micro-organisms of actinomycosis may be found.

In the *fauces* and *pharynx* the bacillus of diphtheria and pyogenic micro-organisms are discovered.

The *sputa* (see Disease of Lungs) yield the tubercle bacilli, the pneumococcus, the bacillus of influenza, and actinomycosis.

The *fæces* (see Disease of Intestines) are examined for the bacillus coli communis, the spirillum of cholera Asiatica, bacillus typhosus, and tubercle bacillus.

The *urine*. Pyogenic micro-organisms, tubercle bacillus, typhoid bacillus, the pneumococcus, and gonococcus are found in the urine. They are secured by cover-slip preparations of the pus, or by culture methods, as described in the section devoted to Diseases of the Kidneys.

Examination of the Products of Infectious Inflammation — Material Secured by Exploratory Puncture.

Material removed by exploratory operation or puncture may be serous, bloody, or purulent (See Chapter XXI.) It must be examined bacteriologically, microscopically, by culture methods, and by inoculation. Serous fluids are not usually productive of bacteria when exam-

ined unless treated by sedimentation, and even then it is often necessary to inoculate.

The most important pathological product is *pus*. Fresh and stained preparations are examined, and cultures are taken. We may find only one, sometimes two at the same time, of the following micro-organisms: 1. Staphylococcus pyogenes aureus. 2. Staphylococcus pyogenes albus. 3. Staphylococcus epidermidis albus (Welch). 4. Streptococcus pyogenes. 5. The tubercle bacillus. 6. The bacillus of syphilis. 7. Actinomycosis. 8. The bacillus of glanders. 9. The bacillus of anthrax. 10. The bacillus of leprosy. 11. The bacillus of tetanus. 12. The bacillus of influenza. (See Sputum.) 13. The micrococcus lanceolatus. 14. The bacillus coli communis. 15. The gonococcus.

Fresh pus may be examined, but the stained is more satisfactory. Staining by the method of Gram is the best, and is as follows: After a cover-glass has been prepared and placed in Koch-Ehrlich's solution of gentian-violet and aniline water, it is put into a solution of iodine and iodide of potassium for two or three minutes. A dull red-brown color is produced. It is then rinsed in absolute alcohol for some time. The micro-organisms are stained dark blue. The iodide of potassium solution is: Iodine, 1 part; iodide of potassium, 2 parts; distilled water, 300 parts. By this method the various forms of micro-organisms just indicated are readily brought out.

Methods of Staining Blood, Pus and Discharges. It is well to consider these collectively. Many have been devised, but those of clinical value are the following:

1. Aqueous solutions of basic anilines.
2. Löffler's alkaline methyl-blue.
3. Koch-Ehrlich's aniline water solutions.
4. Ziehl's carbol-fuchsin.
5. Löffler's method of staining flagella.
6. Gram's method.
7. Friedländer's method.
8. Günther's method.

1. Basic anilines. Aqueous solutions of the basic aniline colors — fuchsin, gentian-violet, and methyl-blue—are used of such strength that they can be seen clearly through an ordinary test-tube. They may be kept on hand in bottles with pipettes, or made from concentrated alcoholic solutions as needed. They are used by simply dropping a few drops on the cover-glass preparation, which is held with the forceps, allowing it to remain about thirty seconds, and carefully washing off in water. It is placed on a slide, *bacteria down*, and the excess of water removed with blotting-paper.

2. Löffler's alkaline methyl-blue solution. Certain bacteria take a stain more readily when an alkali has been added. The formula is as follows:

Concentrated alcoholic solution methyl-blue . . .	30 c.c.
Caustic potash, 1 : 10,000	100 "

It is used in the same way as the simple solutions.

3. Koch-Ehrlich's aniline water solutions. Add to 100 c.c. of distilled water, aniline oil, drop by drop, thoroughly shaking after each

drop until it becomes opaque. Then filter. Add 10 c.c. absolute alcohol and 11 c.c. of a concentrated alcoholic solution of either fuchsin, methyl-blue, or gentian-violet.

4. Ziehl's carbol-fuchsin solution.

Distilled water	100 c.c.
Carbolic acid	5 gm.
Alcohol	10 c.c.
Fuchsin	1 gm.

The use of these various stains will be described in the description of the different bacteria.

5. Löffler's solution for flagella.

Tannic acid, 20 per cent.	10 c.c.
Cold saturated solution ferric phosphate	5 "
Saturated solution fuchsin	1 "

A few drops of this solution are placed on the cover-glass containing the bacteria and very gently heated until they begin to steam, and then the cover-glass is washed off in water. The preparation is then stained with aniline water fuchsin. Different bacteria require different reactions, and so a few drops of an acid or alkaline solution are recommended to be added as the case requires. As a rule, however, the results obtained when neither acids nor alkalies are added are just as satisfactory as those following such additions.

6. Gram's method consists in staining with a Koch-Ehrlich solution of gentian-violet for twenty to thirty minutes, and then decolorizing in

Iodine	1 gm.
Potassium iodide	2 "
Distilled water	300 c.c.

After remaining in this for five minutes the preparations are rinsed in alcohol, and the process repeated until the violet color has disappeared.

For Friedländer's and Günther's methods, see Sputum.

To detect *spores* of bacilli double staining may be employed. The preparation is first stained in a hot Ziehl-Neelsen fuchsin solution, then decolorized with alcohol containing from 0.2 to 0.3 per cent. hydrochloric acid. When stained again with methylene-blue the spores appear red and the bacilli blue.

The "hanging drop." By the examination of colonies in the *hanging drop* we learn of the movement of the micro-organism. Place a drop of physiological salt solution on a cover-slip, and add a tiny portion of colony on platinum wire ; place the slip, drop down, on a glass slide, in the centre of which is a depression or hollow. Fix the slip by applying a thin layer of vaseline around the margin of the depression. Care must be taken in focusing that the lens does not break the glass, which may be readily done because of its transparency. The bacteria are seen in motion ; on account of the motion their position is constantly altered. This motion must not be mistaken for the Brownian movement of suspended articles, which is vibratory from molecular tremor.

Cultivation of Micro-organisms. The object is to isolate the pathogenic organism from all other organisms and to exclude organ-

isms that may be introduced from without by unclean instruments or other means. *Pure* cultures are thus obtained.

Culture-media. Experience has taught us that various forms of bacteria require different pabulum, and that various nutrient media are required for the isolation of different micro-organisms. As to the bacteria hereafter noted, we are familiar with the proper soil for their growth. The media used for bacteria of clinical importance are: a freshly steamed potato, gelatin, bouillon, agar-agar, milk, and blood-serum. They are prepared or mixed in various ways, and other things may be added, as a solution of litmus, to determine the reaction of the bacterial products.

Bouillon. Lean beef, 500 gm., soaked in one litre of water for twenty-four hours in an ice-chest; strain through a coarse towel and press until a litre of fluid is obtained. Add 10 gm. of dried peptone and 5 gm. of salt. Then neutralize with a normal solution (4 per cent.) of caustic soda. Boil till albumin is coagulated. Filter and sterilize.

Nutrient Gelatin. Make bouillon as above (except neutralizing) and add 10 to 12 per cent. of gelatin, and neutralize after dissolving it by heat. Filter.

If not perfectly transparent, clarify by heating to 60° or 70° C., add the whites of two eggs beaten up with 50 c.c. of water; mix thoroughly and boil until albumin coagulates; then filter. Sterilize and keep in flasks or tubes.

Nutrient Agar. Prepare bouillon complete; add finely chopped agar, 1 to 1.5 per cent. Place in a porcelain-lined iron vessel, mark level of fluid, add 250 c.c. of water and boil slowly, with occasional stirring, for three or four hours. Keep the fluid up to the mark by adding water. Take the vessel from the fire and set in cold water. Stir until cooled at 68° to 70° C.; add the whites of two eggs beaten up in 50 c.c. of water. Mix carefully and boil for half an hour, keeping the fluid up to the level. Filter.

Sometimes 5 to 7 per cent. of glycerin is added.

Potatoes. Select old potatoes; scrub under water-faucet with stiff brush; cut out eyes and defects. Then place in 1 : 1000 $HgCl_2$ for twenty minutes. Then place in steam sterilizer and steam forty-five minutes. Leave them in and steam fifteen or twenty minutes each day for three days. Cut with knife sterilized in flame and lay with cut surface upward in a sterilized covered dish.

Another way of preparing potatoes is to cut cylinders with a cork borer of such size as to fit loosely in a test-tube. A slanting surface is then cut from the junction of the first and second thirds of the cylinders diagonally to the opposite edge. These are left in running water over night, then placed in test-tubes with a cotton plug and steamed for forty-five minutes. On the second and third days they are steamed fifteen to twenty minutes.

Milk. It should be sterilized in a steam sterilizer by the fractional method. It is a good soil for the tubercle bacillus (Abbott).

Blood-serum. The original method of preparing blood-serum, as recommended by Koch (given in the text-books on Bacteriology), has, in this country at least, almost entirely given place to the method of

Councilman and Mallory, the popularity of which is due to the following advantages: By it the serum is more quickly and easily prepared; rigid precautions against contamination during collection of serum are not necessary, and the resulting medium, while not transparent or even translucent (points aimed at in the original method), fully meets all the requirements.

The special points in the method are: the serum is decanted into test-tubes as soon as obtained; it is then firmly coagulated in a slanting position in the *dry-air* sterilizer at from 80° to 90° C.; it is then sterilized in the steam sterilizer at 100° C. on three successive days, as in the case of other culture-media. It may then be protected against evaporation by sterilized rubber caps or sterilized corks, and set aside until needed.

Unless the coagulation in the dry sterilizer be *complete*, the surface of the serum will be found to be lacerated by bubbles and cavities after it has been subjected to the steam sterilization. A similar formation of cavities over the surface of the serum will occur if the temperature of the *hot-air* sterilizer, in which it is solidified, is allowed to get above 90° C., or if it be elevated to this point too quickly.

It is of no special advantage to have the serum clear, as the admixture of blood-coloring matter does not affect its nutritive properties.

Löffler's blood-serum mixture:

Neutral meat infusion bouillon (see Bouillon) . . .	1 part
Grape-sugar	1 per cent.
Blood-serum	3 parts.

Tube-cultures and Plate-cultures. The plate method was introduced by Koch for the purpose of isolating individual species of bacteria from mixtures. It may be practised either with gelatin or agar-agar. Three tubes previously filled with the culture-media are liquefied by warming in a water-bath, then cooled to the lowest point at which the medium remains fluid. One of the tubes is then held in the left hand. A sterilized looped platinum wire inserted in a glass handle is taken in the other hand, passed through a flame, and cooled for a few seconds. With this a bit of the material to be examined is taken up, the cotton plug is removed from the tube with the free fingers, and the wire inserted into the medium. By rolling the tube it is thoroughly mixed. Then a second tube is inoculated with three loopfuls from the first, and a third with three loopfuls from the second. Plates have been previously sterilized and placed in covered dishes also carefully sterilized. The plates are levelled and the contents of the tubes poured upon their surface. Then they are cooled over ice-water until the medium becomes solid, when they are placed in a proper temperature for development. In this way the bacteria are sufficiently diluted to form distinct colonies from which pure cultures may be obtained.

A convenient modification of the method is the use of *Petri's plates*, which are flat, round dishes with covers, the bottom of the dish serving as the plate.

Another modification (*Esmarch's tubes*) is the use of tubes with a small quantity (5 c.c.) of the medium. By rolling the tube in the

fingers the sides are coated with the media. They are then rolled on ice, so that the medium solidifies in a thin layer about its walls.

Smear-cultures and Stab-cultures. When the bacteria has been isolated by one of these methods pure smear-cultures or stab-cultures must be made from them. A tube of the proper culture-medium is taken in the left hand, a bit of pure colony taken up on a sterilized straight platinum needle, the cotton plug removed as above, and the needle thrust straight into the medium for a stab-culture, or rubbed over a slanting surface of media for a smear-culture. The plug is immediately inserted and the tubes transferred to the incubator.

When pure cultures have been obtained the species are recognized by their *mode of growth* and behavior in different culture-media, the *reaction* produced by their growth, and their appearance under the microscope when stained and unstained.

When nutrient media are inoculated they must be kept at a favorable *temperature*. This will be detailed when each micro-organism is discussed, as a number of pathogenic bacteria require a definite and continuous temperature.

The primary inoculation will often yield numerous colonies, the nature of the bacteria comprising which must be determined by their morphology and biological characteristics. It is frequently necessary to repeat the process of plating with several of the colonies obtained on the original plates, otherwise one cannot always be certain that the organism for which he is seeking has been isolated in *pure* culture.

Microscopical Examination of Colonies. Just here may be stated the methods employed for the study of the morphology of the colonies secured by plate and other means of cultivation.

Cover-glass preparations are made as follows: Place on the cover-glass a small drop of distilled water. With a platinum needle take up the smallest possible quantity of the colony to be examined, mix it with the drop and spread over the surface of glass. Dry under cover or by holding *with fingers* over a flame, the layer of bacteria being away from the flame. When dry pass it with forceps three times through the gas or alcohol flame to "fix" the albumin. It is then ready for staining.

Inoculation of Animals.

Another method of determining the pathogenic character of morbid material, as sputum, pus, or exudation, is by inoculating animals with a pure culture. This is done either by feeding or injection, as subcutaneous or intravenous, into the peritoneal or pleural cavity, and, in rare instances, into the anterior chamber of the eye or into the cranial cavity.

As animals are subject to only a few of the microbic diseases of man, many experiments must often be made before a susceptible animal is found, and no conclusions can be reached as to the pathological power of a micro-organism until this point has been determined. The clinical course of the artificial disease must be observed to fulfil the diagnosis, and the difficulty of reproducing faithfully in animals

the clinical manifestations seen in man is often one of the gravest obstacles to this method of diagnosis.

Examination of the animal is made as soon as possible after death. The autopsy is made with antiseptic precautions. After the skin is removed only sterilized instruments are to be used. The macroscopical appearances and the mode and progress of infection are noted for the purpose of aiding in the diagnosis. When the organs are exposed, material for culture is first obtained by inserting a platinum needle through a small puncture in the capsule. Afterward cover-glasses may be prepared for immediate examination. Blood is taken from one of the cavities of the heart. After the autopsy all remains are to be burned and all instruments carefully sterilized.

Special Bacteriological Diagnosis. The following points must be investigated in order to determine the specific nature of the micro-organism which is supposed to be the productive agency of the disease in question, viz.: The *form*—micrococci, bacilli, spirilla, polymorphous; *relation to oxygen*—aërobic, facultative anaërobic, strict anaërobic; *growth in nutrient gelatine*—liquefy, do not liquefy, do not grow at "room temperature;" *growth on potato; growth on milk*—coagulate milk, do not coagulate, etc.; *color of growth*—chromogenic, non-chromogenic; *spore-formation; movement; pathogenic power.*

NOTE. For further information concerning technique the student must refer to the work of Abbott on the "Principles of Bacteriology" and to Sternberg's "Manual of Bacteriology" for an exhaustive account of the technique, and the morphological and bacteriological characteristics of all bacteria, pathogenic and non-pathogenic.

CHAPTER XVIII.

THE DATA OBTAINED BY OBSERVATION—(*Continued*).

FEVER. THE INFECTIOUS DISEASES.

Infections Not Recognized by Bacteriological or Blood Examinations.

This group includes most of the eruptive fevers which are contagious and epidemic. Their recognition must be based on data of the *social history*, the duration of the period of incubation, and upon the mode of onset and course of the respective infection. They are: *typhus fever, smallpox, varicella, scarlet fever, measles, rubella, mumps or epidemic parotitis, glandular fever, whooping-cough, rheumatic fever, dengue, beri-beri, syphilis, Weil's disease, milk-sickness, miliary fever, foot and mouth disease, hydrophobia.*

It must be remembered that other infections are not always recognized by bacteriological examinations, although if such examination gives a positive result the diagnosis is final. The following data should be sought for in the diagnosis of any infection, but especially in case of failure of the bacteriological methods; or, if such methods are successful, as a control in the diagnosis.

Social History. In the diagnosis of the infectious diseases valuable data are obtained from the *social history.*

Age. Thus early age is the period of life in which the eruptive fevers are more common; adolescence, that of typhoid fever and tuberculosis. In the sex, however, we find but little of diagnostic value.

Exposure. Bearing in mind the possible cause of the disease, we inquire for all those circumstances which contribute to the origin of the infection. Hence we inquire into the food, the character of the water, and other material ingested. We inquire if an opportunity for inhalation of infectious material could have occurred, as dried sputum from a case of tuberculosis, or if exposure to the patient was possible. We learn the hygienic conditions and place of residence (malarial districts, the tropics). The occupation—wool-sorter, hostler, farrier—points to the nature of the infection. In short, we inquire if the patient has been exposed to any infection.

The Presence of an Epidemic. We inquire if an epidemic of the suspected disease prevails, and if the patient has been exposed to the contagion. We consider etiological factors, as the season in which the infection prevails.

History of Previous Infection. In the history of *previous diseases* we inquire if the patient has had previous infections. Some contagious disorders rarely take place a second time, as scarlet fever, or measles.

Hence, if the patient has never had them—is not immune—his susceptibility is of diagnostic importance. Other infections predispose to subsequent attacks, as pneumonia or erysipelas, hence the occurrence of a previous attack is important.

Having secured the data above indicated, we proceed to an examination of the patient, noting the length of time since he had been exposed to contagion, the mode of onset of the symptoms, and the subjective and objective symptoms at the time of the examination. These separate data will be discussed in the account of the various infections included in this chapter.

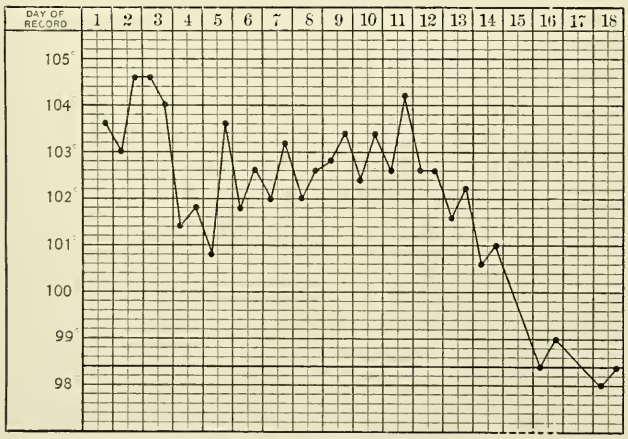
THE ERUPTIVE FEVERS.

The following infections are characterized by a specific eruption which permit them to be given the above title. They are also members of Class I., spoken of in Chapter XVII. The fever, in a measure, runs a definite clinical course, and is of diagnostic significance. The infection bears such definite relation to the eruption, however, that the diagnosis is usually based upon the latter.

Typhus Fever.

In this infection the *temperature* rises rapidly, reaching to 104° or 105° by the end of the second or third day. It is an acute contagious fever, occasionally occurring sporadically, and often becoming epidemic in the presence of destitution, filth, over-crowding, and bad ventilation.

FIG. 48.



Typhus fever—typical. (DOTY.)

It is characterized by abrupt onset with chill or with chilliness, a *rapid rise* of temperature, lassitude, headache, and pains in the back and limbs. On the fourth or fifth day a peculiar spotted eruption appears, which at first is macular and subsequently petechial. It is further characterized by adynamia or ataxia, low muttering delirium, a suf-

fused, heavy, drunken expression of countenance, by the absence of local disease, and by a crisis which occurs on or about the fourteenth day.

Typhus fever is variously known as *ship fever, jail fever, camp fever.*

The period of *incubation* is usually about twelve days; it may be five or eight days, or even a shorter time, depending upon the virulence of the poison and the susceptibility of the patient. Malaise may precede by a day or two the onset of the disease.

Invasion is characterized by headache, faintness, vertigo, chilliness, or a distinct rigor, pains in the back and thighs, loss of appetite, nausea, constipation, and extreme weakness. The prostration is sometimes so great as to compel the patient to go to bed at once. The *pulse* is frequent, 100 or 140, and in grave cases shows a marked tendency to become small, soft, and feeble. The patient is restless and sleepless, and is annoyed by tinnitus. The *expression* of the flushed face is listless and dull.

About the fourth or fifth day the typhus *eruption* begins to appear. It consists at first of dull red spots of irregular size and shape. They are most numerous on the covered parts. Moore[1] says they are detected first near the axillæ and on the wrists, then on the sides of the abdomen, afterward on the chest, back, shoulders, thighs, and arms. The skin is also mottled by another crop of maculæ under the skin ("mulberry rash").

When the disease is fully developed the face is flushed, the conjunctivæ red, the pupils contracted, so as to resemble pin-holes ("ferrety eye"), the tongue dry and brown, the teeth covered with sordes, the skin dry, hot, and stinging to the touch. The patient lies upon his back oblivious to all his surroundings. Headache has given place to delirium, which may be wild and fierce, but is more commonly low and muttering. There are marked ataxic symptoms—subsultus tendinum, tremors, picking at the bedclothes. Incontinence of urine and fæces sometimes occurs. The breathing is frequent, shallow, and noisy, and the pulse frequent, soft, and feeble. The macular rash now becomes petechial. The patient is in a typical "typhoid state." The stupor may gradually clear up, or, on the other hand, deepen into coma; or the patient may die from progressive weakening of the heart, with or without pulmonary complications.

In the majority of favorable cases, on or about the fourteenth day, the first sign of recovery is a sound sleep, from which the patient awakes refreshed and rational. The temperature falls with great rapidity, the pulse and temperature improve; a typical crisis has occurred.

Certain *objective phenomena* of the disease require special mention. The eruption is more copious in severe than in mild cases. A dull and livid color is a grave sign. Purpura and hemorrhages are sometimes met with in bad cases. The eruption does not occur in successive crops.

The patient seems to be surrounded by a vapor of a pungent, musty *odor* which is peculiar.

[1] Eruptive and Continued Fevers, by J. W. Moore, Dublin, 1892.

The *heart* early shows the effect of the poison. The impulse is diminished, and the first sound is less distinct. In grave cases, with threatening heart-failure, the sounds are feeble and distant, the impulse imperceptible.

The *pulse* is usually very much more frequent than normal, but may be abnormally slow (50 and even 30 per minute); this is sometimes a bad sign.

The *weak heart* and prostrate position of the patient favor congestion, with œdema of the lungs. This condition is common.

Digestive symptoms have already been referred to. Vomiting, tympanites, and diarrhœa are rare, and still more so is intestinal hemorrhage.

The *urine* is scanty and high-colored. Slight albuminuria is common, and a few casts are found, but distinct nephritis is unusual. Convulsions, when they occur after the first week, are almost always uræmic and almost invariably fatal. They may be due to retention of the urine, as recorded by Stokes and Corrigan.

The *duration* of the disease is from six to fifteen days; the average period is twelve to fourteen days. An abortive form is met with in some epidemics, the disease being of a mild type and subsiding at the end of a week. In some cases so large a dose of the poison is absorbed by the patient that he is stricken down in a few hours or a few days. To this form the name "blasting typhus" has been appropriately given. The most important complications are hyperpyrexia, laryngitis, bronchitis, and congestion of the lungs, extreme ataxia or profound adynamia, nephritis, heart-failure, and parotitis, or other inflammatory glandular swellings.

Laryngitis with œdema is a very rare but very dangerous complication.

Diagnosis. *Cerebro-spinal fever* is distinguished from typhus fever by greater intensity of the headache, by retraction of the head and hyperæsthesia, by greater liability to vomiting, and by the absence of the macular petechial eruption and the drunken, besotted aspect of typhus fever. In cerebro-spinal fever the patient suffers with photophobia, and is liable to local palsies of the eye-muscles (strabismus) and to general convulsions. Convulsions do not occur in typhus except from a complicating nephritis or retention of urine.

Uræmia is distinguished from typhus by the preceding history, by the absence of high temperature, and by the presence of œdema of the face or extremities, a history of vomiting or diarrhœa preceding the stupor. The condition of the urine and the absence of eruption are the final tests.

Pneumonia is distinguished by the frequent respiration and relatively slower pulse, and by the local physical signs and absence of eruption.

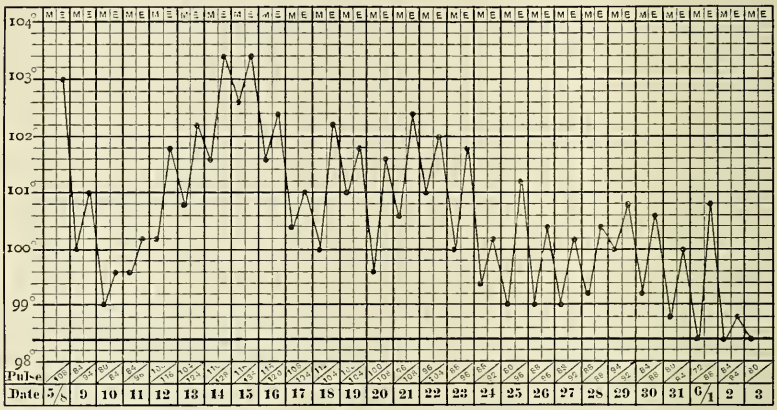
Typhoid fever is distinguished by its slow onset and marked abdominal symptoms. The eruption of typhus is petechial and comes out on the fourth or fifth day; that of typhoid fever consists of rose-spots and appears on the seventh or eighth day. In typhus fever the severe initial chill, the sudden onset, the greater prostration, and

the earlier appearance of cerebral symptoms are helpful in distinguishing it from typhoid fever.

Variola.

The temperature in variola, or smallpox, pursues a definite course, which renders it of value in the diagnosis. Its sudden rise to an unusual height without local inflammation but with severe backache is significant. Its fall with the appearance of the eruption, followed in two or three days by a secondary rise, is very characteristic.

Fig. 49.



Temperature in smallpox. Adult; mild case.

Variola, or smallpox, is a specific infectious and contagious fever, beginning abruptly with chill, high temperature, headache, vomiting, sweating, and intense pain in the back. On the second or third day of the disease a characteristic shot-like, papular eruption appears, the papules rapidly developing first into vesicles and then into pustules; with the appearance of the rash the temperature falls, but rises again toward the end of the week in the pustular stage (fever of maturation or suppuration). The contents of the pustules are discharged, crusts form and are cast off about the eighteenth day. The disease may be accompanied by a number of complications, particularly hemorrhages into the skin (purpuric smallpox) and from the mucous membranes (hemorrhagic smallpox), both forms being popularly called black smallpox. For convenience of description the disease may be divided into four stages: (1) Incubation, (2) invasion, (3) eruption, (4) desquamation.

Incubation. This stage lasts from ten to fourteen days, and is usually unaccompanied by any symptoms except, toward its close, by malaise.

Invasion. The invasion is abrupt, and is marked by chilliness or a distinct rigor, headache, severe pain in the lumbar region, and sometimes delirium or convulsions, especially in children. The most promi-

PLATE IV.

Variola on the Seventh Day of Eruption. Confluent on Face and Semi-confluent on Extremities.

nent symptoms are the excruciating headache and backache. The temperature usually rises rapidly to 104° F. or higher in the first twenty-four or forty-eight hours. (See Fig. 49.) Headache and backache continue; there are pain in the epigastrium, a coated tongue, loss of appetite, nausea or vomiting, constipation, and copious perspiration. Prostration is extreme. Erythematous eruptions are not uncommon, especially on the inner surfaces of the legs and thighs. Petechiæ are found in Simon's triangle, the base of which is at the umbilicus and apex at the knees.

The stage of *invasion* lasts generally three days; but it may be shortened to two in very severe cases or lengthened to four in very mild ones, and in complicated and hemorrhagic cases it merges into the stage of eruption. (See Plate IV.)

Eruption. The characteristic eruption of smallpox appears first as minute specks resembling flea bites. These in two or three days develop into small papules which feel like shot under the skin. In a day or two more the papules become vesicles, at first containing a clear fluid, which, however, rapidly becomes turbid; they are umbili-

FIG. 50.

Discrete variola on the sixth day of eruption. (WELCH.)

cated. In the course of another day or two the vesicles have become pustules and are globular in shape. The period of ripening or maturation, when pustulation is at its height, lasts about three days; it is characterized by a marked secondary fever, the temperature rising as high as, or higher than, in the onset of the disease. The pustules now begin to dry up (desiccation) and form dry scales or scabs, which are cast off toward the end of the third week of the disease (eighteenth

day); when the pustules have been deep enough to involve the true skin, characteristic scars, called pits, are left.

The *eruption* appears on the forehead, along the margin of the hair, and in the scalp, then over the rest of the face, especially about the nose and lips, subsequently progressing over the rest of the body from above downward. The eruption is most abundant upon the face and hands, often being confluent here when discrete elsewhere. The face may appear horribly swollen, bloated, and disfigured, and both face and hands are extremely painful from the great distention and the pustules, which are really small dermal abscesses.

Varieties. Three varieties of variola, depending upon the number and disposition of the pocks and upon the presence of complications, are recognized: (1) Discrete; (2) confluent; (3) malignant.

In DISCRETE variola the pocks are not numerous, and are separated from each other by intervening healthy skin.

In CONFLUENT smallpox the pustules are close-set, occupy almost the whole body, and coalesce, so that the face looks as though covered with a black, rough mask; the mucous membranes are also covered. The symptoms of the invasion are intensified, and the eruption may appear before the third day. Patients are liable to suffer with profuse salivation, uncontrollable vomiting or diarrhœa (especially in children), and with delirium, which is often violent and destructive. The face is dreadfully swollen and the eyelids may slough; the feet and limbs also may be swollen and painful. There may also be severe bronchitis and pneumonia, abscesses, extensive sloughing, and a pyæmic condition.

MALIGNANT, or BLACK, SMALLPOX is a form in which the blood is so altered that hemorrhages into the skin or from the mucous membranes occur. In the former case there are petechiæ and ecchymoses upon the skin; in the latter more or less profuse hemorrhages occur from the womb, kidney, bowels, lungs, and stomach. The mind of the patient remains clear and he is conscious of his peril. The eruption is delayed or does not occur at all.

Varioloid is a mild form of smallpox occurring in a person protected, but not completely, by previous vaccination, or in a person who, from other causes, does not possess the average susceptibility. It is characterized, apart from its mildness, by great irregularity in the development of the symptoms. The initial symptoms, as a rule, are as severe as in ordinary smallpox. Prodromal eruptions, especially the erythematous, are very common. The eruption may appear first on the face, or on the chest and trunk first, and later upon the face. The fever subsides with its appearance. The eruption passes from the papular to the vesicular stage, as in ordinary smallpox; but here the process, as a rule, ceases, the vesicle drying up on the fifth or sixth day of the eruption. If pustules form they do not reach their full development. The eruption is always discrete. There is usually no secondary fever.

Diagnosis. When fully developed, smallpox will not be mistaken for any other disorder. In the initial stage, however, there may be doubt whether the disease will prove to be pneumonia, cerebro-spinal meningitis, or typhus. If the patient has been exposed to smallpox and is unprotected by vaccination, and he is suddenly seized with a

chill, high temperature, and excruciating pain in the lumbar region, there is great probability in favor of smallpox. If the patient has complained of headache, pains in the ankles and other joints, and is seized with a severe rigor, explosive vomiting, and great weakness of the limbs, the chances favor meningitis in the absence of known exposure to smallpox. In *pneumonia*, vomiting, chill, and high temperature succeed each other, but excruciating backache is wanting, and, on the other hand, the respiration is increased out of proportion to the pulse, and even in this early stage there may be cough and roughening of the respiratory murmur on one side.

Typhus fever begins abruptly with chill and high temperature; but the eruption which comes out on the fourth or fifth day is first macular and later petechial, the temperature does not fall with the appearance of the eruption, the aspect of the patient is drunken and stuporous, the conjunctivæ are injected, the eye ferrety, the skin dry, hot, and biting to the touch (calor mordex).

In the papular stage of the eruption it may be mistaken for *measles;* but the red, swollen, blear-eyed, photophobic little patient with measles, with the characteristic coryza and obstinate cough, presents a very different appearance from that seen in variola. Moreover, the eruption of measles is relatively flat, smooth, and velvety; that of smallpox is acuminate, hard, and shot-like. The temperature in smallpox falls as the eruption appears; that of measles remains high and even increases. The papules of measles do not develop into vesicles.

In the vesicular stage varioloid may be mistaken for *chickenpox*. In the latter the eruption is practically vesicular from the start, occurs without prodromata, appears first upon the chest and neck, later upon the face and scalp, is usually very scanty, and rarely becomes umbilicated or pustular. There are, however, severe forms of varicella, in which fever, restlessness, and cough precede the appearance of the rash, which is copious, some of the vesicles being inflamed at the base, some umbilicated, and some with purulent contents. These cases are most common in scrofulous children whose hygienic surroundings are bad. In such cases the diagnosis cannot be made from the eruption. A consideration of the following points must decide: 1. History of exposure to varicella on the one hand or smallpox on the other. 2. The presence or absence of effective vaccination or of scars of antecedent varicella. 3. The age of the patient; smallpox occurs at all ages, varicella only in childhood. 4. The discovery among neighboring children of varicella or varioloid. 5. The rapid evolution of a varicella pock.

Varicella.

Varicella is one of the infections of childhood in which the febrile course is very mild. It is an acute specific infectious fever, occurring almost exclusively in children, and characterized by the appearance, in successive crops, of colorless or pearly vesicles, which dry up and are shed in from two to five days. It is attended with very little constitutional disturbance. A second attack is extremely rare.

The *incubation* is generally about two weeks, but may be one or

three weeks. In ordinary cases the first evidence of the invasion of the disease is the appearance of the eruption. In other cases, the severer ones, the child may be noticed for some hours or several days to be indisposed, complaining of loss of appetite, nausea, headache, and vague muscular pains. The fever is almost always moderate—100° to 101°.

The *eruption* consists first of hyperæmic macules, compared by Trousseau to the rose-rash of typhoid fever. These macules rapidly become first papules and then vesicles. The papules are not hard as in variola. They appear at first upon the chest, neck, face, and scalp, then upon the trunk and limbs. The development of the vesicles is so rapid that the eruption appears vesicular from the start. The vesicles vary in size from a pinhead to a small pea. They are very superficial, and usually

FIG. 51.

Varicella on the fifth day of eruption.

rest upon a base that is slightly or not at all hyperæmic. The contents are at first watery, but subsequently become pearly. The reaction of the fluid is alkaline. Distinct umbilication is rare, and pustulation still more rare, but both occur. The vesicles almost always dry up and form scabs, yellowish or brownish, which drop off, leaving a slightly reddened, sometimes depressed spot. Sometimes the vesicles are to be seen upon the buccal mucous membrane and upon the throat. While most of the eruption appears on the first or second day, fresh vesicles continue to appear for several days.

Desiccation usually occurs by the fourth or fifth day, and may be present in the first day or two. As the eruption appears in successive crops, often all stages, from the initial macule to the dried scales, can be seen in one case.

Usually the vesicles are widely scattered, a dozen or two over the entire body. They are most numerous upon the back, and may be as close together as in discrete variola.

In scrofulous and badly nourished children the lesions are more inflammatory and pustules are more common. If they are scratched, ulceration ensues. A gangrenous form has been described by Eustace Smith and others; the cases are apt to be fatal.

In ordinary cases during the eruption the child is rarely more than indisposed; complications are rare, and the prognosis most excellent. The physician is not often consulted except to have his opinion as to the diagnosis. (For the differential diagnosis from smallpox, see Variola.)

It is distinguished from *vesicular* and *pustular eczema* by the fever, the symmetrical grouping and discrete character of the lesions, the comparative absence of itching and burning, and its shorter course.

Impetigo is distinguished by the absence of fever, the more local character of the eruption, and the fact that it is generally pustular. It is more common upon the face and hands than is varicella.

Scarlatina.

In this eruptive fever the course of the temperature varies somewhat with the severity of the infection. In many instances fever would not be detected without the use of the thermometer. In others it may rise to a great height, and even be hyperpyretic. Its onset is sudden; it reaches its greatest height when the eruption is complete.

The *temperature* in *scarlet fever* usually conforms to a clearly defined type. The temperature increases gradually to the third or fourth day, when the acme is reached. It declines by lysis in a period of four days. A seven days' chart would be pyramidal in shape. In septic forms (scarlatina anginosa), with ulceration of the fauces, the fever continues and becomes remittent. In scarlatina maligna, hyperpyrexia is likely to ensue rapidly.

Scarlet fever is an acute, specific, contagious, and infectious fever, characterized by a sudden onset, with vomiting, sore-throat, and high fever, followed in twelve or twenty-four hours by a bright-red, punctiform eruption, by a very frequent pulse, by a desquamation which is often in large flakes, by a very variable degree of severity, and by a large number of complications and sequelæ, especially nephritis and inflammation of serous membranes.

Scarlet fever preferably affects children from one to five years of age. The liability to it diminishes after the tenth year; but it is very rare under the age of six months. Puerperal women are very susceptible to the poison, and the existence of open wounds favors infection. The disease occurs in epidemics at longer intervals than is true of measles. Cases are most numerous in the autumn and winter months. The peculiar poison is doubtless a living organism, but it has not been isolated as yet. It is very tenacious of life, being capable of infecting, through clothing in which it has been retained, months after the clothing absorbed the poison.

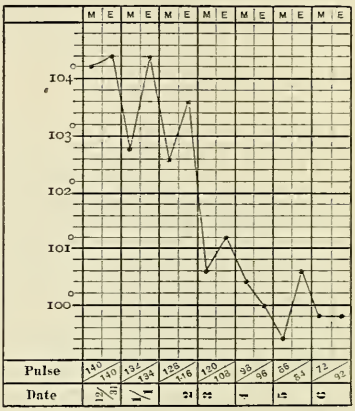
Few diseases vary so greatly in severity in different cases and in different epidemics. It may be the mildest or most malignant of diseases.

The period of *incubation* is remarkably short, generally from three to five days; but it may be a few hours, and, in exceptional cases, six days.

The *invasion* is abrupt. It is very common to be told that a child was apparently well on going to bed, but awoke in the middle of the

night, vomiting profusely and complaining of sore-throat. The child is found in the morning with a temperature of 103° or 104°, a pulse of 120 to 140, and a scarlatinal eruption beginning to show upon the neck and upper part of the chest. Close observation in such cases might have discovered that the child was feverish on going to bed, and that he had been somewhat chilly before that. Onset with decided chill, vomiting, and nervous symptoms indicate a severe case.

Fig. 52.



Scarlet fever. Mild attack; intense eruption.

The *subjective symptoms* of scarlatina are few; they consist usually of pain in swallowing, with stiffness of the neck-muscles, some headache, thirst, malaise, and a moderate amount of weakness. In the eruptive stage the skin itches, burns, and is frequently hyperæsthetic.

The *objective symptoms* and their order of succession are very characteristic. Vomiting is the rule, except in mild cases, and hence is of importance in diagnosis, especially in otherwise doubtful cases. The temperature is high at the onset, frequently 103° or 104°. It falls a degree or so in the morning; but the following evening, when the eruption is usually at its height, it rises to 104° or 105°, and then gradually falls to normal in the course of a week in ordinary cases. (Figs. 34 and 52.)

The *pulse-rate* is characteristically frequent, being 120 to 160 oftener than slower. This frequency is not an indication of danger.

The *blood* shows a leucocytosis, beginning on the first day and continuing through convalescence. A close relationship exists between the degree of leucocytosis and the rash. Suppurative complications tend to increase the number of white cells. The finely granular eosinophiles are greatly increased during the first few days. The mononuclear cells and lymphocytes are diminished at first, but after a short time their percentage increases.

The *throat* exhibits a uniform flush extending over pharynx, tonsils, soft palate, and sometimes forward on the hard palate, nearly to the

teeth. Sometimes dark-red points can be distinguished on the soft palate. The tonsils are inflamed and projected toward the median line from each side. Frequently the mouths of the follicles are blocked by a creamy-white exudate. It is not uncommon to find a severe follicular tonsillitis at the first visit.

The *tongue* is at first covered with a thick, creamy fur, through which enlarged red papillæ show. The enlarged papillæ look like small grains of red pepper sprinkled on the tongue. Sometimes the papillæ are elevated and have a button-like appearance. The symptoms appear very early in the disease, and may continue for three or four weeks. The coating soon disappears from the tip, leaving it bright red—the "strawberry tongue."

The *skin* is hot and dry. The characteristic eruption usually appears within twenty-four hours, often within six to eighteen hours, of the chilliness or vomiting which marks the onset. Sometimes it comes out very slowly, seeming to be just ready to appear, but not appearing in its full development for four or five days.

The intensity of the *eruption* varies from a scarcely perceptible erythema to the color of a boiled lobster. Usually its intensity varies with the severity of the disease. In ordinary cases the patient appears to be covered with a uniform red efflorescence; but a closer inspection shows that there are darker red spots between which the skin is more or less erythematous. It is first seen about the ears and neck, and spreads with great rapidity, covering the entire body in a day. It is most intense upon the trunk and flexor surfaces. Upon the extensor surfaces the punctate character is better seen. Pressure causes the redness to disappear, but it immediately reappears. Papular and vesicular forms of eruption are also seen. The *physiognomy* of the disease is peculiar. The circle about the eyes, nose, and lips remains pale, and in marked contrast with the rest of the fiery red face. Itching and burning are annoying symptoms at times. The eruption fades gradually, in ordinary cases disappearing, except when there is pressure or irritation toward the end of the week.

The eruption is succeeded by *desquamation*, which is extensive in proportion to the intensity of the eruption. The flakes are larger than in measles, and in severe cases the epidermis may come off in long strips. About the hands and feet this shedding is sometimes so great as to be compared to a glove. This stage may be protracted for several weeks, danger of infection lasting as long as desquamation continues.

The urine is at first scanty, high-colored, and febrile. Later, when desquamation is in progress, there is great liability to albuminuria as a complication.

Varieties. In addition to the ordinary form already described scarlatina exhibits many irregular forms. There may be only a sore-throat or follicular tonsillitis. If a rash is present, it is very faint, and hence easily overlooked. The diagnosis in such cases must be made from the fact of exposure to infection and from the appearance of the throat. The occurrence of vomiting is very important in the diagnosis, as it is rare in ordinary pharyngitis and tonsillitis. Often such cases escape

detection altogether, until possibly a dropsy from scarlatinal nephritis indicates their nature.

Severe diarrhœa may prevent the eruption from developing upon the skin. It appears upon the fauces, and the diagnosis is based upon this, the pulse and temperature, and the fact of exposure.

In *scarlatina anginosa* the strength of the poison is spent upon the throat. Pain is great and deglutition difficult. The tonsils are greatly swollen, so as almost to occlude the fauces, and their surfaces are covered with creamy exudate. The cervical glands are swollen, and there is a tense and brawny cellulitis. Sometimes the tonsils become gangrenous, and the cervical or submaxillary glands suppurate or become gangrenous, with resulting pyæmia and death. Suppuration may extend to the ears and maxillary sinuses. In this form, also, a false membrane is sometimes found upon the fauces—post-scarlatinal diphtheria. It is probably not due to the Klebs-Löffler bacillus, but to a streptococcus.

In *malignant* forms the attack is ushered in with chill, followed by hyperpyrexia, convulsions, marked ataxic symptoms, or stupor. The profound blood-disturbance is shown by the dusky hue of the eruption. Some patients lie in coma-vigil, others are very restless and delirious. Vomiting and diarrhœa are sometimes superadded. Patients may emerge from this condition and succumb later to a nephritis or to grave anginose symptoms ; but death in a few days is the rule. In rare cases the dose of poison is so enormous that death takes place in a few hours, without the appearance of any eruption.

Complications and Sequelæ. The severe local symptoms mentioned under the anginose variety, together with convulsions, hyperpyrexia, and ataxic symptoms, may properly be regarded as complications. Apart from these the most frequent are nephritis and endocarditis or pericarditis. Nephritis generally appears with the beginning of desquamation. It is nearly as frequent in mild as in severe cases, probably because the danger of exposure to cold is greater in the former, although the scarlatinal poison unquestionably has a selective affinity for the epithelium of the kidney. The symptoms do not differ from those of acute parenchymatous nephritis occurring under other circumstances. In some cases we have weakness, languor, slight fever, and prolonged convalescence ; in others, œdema, anuria, convulsions or coma from uræmia. Endocarditis is often preceded by tenderness and soreness of the muscles and joints—scarlatinal rheumatism.

Endocarditis and pericarditis develop in the course of the fever, giving rise to an increase or continuance of the fever, to local pain or dyspnœa, and to the usual physical signs.

Pleuritis and meningitis also may occur. Much more common complications are otitis, peripheral neuritis, and affections of the joints, grouped as scarlatinal rheumatism. Paralyses, peripheral and central in origin, are occasional sequels of the disease. Scarlatina is found also in association with other diseases.

Diagnosis. Sudden onset, rapid rise of temperature, persistent and causeless vomiting, and sore-throat lead one to suspect this affection. The characteristic eruption and its mode of evolution, the rapid pulse,

the peculiar tongue, the circle of pallor on the face, are characteristic of the eruptive stage. "The appearance of a punctate eruption in the axilla and in the groins, together with the congestion of the tonsils and a punctate eruption in the roof of the mouth, no matter whether there is any eruption anywhere else or not, are positive proofs of scarlet fever" (McCollom).

Unfortunately, all cases do not develop to the same degree, so that frequently we must wait for the period of desquamation; more unfortunately, for the occurrence of sequelæ, as acute nephritis, otitis, or adenitis.

Scarlet fever is distinguished from *measles* by the mode of onset, which is sudden, with chilliness, high temperature, vomiting, and sore-throat, and great rapidity of the pulse; whereas the onset in measles is gradual, with coryza, cough, moderate fever, perhaps looseness of the bowels, but no sore-throat. The eruption of scarlatina occurs on the first day, that of measles on the fourth; the former consists of dark-red spots with intervening erythematous skin, the whole looking at a distance like a uniform bright-red flush; the latter consists of raised, rounded, or flattened spots or blotches, velvety to the touch, and, upon the body and extremities, grouped in patches with crescentic outlines. The temperature in scarlatina subsides gradually after the rash has reached its height; that of measles increases until the eruption is complete, then subsides by crisis. The rash of scarlet fever persists for six or eight days; that of measles fades as soon as it is complete, on the fourth day. In the former, desquamation is in flakes or large strips; in the latter it is branny and nearly invisible. Scarlatina involves by preference the serous membranes and kidneys; measles the mucous membranes and lungs.

Scarlatina has to be differentiated from *pharyngitis*, *tonsillitis*, and digestive disturbances, attended with vomiting, high temperature, and occasionally erythematous eruptions.

In ordinary pharyngitis and tonsillitis the redness is more apt to be confined to the pharynx, tonsils, and arches of the soft palate; in scarlatina it extends as a flush over the soft and hard palate and buccal surfaces. In the former, high temperature, a very frequent pulse, and vomiting are unusual; in the latter they are the rule.

The glands of the neck also are more apt to be involved in the latter.

In *acute gastritis* there is usually a history pointing to indiscretion in eating, with constipation. The pulse is not so frequent as to suggest scarlatina, sore-throat is absent, and any erythema present lacks the characteristic dark-red points, and is not followed by desquamation.

The diagnosis from *rubella* is difficult at times. It differs from scarlatina in presenting mild catarrhal symptoms, sneezing, suffusion of the eyes, and cough, with a relatively fleeting eruption. The latter perhaps appears most frequently upon the back and chest. Often the eruption is the first thing noticed amiss with the child. It more commonly resembles the rash of measles than that of scarlatina, but when it resembles the latter most it is apt to be discrete and of a darker red. There may be a very intense rash without much constitutional disturbance, the temperature being lower and the pulse much slower than

would be expected in a scarlatina presenting the same appearance. Nausea may be present, but vomiting is very rare. The post-cervical and post-auricular glands are more commonly enlarged in rubella than in mild scarlatina, though this symptom is not invariable.

Diphtheria is distinguished by its gradual onset, patches of false membrane developing upon the fauces early. In anginose scarlet fever, with severe follicular tonsillitis, the differential diagnosis is essentially the same as between simple follicular tonsillitis and diphtheria (*q. v.*).

In addition, the pulse and temperature have a much higher range in scarlatina. The erythema of diphtheria is distinguished from the eruption of scarlatina by its fleeting character and the absence of desquamation.

Grave cases which begin with repeated vomiting, convulsions, delirium, and insomnia simulate *meningitis;* but a satisfactory cause for the latter is lacking, while the excessive heat of the skin, sore-throat, very frequent pulse, and early eruption clear up the diagnosis.

So, also, the onset with vomiting, convulsion, and high temperature resembles *pneumonia;* but in the latter the respiration is proportionately more frequent than the pulse, with altered breath-sounds and percussion-sounds, while sore-throat and eruption are wanting.

Measles.

The course of the fever in this affection resembles that of smallpox in that after the initial rise of the first twenty-four hours the temperature remains normal until the appearance of the eruption on the third day. It is an acute, specific, infectious, and highly contagious fever, characterized by coryza and bronchitis, a red papular eruption, coming out on the fourth day and followed by a branny desquamation about the ninth or tenth day. The mucous membranes are especially liable to complications.

Measles occurs in epidemics, especially in cold weather, but individual cases are met with in large cities at all seasons of the year. It is so contagious that when one case develops in a household or institution almost every person exposed to it and not protected by a previous attack acquires it. Children from one to five years of age are most susceptible to the poison, but it may occur *in utero* and in old age; moreover, the same person may have several attacks, showing that one attack does not afford the same protection as an attack of scarlatina or variola.

Measles is sometimes found in association with scarlatina and varicella, but it is especially liable to occur after pertussis.

The specific cause of the disease has not yet been isolated.

The period of *incubation* lasts from eleven to fourteen days. During this time the patient may exhibit no symptoms, or may be irritable and restless, with disturbed sleep and occasional cough, and looseness of the bowels.

The *invasion* is marked by cough and fever, and by redness of the eyes and lacrymation, sometimes with photophobia, sneezing, and an irritating, watery discharge from the nose, which subsequently becomes

PLATE V.

Fig. I.

Fig. II.

Fig. III.

Fig. IV.

The Pathognomonic Sign of Measles (Koplik's Spots).

Fig. 1.—The discrete measles spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

Fig. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

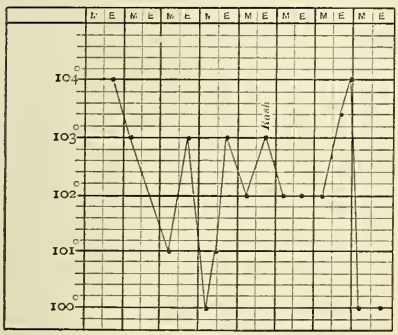
Fig. 3.—The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthema on the skin is at this time generally fully developed.

Fig. 4. Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in line. Minute *yellow points* are surrounded by a red area. Always discrete.

mucopurulent, and by cough and fever. In short, the early symptoms are those of a severe coryza. These symptoms last from three to five days (generally four) before the *eruption* appears.

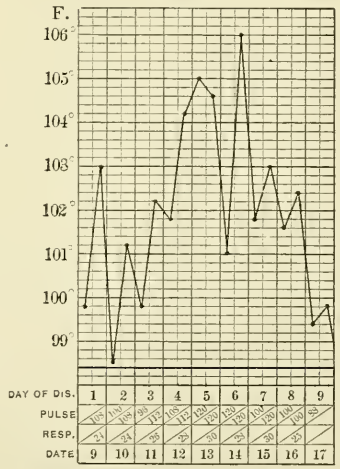
But an eruption is commonly visible upon the base of the uvula and soft palate, as raised, discrete dark-red papules, several days before it appears upon the body. The peculiar appearance of this eruption has been accurately described by Koplik (1897). His observations have been corroborated, so that "Koplik's sign" is a well-established fact. Its importance can be understood when the necessity for early diagnosis for quarantine purposes is realized. This sign appears twenty-four hours, forty-eight hours, and even three to five days before the skin eruption. It precedes the conjunctivitis and begins at the first rise of temperature. The eruption appears on the mucous membrane of the cheeks and lips. It is not seen on the palate or the fauces. It is at first discrete and then becomes confluent. It is at its height when the skin eruption appears and is spreading. In strong daylight this pathognomonic eruption is seen to consist of small irregular spots of a

FIG. 53.



Measles. Temperature taken on the first day, made higher as the result of school and exertion.

FIG. 54.

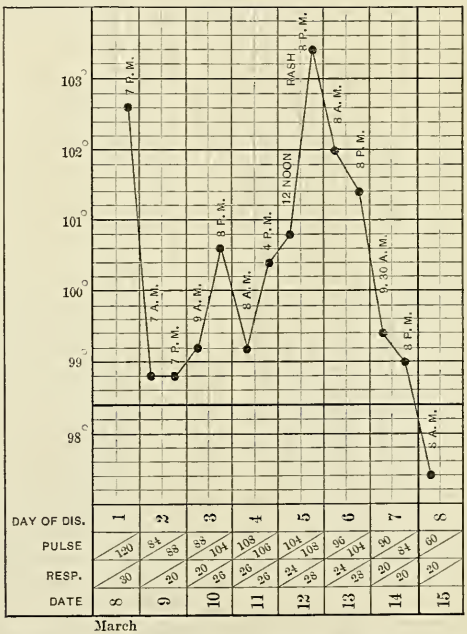


Measles. Lower temperature second and third days. Hyperpyrexia sixth day. Abundant eruption. Bronchitis severe.

bright-red color, in the centre of which is seen a minute bluish-white speck. The bluish-white speck is very small and delicately colored, requiring direct and strong daylight to see it. A combination of the speck on the rose-red background is a positive sign of the invasion of measles. The spots must not be mistaken for *sprue*, which is opaque, white, coarse, and plaque-like. When the rose-red spots coalesce, Koplik describes the appearance of the mucous membrane to be made up of large areas of rose-red, studded all over with minute raised bluish-white specks, relieved here and there by the normal hue of the uninvaded mucous membrane. The accompanying figures from Koplik's latest paper illustrate this important sign. (Plate V.) By this sign measles

can be differentiated from rötheln, scarlet fever, aphthous stomatitis, forms of erythema and urticaria, drug eruptions, the antitoxin eruption, and forms of syphilis. The *temperature* rises during the first day to 100° or 102°, or higher, if the case is to be a severe one. The bowels are frequently inclined to be loose and the passages somewhat greenish. The temperature falls on the second day to normal or nearly normal, and then steadily rises until it reaches its acme with the full development of the eruption, when, in uncomplicated cases, it falls rapidly to normal. With the coming out of the eruption the coryza increases in severity, and cough is a prominent and annoying symptom. It consists of a series of five or six explosive efforts without expectoration. In severe cases the cough is almost incessant, so that rest is much interfered with. It depends upon a catarrhal inflammation of the entire respiratory tract, from the nose to the bronchioles.

FIG. 55.



Measles. Characteristic chart. Female, aged twenty-seven.

Objective Symptoms. The eruption on the body appears first about the neck, face, and wrists, and spreads in two or three days over the entire body. It is usually most copious upon the face, which is swollen, dark-red in color, and closely set with papules, which are elevated, rounded at the summits, and feel like soft velvet to the touch. When to this picture is added that of a severe coryza with mucoserous exudate, which often glues the eyelids together and oozes out upon the face, and a corresponding condition of the nasal orifices, the physiognomy is at once seen to be very unusual. At this stage, moreover,

photophobia is often considerable, the child burrowing its head in the pillows to escape light.

The *eruption* is not apt to be confluent upon the body; here the dark-red, elevated, smooth papules are very distinct. Sometimes they are grouped so as to form crescentic outlines. The eruption fades in the order in which it appeared, and is followed by a fine branny desquamation. With the completion of the eruption the fever falls rapidly to or below normal, the coryza and bronchitis improve correspondingly, and in forty-eight hours convalescence is fully established.

Complications. The complications of measles affect for the most part the mucous membranes of the respiratory and digestive tracts. The bronchitis, which is always present, may become capillary, or be associated with œdema or with areas of catarrhal pneumonia. These are the most frequent and the most dangerous complications. Pneumonia may develop while the eruption is coming out, in which case the eruption is delayed or the spots have a dusky or bluish hue (black measles). More commonly, perhaps, pneumonia is discovered when, the eruption being complete, a crisis should occur.

Epistaxis is not usually dangerous. Profuse diarrhœa is very exhausting and delays the evolution of the eruption. Severe conjunctivitis, sometimes with ulceration of the cornea, is not uncommon. Otitis media occurs oftener as a sequel than as a complication. Noma, or cancrum oris, is a rare complication of measles occurring in ill-fed, badly nourished children. It is frequently fatal.

Convulsions may occur as a complication, especially when pneumonia is developing.

Sequelæ. In cases in which there has been diarrhœa, measles is sometimes followed by considerable weakening of the digestive power. The catarrh of the respiratory tract, which almost invariably accompanies it, predisposes to the development of whooping-cough and tuberculosis.

Paralysis may follow measles. It may be central or peripheral in origin, but generally is of the hemiplegic type; cases of acute poliomyelitis, acute ascending paralysis, and disseminated myelitis have also been reported.

Varieties. Measles without catarrh is rare. It cannot be recognized from a measles-like rash, seen in rötheln, except by the occurrence in the neighborhood of other cases of undoubted measles.

Measles without eruption is to be recognized by the coryza, possibly with eruption on the soft palate, the course of the temperature, and the exposure to specific infection.

Black measles is the name given to malignant forms in which, owing to complications, particularly pneumonia, the skin is dusky and the eruption comes out poorly and has a bluish color. In rare instances the eruption shows a hemorrhagic tendency, the spots being livid or ecchymotic. Actual hemorrhages from mucous surfaces may occur, the patient dying in coma or convulsions.

Rubella.

In a few instances this infection may run its course without fever. In the large majority of cases, however, a moderate degree of fever prevails and in some it may reach a considerable height.

Rubella is an acute, specific, contagious, and infectious fever, characterized by a gradual onset, with moderate fever, sore-throat, and slight coryza. The eruption, which appears without prodromata, usually resembles measles more than scarlatina. The duration, however, is shorter than measles, the disease milder, and complications are rare.

The disease is amply proved not to be a hybrid of measles and scarlet fever. The incubation-period varies from one to three weeks, but is generally about two. As a rule, this period is past without symptoms.

The invasion is without prodromata, or none more definite than languor and indisposition, the first thing noticed being the eruption. This in some cases consists of pale-red, smooth, slightly raised blotches, closely resembling measles, but more pronounced on the trunk, and discrete. This is probably a very rare form. More commonly it consists of rose-red maculæ or papules, occasionally confluent, but usually discrete, and most marked upon the trunk. In still other cases the eruption closely resembles that of scarlatina, differing chiefly in being a paler red and accompanied by less heat of skin. Sometimes the eruption is circumscribed, as upon the face or limbs. It is usually the seat of considerable itching, and this may be the first symptom that attracts the patient's attention. It will be seen that the eruption is multiform in character. Concurrently with the eruption, there is usually slight rise in temperature (100°–101°), suffusion of the eyes, with slight lacrymation and photophobia, and slight pharyngitis; nausea is not uncommon, but vomiting is very rare. Higher temperatures have been recorded in a few cases, and so have nervous symptoms, such as delirium and convulsions, but they are chiefly interesting as very exceptional possibilities. On the other hand, the disease may run its course without any fever.

The eruption extends over the body in twenty-four to thirty-six hours, less rapidly than in scarlatina, and pales much more quickly, fading on the portions of the body first attacked before reaching its height on the last, and being completed in three or four days. Sometimes a branny desquamation succeeds.

In addition to the mild coryza and eruption, the most important objective symptom is swelling of the cervical glands, all of them being sometimes swollen, especially those behind the sterno-mastoid, the auricle, and along the margin of the hair. This adenopathy, however, cannot be relied upon exclusively in the differentiation from scarlatina and measles.

Rubella has few complications: bronchitis, pneumonia, and otitis occur rarely, and still more rarely false membrane on the throat, and albuminuria. The prognosis is excellent. It ends almost invariably in recovery, except in very feeble children.

Infectious Diseases with Local Symptoms.

The following infections are characterized by local manifestations which are of greater diagnostic significance than the fever. These local manifestations must, therefore, be carefully considered in the diagnosis, and, as intimated, must be relied upon for recognition of the particular infection. The infections belong to Class I. and Class II. of the classification in Chapter XVII.

Mumps.

This infection presents marked local changes about the jaws coincident with the rise of temperature. The infection is recognized by the swelling of the parotid and submaxillary glands or by the occurrence of orchitis. It has been described in the chapter devoted to objective changes of the face.

Glandular Fever.

Glandular fever is an infectious disorder, the cause of which has not been accurately determined. It is characterized by fever, usually occurring abruptly, with headache, pains in the limbs and in the lymph glands of the neck. On examination of the fauces a slight pharyngitis is observed and the tonsils are enlarged. With the rise of temperature there is frequent nausea and vomiting. The temperature rises abruptly to about 102°. In the second twenty-four hours the glands of the neck, particularly those behind the sternocleidomastoid muscles, enlarge. They are tender. Although there may be some slight œdema there is no redness or swelling of the skin. The fever continues for three or four days; the enlarged glands, however, may remain for several weeks, and may end in suppuration.

The infection usually occurs in children between the age of five and eight years. It may be epidemic and occur often earlier in life than just mentioned. The other lymphatic glands about the neck and in the axilla and groin may be enlarged. In not a few instances there is enlargement of the spleen, and cases of enlarged liver and mesenteric glands are reported. The absence of an eruption serves to determine the infection from the eruptive fevers associated with adenitis, particularly measles and rötheln.

Pertussis.

The attention of the physician is called to this infection by the peculiar character of the respiratory symptoms. Fever is more notable as an expression of one of the complications—broncho-pneumonia—than of the general infection. It may, however, be a serious symptom of the infection.

Whooping-cough is a specific catarrhal inflammation of the respiratory passages, involving especially the trachea and bronchi, and characterized by paroxysms of cough, which are succeeded by spasmodic closure of the glottis and a peculiar inspiratory whoop. The disease occurs especially in childhood, is contagious and infectious, and is some-

times epidemic. Whooping-cough may be conveniently divided into three periods :

1. The catarrhal stage.
2. The spasmodic stage.
3. The stage of gradual subsidence of the disease.

First Stage. The patient appears to have an ordinary cold. The amount of redness of the mucous membrane of the eyes, nose, and throat varies considerably, but there is not much discharge from the mucous surfaces. The cough is dry, and sometimes a ringing quality can be detected. The patient is irritable, has slight fever, diminished or capricious appetite, and restless sleep. A mild bronchitis of the larger tubes can be detected by physical exploration.

The cough gradually becomes more frequent and paroxysmal, the eyes are red and suffused, and there is a mucopurulent discharge from the nose. The face often looks slightly swollen, especially about the upper part and under the eyes. Lymphocytic leucocytosis is common.

The Second Stage. Transition from the first to the second stage is marked by the appearance of the characteristic whoop. The paroxysmal cough is made up of a series of rapid expiratory efforts, diminishing in force and duration ; when these cease there succeeds a prolonged crowing inspiration—the whoop. There may be only one paroxysm of coughing at a time, but more commonly, and always in severe cases, one paroxysm is succeeded by another. During the coughing the child's eyes become suffused, the tears overflow, and there is a discharge of serum or mucopus from the nose, and of saliva and bronchial secretion from the mouth. The face becomes swollen and dusky. If the child is walking about, it catches some object for support during the paroxysm ; or, if old enough, rushes for the water-closet or a basin, because the seizure usually terminates in vomiting. The matters vomited consist of tenacious mucus and the contents of the stomach. With the mucus there may be streaks of blood, and occasionally there is pure blood. During severe paroxysms, hemorrhages are apt to occur ; these are generally small and most frequently submucous. In well-marked cases, when the disease has lasted some time, the face has a characteristic appearance—it is swollen, sodden, and dusky, with dull, heavy, red, and watery eyes. There is often ulceration of the lingual frænum.

The number of paroxysms varies from two or three to twenty or thirty or more in twenty-four hours, and they are worse at night.

The whoop, while characteristic, is not present in every case, being absent especially in babies and very young children. Sometimes children have " choking spells " without much coughing and without the whoop. Again, when pneumonia or measles occurs as a complication, the whoop usually ceases for the time, but may reappear later.

Third Stage. The third stage is less well defined than the first two. It may be said to begin when the nocturnal exacerbations become less frequent and severe. The number of paroxysms during the day diminishes, and vomiting is a less frequent accompaniment. Appetite begins to improve, and the child begins to gain in flesh and to pass more restful nights.

The duration of the disease is variable. Ordinarily it lasts from six to eight weeks, but it may be prolonged for several months. The patient is liable, whenever he catches a fresh cold, to a temporary return of the spasmodic cough, sometimes with the whoop.

The great majority of the cases occur before the sixth year, and most of these between the second and fourth years.

Rheumatic Fever.

Rheumatic fever is an infection associated with local symptoms of joint-, endo-, and pericardial inflammation. The local symptoms are so extreme as to call attention at once to the nature of the infection apart from the course of the fever, as it is largely upon these symptoms that the diagnosis is made. The reader is referred to Chapter XIII., in which the diagnosis of rheumatic fever is discussed.

Dengue.

The peculiarity of the fever in this infection is that it is attended by severe pains in the muscles and joints. It is an acute contagious disease, occurring in epidemics and characterized by severe pains in the head, back, and joints, various skin eruptions, a prolonged convalescence, and a very low rate of mortality.

The disease occurs in epidemics in tropical and subtropical countries, and rarely in cooler climates. It derives its name, dengue (dandy), from the stiff and unnatural gait assumed by convalescent patients. In the southern parts of the United States an expressive name given to the disease is "breakbone fever."

The specific cause of the disease is believed by Dr. McLoughlin to be a micrococcus which is isolated. The period of incubation is short, varying, however, from a few minutes to several days, or even a week. Invasion is very sudden and is rarely preceded by any prodromata. It is marked by chilliness or a chill, and very severe pains in the head, back, and limbs. In children the onset may be by convulsions, which are sometimes followed by stupor and vomiting. The pains are sometimes excruciating, and are accompanied by tenderness of the muscles; there is extreme debility. The temperature rises to 102° or 103°, but rarely is much higher.

The pulse is frequent—110, 120, or more. In from one to three or five days the temperature falls to or below normal (the remission), accompanied by sweating or diarrhœa, and fluctuates about this level for several days, when a second and moderate rise in temperature, which is of short duration, occurs. During the first rise in temperature there is a transient, generally scarlatiniform rash, which is not followed by desquamation. The urine is febrile but not albuminous. During the remission eruptions—scarlatiniform, herpetic, urticarial, or like miliaria—begin to appear, accompanied by the secondary rise in temperature. The eruptions may be in successive crops, and are followed by desquamation. Convalescence is now established, but may be interrupted by relapses. Strength is regained very slowly. The most frequent complications are disorders of the nervous system, but bronchitis and diarrhœa occasionally occur.

Beri-beri.

Beri-beri is a febrile infectious disorder which prevails in epidemic form, limited to tropical and subtropical countries. It is characterized by multiple neuritis associated with anasarca. By most observers it is believed to be an acute infection, although not a few think it is an intoxication due to certain kinds of food. This is the view which prevails in Japan. The circumstances predisposing to infections generally prevail, however, such as overcrowding, the prevalence in hot and moist seasons, and the exposure of the patient to climatic influence. It is far more common in men, and usually attacks subjects whose ages range from sixteen to twenty-five.

Several clinical forms are seen. In the most complete form there is rapid loss of power in the legs and arms, with atrophy of the muscles. The patients complain of pain, and later œdematous symptoms may appear. With the loss of power in the legs there is paræsthesia, with frequent palpitation of the heart and dyspnœa. The pain in the muscles is associated with weakness and tenderness. In milder degrees of this form, pain, weakness in the legs, diminishing of the sensibility, and paræsthesia are the most common symptoms. Their onset will be gradual and be accompanied by catarrhal symptoms. The symptoms may recur from time to time, and are much more aggravated during the warm season. Its recurrence and incomplete form may continue ten or fifteen years.

Following the pain and weakness of the muscles, in some cases œdema becomes very pronounced, associated with effusions into the serous cavities. General anasarca is attended by palpitation and rapid action of the heart and dyspnœa. In this so-called wet or dropsical form atrophy of the muscles is not observed until the œdema disappears. In some instances the infection is very intense, and is characterized by more marked cardiac symptoms. In these instances acute dilatation may be followed by cardiac paralysis and death in twenty-four or forty-eight hours.

The diagnosis is based upon the occurrence epidemically or endemically in tropical regions of peripheral neuritis with œdema. Thus far no bacteriological diagnosis obtains.

Constitutional Syphilis.

Intermittent, remittent, or continuous fever is attendant upon this infection sometime during its course. (See Afebrile Infections, Chapter XVI.) Want of recognition of the cause of this febrile phenomena leads to many mistakes in diagnosis. (See Fig. 63.)

Constitutional syphilis may be acquired or congenital.

Acquired syphilis is characterized, first, by the initial lesion, or chancre, which appears usually in a week after contagion; second, by a period of incubation generally lasting six weeks, but varying from one to three months; third, by so-called secondary symptoms, comprising febrile symptoms, polymorphous skin-eruptions, ulcers upon the tonsils, adenitis, less frequently mucous patches in the mouth, or condylomata about the anus, iritis and retinitis, and loss of hair. The lesions of this period are symmetrical. Fourth, after an interval vary-

ing from several months to twenty years, by so-called tertiary phenomena, which manifest themselves in some cases. These are due to chronic inflammatory indurations of the skin and subcutaneous tissue, resulting in suppuration and ulceration ; or of the bones, producing periostitis and necrosis ; or of organs, producing gummata and cirrhosis ; or of the nervous system, resulting in gummata or chronic degenerative changes. The lesions of this period are unsymmetrical.[1]

The course of syphilis in different persons varies as widely as any of the eruptive fevers. In some the chancre is a mere papule which heals almost unnoticed ; no secondary symptoms appear, and tertiary symptoms also are altogether wanting, or a chronic degeneration of the nervous system develops after the lapse of many years, the patient in the meantime remaining in apparent health. All this may occur, too, without the aid of specific treatment. In other cases the disease is malignant ; tertiary symptoms appear very early or appear to take the place of secondary symptoms ; ulceration may rapidly melt down and destroy the alæ of the nose or the soft palate ; or rebellious periostitis with necrosis may attack the tibiæ, the nasal bones, or the cranium.

In an ordinary case of acquired syphilis, in about six weeks after the appearance of the chancre, the patient complains of languor, weariness, slight fever, pains in the bones, impaired digestion, and a tendency to anæmia. An eruption now appears. It is most marked on the trunk and upper extremities, especially the chest and forehead (corona Veneris). The eruption may be roseolous, squamous, vesico-papular, papular, pustular, bullous, or tubercular. The color has been aptly compared to that of a slice of raw ham. The enlargement of the inguinal, epitrochlear, and postcervical glands, which precedes the eruption, persists. Shallow ulcers with a sharply defined grayish outline appear on both tonsils. They are painless and do not spread. Ulcers are also liable to appear upon the pharynx, buccal surfaces, tongue, angles of the mouth, penis, vulva, vagina, and around the anus. In the mouth these are apt to be very painful, and may persist in spite of treatment for weeks or months. Relapses are not uncommon. Sometimes there are raised white patches upon the pharynx. Sometimes the hair becomes very thin and falls out, leaving the patient without eyebrows and more or less bald. Iritis and retinitis are usually later symptoms. Other symptoms occasionally occurring at this stage are periostitis, usually slight, and onychia.

The most common of the symptoms enumerated are the eruption and the tonsillar ulceration.

The eruption comes out gradually during two or three weeks, and persists for about two months. Rarely, however, it is fleeting, or, on the other hand, is unduly prolonged.

The secondary symptoms last from six to eighteen months. After their disappearance the patient may remain entirely well for life. In other cases after apparent health, lasting for months or years, the tertiary phenomena already mentioned appear. In the interval the patient may have suffered with various local skin eruptions or with ulcers upon the buccal mucous membrane.

[1] *Fever* is a constant accompaniment of all forms of syphilis. (See Fever.)

The tertiary lesions of syphilis are the late *syphilides* (see Skin) and *gummata* of the skin, subcutaneous connective tissue, muscles or internal organs. Visceral syphilis is seen at this stage. In the brain and spinal cord gummatous tumors, gummatous meningitis, gummatous arteritis, and localized scleroses are found. The symptoms are those of brain tumor when the cerebrum is affected, and of tumor, meningitis, or sclerosis when the cord is affected. In syphilis of the lung we may find gummata scattered through the lung or a fibrous interstitial pneumonia beginning at the root of the lung. Diffuse syphilitic hepatitis or gummata may be found when the liver is affected. The rectum is the most common seat of syphilis of the digestive tract. Myocarditis and localized gummata and endarteritis occur in cardiac syphilis, while in vascular syphilis obliterating endarteritis and gummatous periarteritis are found. Syphilitic orchitis often occurs. Its presence may aid in the diagnosis of obscure visceral syphilis.

Hereditary syphilis differs in some respects from the acquired form. At birth the syphilitic infant usually exhibits no evidence of its inherited taint. In the course of from one to twelve weeks it develops a catarrhal inflammation of the nasal mucous membrane, which causes snuffling in breathing, and hence is called "snuffles." An eruption soon appears, symmetrical in distribution. It is most frequently erythematous or papular, but it may be squamous, vesicular, pustular, or bullous. In hereditary syphilis it is more apt to be moist and to favor the genitalia and flexures of the thigh than in acquired syphilis. It is of the same ham-color as in acquired syphilis. Coincident with the "snuffles" and eruption appear stomatitis and ulcers at the angles of the mouth, and sometimes condylomata around the anus. Meantime the child has begun to waste, to be peevish, to be anæmic, and gradually to assume the appearance of a wizened, dried-up old man. As in acquired syphilis, there may be iritis, though it is uncommon, and inflammation of the other structures of the eye, but nodes and disease of the liver are rare. The infant very frequently dies during this period from exhaustion and inanition.

If the child survives for a year the secondary symptoms usually disappear and the disease becomes latent. Relapses may occur, and in them, according to Mr. Hutchinson, condylomata are likely to appear. The same observer states that the tertiary period may begin at any time after the fifth year, but it is commonly delayed till about the period of puberty. In the meantime the patient may appear fairly well, but usually his development is retarded, there is a tendency to anæmia, and he has often nasopharyngeal catarrh, flattening of the bridge of the nose, premature decay of the upper incisor teeth, and protuberant forehead.

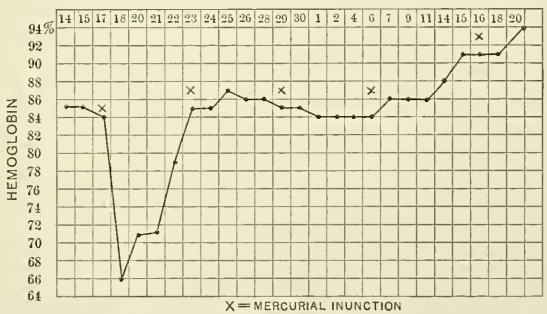
The teeth may be perfectly normal, in other cases characteristically syphilitic. The malformation affects especially the upper central incisors of the permanent set. It was first described by Mr. Hutchinson. It "consists in a dwarfing of the tooth, which is usually both narrow and short, and in the atrophy of its middle lobe. This atrophy leaves a single broad notch (vertical) in the edge of the tooth, and sometimes from this notch a shallow furrow passes upward in both anterior and posterior surfaces nearly to the gum. This notching is

usually symmetrical. It may vary much in degree in different cases; sometimes the teeth diverge, and at others they slant toward each other." (See Part II., Chapter IV.)

Further, the patient may have had or may now be attacked with keratitis, affecting both eyes, producing cloudy opacities and accompanied by great photophobia. Again, there may be nodes upon the long bones, with nocturnal exacerbations of pain. Cerebral deafness, according to Hutchinson, is not rare, but cerebral blindness is. There may be ulceration upon the legs, and periostitis and necrosis. The patient usually recovers completely, but he is more liable to be carried off by intercurrent disease than a healthy person, and in general has less resisting power, especially to tuberculosis.

Diagnosis. The diagnosis of hereditary syphilis is based upon the occurrence of snuffles and skin eruptions, and the existence of keratitis or of cicatrices, especially about the angles of the mouth. A history of repeated miscarriages is suggestive of maternal syphilis. The diagnosis of acquired syphilis is based upon the history of chancre, when that history is obtainable; upon the existence of polymorphous eruptions, or of non-traumatic ulcers upon the legs of young adults, or of scars in the groins or over the tibia, or of nodes, or of alopecia associated with sore-throat or mucous patches. The presence of obscure disease of the bones, glands, or spinal cord should lead to the search for a possible syphilitic infection. (See Malaria, Chapter XIX.)

FIG. 56.



Reduction of hæmoglobin after mercurial inunction in syphilis.

Examination of the blood during mercurial treatment may, in accordance with Justus' observations, show the presence of syphilis. If this disease is present the percentage of hæmoglobin falls suddenly and rapidly during the hours immediately following the first administration of the drug. Cabot has confirmed his observations. The accompanying chart shows the effect of mercury upon the blood. (See Fig. 56.)

Weil's Disease.

The occurrence of *jaundice* without local hepatic symptoms during the course of fever suggests an infectious process. It is a well known symptom of pyæmia and septicæmia. In the following infection fever and jaundice are coördinate symptoms. *Acute febrile jaundice*, which rapidly becomes malignant, occurring in butchers, laborers,

and brewers, has been described by Weil. After exposure to cold generally, as in a beer-vault, the patient is seized with a chill, followed by fever, with headache, vomiting, and epigastric pain. Jaundice sets in rapidly. The temperature remains high, or may be intermitting. Stupor, delirium, and coma, albuminuria, with suppression of urine, subcutaneous hemorrhages, and hemorrhages from mucous membranes, rapidly ensue. Black vomit occurs early. In one of my cases there was enlargement of the liver, with subcutaneous œdema over the hepatic area. The microscopical appearances were those of acute diffused parenchymatous inflammation. In another, a brewerymman, the liver was enlarged, but without unusual change, save congestion.

The delirium is sometimes violent. The appearance and symptoms suggest acute yellow atrophy of the liver. The etiological distinctions are noteworthy: the liver is not small; leucin and tyrosin are not found in the urine; the jaundice is more intense. The diagnostic circumstances of epidemic and contagious diseases serve to exclude yellow fever. (See Yellow Fever.)

Miliary Fever.

The occurrence of fever in association with profuse sweating is rarely seen without attendant signs of pyogenic infection. When several cases with these symptoms occur at the same time, suggesting an epidemic, the infection we are about to consider must be thought of.

Miliary fever, or sweating-sickness, is an infectious disease, occurring in epidemics, and characterized by moderate fever, profuse sweating, tenderness and a sense of oppression at the epigastrium, and a vesicular eruption. The disease has occurred epidemically in England, but is not met with now outside of France and Italy.

After mild prodromal symptoms the disease sets in suddenly with moderate fever, profuse sweating, and epigastric distress, sometimes amounting to anguish. The characteristic eruption appears on the third or fourth day. It consists first of small reddish maculæ, in the centre of which a vesicle develops. The latter varies in size from a pinhead to a pea. The contents are at first clear, but subsequently become purulent. Desiccation and desquamation follow. The eruption is most profuse generally upon the neck and trunk. Sometimes there are marked nervous symptoms, and even convulsions and fatal collapse.

It is distinguished from rheumatism by the moderate fever and absence of joint-swellings, and from malarial fever by the absence of chills, of periodicity in the febrile movement, and absence of malarial organisms from the blood.

The duration of the disease is from one to four weeks. The mortality in some epidemics has been very high, in others very low.

Infections Transmitted from Animals to Man.

When fever occurs in persons in contact with animals or their products the possible occurrence of the infections—milk-sickness, foot-and-mouth-disease, and rabies, as well as *glanders* and *anthrax*—must be thought of. The infections which follow are of uncertain bacteriology, and are recognized not alone by the fever but also by the local symptoms and a history of infection.

Milk-sickness.

It is an acute disease affecting cattle, and transmitted from them to human beings in the milk or meat. The disease is limited to a few sparsely settled localities west of the Allegheny Mountains. It is characterized by great debility, with muscular tremor upon motion (hence the name "trembles"), vomiting (hence called "puking fever"), a peculiar fœtor of the breath, obstinate constipation, and moderate fever or subnormal temperature. The vomited matters are said to be of a peculiar soapy material of yellowish or greenish color. The duration is usually less than a week. The patient may sink into a typhoid condition and die in coma, or he may die in a few hours. Convalescence is protracted.

Foot-and-mouth Disease.

A specific, infectious disease, communicated to man through cattle, sheep, or pigs, and characterized by a stomatitis. It is communicable by milk; the period of incubation is from three to five days. Invasion is characterized by slight fever, heat, and soreness of the mouth, and the development of vesicles, which burst and leave shallow ulcers. Saliva is freely poured out. The tongue swells greatly, and eating is painful. Vesicles sometimes appear about the fingers, but not upon the feet. The disease lasts from one to two weeks, and ends almost invariably in recovery.

Hydrophobia.

An acute, specific disease communicated to human beings by the bites of animals similarly affected. The animals most frequently affected are the dog, fox, wolf, cat, and skunk; 90 per cent. of the cases in human beings are due to dog-bites.

The period of *incubation* is uncommonly long and very variable—from two weeks to two months usually. It is said in some cases to be a year or more. The disease has been divided into three stages—the melancholic, the spasmodic, and the paralytic.

In the *melancholic stage* there is pain, hyperæsthesia, or even reopening of the healed wound. The patient is extremely depressed in spirits, and may be irritable. He seems to be laboring under a constant tension of fear, and is keenly sensitive to light, sounds, or draughts. He is affected with thirst, but attempts to swallow water cause intensely painful spasm of the larynx.

The *second stage* is reached usually on the second day. The laryngeal spasms are increased and lead to intense dyspnœa and to pitiable struggling and gasping on the part of the patient. In addition to the convulsive seizures, the patient foams and froths at the mouth, and his face expresses the extreme terror and mental anguish he feels. The second stage lasts from one to three days, and is followed by the *third stage*, exhaustion intermitting with paroxysms of less severity. The patient may now be able to swallow easily, but there is great weakness of the heart, and death may occur from failure of the heart, from asphyxia, or in a convulsion. The duration, as indicated, is only a few days. The result is practically always fatal, but recovery may be possible. Bites of the face are the most likely to be fatal.

CHAPTER XIX.

THE DATA OBTAINED BY OBSERVATION—(*Continued*).

FEVER. THE INFECTIOUS DISEASES.

Infections Recognized by Examination of the Blood.

Microscopical Examination. The following infections are recognized by the examination of fresh blood: *Relapsing fever, malaria, yellow fever, anthrax.* Typhoid fever is also recognized, but is more frequently diagnosticated by means of serum diagnosis and by culture methods. By *staining* cover-slip preparations of the blood the diagnosis by the direct method is confirmed.

Serum diagnosis enables us to determine the presence of typhoid fever, yellow fever, and Malta fever.

Bacteriological examination of the blood corroborates the diagnosis of typhoid fever made by the above methods. By it we are also enabled to determine the presence of gonorrhœal infection, of cerebro-spinal meningitis, of the pneumococcus infection, and, in many instances, of infection due to the staphylococcus, streptococcus, and bacillus coli communis. The gonococcus infection alone will be considered. It must be remembered that the micro-organisms cannot be found in the blood until late in the course of the disease, and even then the infection must have a certain degree of intensity. Unfortunately, they cannot be demonstrated in the majority of cases. Positive cultures for the above reasons are very valuable. Negative cultures do not exclude septic infections.

Relapsing Fever.

Relapsing fever is the first infection which we will consider, because historically it is the most important. It is the first infection in which a micro-organism was found to be causal, and is one to which Koch's laws can be applied. It is an acute, infectious, and contagious fever, occurring in epidemics, and characterized by the sudden onset of a febrile period lasting five or seven days, which is followed by an intermission lasting usually a week, and this in turn by a relapse lasting three days. Its development is favored by filth and famine, but the specific cause is believed to be the spirillum of Obermeier, which is constantly present in the blood during the febrile stage.

The stage of *incubation* lasts from five to eight days (Pepper), during which the patient may complain of malaise, lassitude, and flying pains. The *invasion* is sudden. It manifests itself by a chill or chills, frontal headache, pains in the back and limbs, vertigo, and great physical weakness. The temperature rises very rapidly, reaching 105°, 106°,

or even higher, in the first day or two. The face is flushed, epistaxis sometimes occurs, the headache and other pains persist, but delirium is not common. The appetite is usually lost, thirst intense, the tongue coated white but moist, the bowels constipated. A mild catarrhal jaundice is not infrequent. Pepper states that nausea and vomiting are prominent symptoms, the matters vomited at times containing blood. Tenderness with pain in the epigastrium is frequently complained of.

The *urine* is scanty, high-colored, and frequently contains albumin and casts; when jaundice exists the urine contains bile-pigment and sometimes blood.

There is no peculiar *eruption* in relapsing fever, but in this, as in other fevers, erythemata, petechiæ, and sudamina may be present.

The *pulse* is often very frequent and soft, and hæmic murmurs may be audible.

The *objective symptoms* are few. They consist of the flushed face, sometimes with slight jaundice and epistaxis, tenderness in the epigastrium, with moderate enlargement of the spleen and liver, and considerable cutaneous hyperæsthesia, with tenderness along the nerve-trunks.

Bronchitis and sometimes hypostatic congestion of the lungs, with their usual physical signs, may be present.

These symptoms continue without much change until the fifth or seventh day, when a decided *crisis* occurs. Sometimes this is deferred until the tenth day. The *temperature* within twelve hours falls from 106° or 108° to or below normal; the pulse diminishes in frequency from 120 or 130 to 60 or 70; vertigo, headache, and other pains disappear as by magic. The crisis is marked most frequently by a profuse sweat, sometimes by diarrhœa, epistaxis, metrorrhagia, or intestinal hemorrhage. The patient now enters upon convalescence without fever, and apparently makes rapid strides toward complete recovery. On the seventh day from the crisis, however, a sudden relapse occurs, with a repetition of the symptoms of the first attack. The temperature may be higher and the febrile symptoms more severe, but the duration is shorter—only three or four days. The spirilla, which disappeared in the apyretic interval, are again found in abundance. A second crisis, with its associated symptoms, now occurs. The spirilla again disappear, and in the majority of the cases there is no further bar to complete recovery. A second, third, and even a seventh relapse may occur, as in a case reported by Pepper. Organic lesions are not usually left behind, unless they have occurred as complications; but even in ordinary cases the patient is left weak, anæmic, and with poor circulation.

Examination of the Blood. Microscopical Examination. In the blood at the height of the disease the spirillum of Obermeier is found.

These are slender, wavy, thread-like organisms of spiral shape, seven or eight times the length of a red blood-cell, with a very lively forward movement in the direction of the long axis. They are from 16 to 40μ by 0.1μ. Under a low power the blood may appear to be in motion, as the result of their movement. They have so far been found only in the height of the febrile attacks; but Von Jaksch states that as long as a relapse is to be feared the blood contains peculiar, highly refracting bodies

resembling diplococci, which are especially numerous before the attack; in some cases it has seemed to him that these diplococci at the very beginning of an attack develop into short, thick rods, from which the spirilla develop; they may, therefore, prove to be spores. Staining is unnecessary for the detection of spirilla, but cover-glass preparations of the blood can, if desired, be stained with fuchsin or gentian-violet or Löffler's methylene-blue. (Plate III., Fig. 4, a.)

Serum Diagnosis. It sometimes happens that a diagnosis should be made during the afebrile period when the organisms have disappeared entirely from the peripheral circulation. Löwenthal's method is as follows: A drop of the suspected blood is mixed with one containing the living micro-organisms. The mixture is sealed up with wax between slide and cover-glass and left in the thermostat at 37° for half an hour. Blood from a patient who has just had a paroxysm will destroy the spirilla, so that they lose their motility and spiral curl and accumulate in bunches. The reaction is like that of Pfeiffer's phenomena rather than agglutinative. It is to be remembered that the bactericidal power of the blood dies out before the next paroxsym.

Inoculation. As further aid to diagnosis typical relapsing fever can be produced by injecting the infected blood into monkeys.

The most frequent *complications* are on the side of the lungs, kidneys, and heart. Lobar pneumonia is the most frequent. The heart becomes weakened by the very high fever and thrombosis, or sudden failure results. Embolism is very frequent. Suppurative parotitis, abscess of the spleen, profuse epistaxis, abortion in pregnant women, and neuritis deserve mention.

Relapsing fever occurs at all ages, but is most common in adults.

The duration varies according to the number of paroxysms. If there is only one, it is about eighteen days. Under the name "bilious typhoid" a malignant form of relapsing fever has been described. It is characterized by intensity of the symptoms of the ordinary form, and by bilious or bloody vomiting, jaundice, and delirium, or by collapse, with purple nose, a small, frequent weak pulse, rigidity of the abdominal muscles, tenderness in the epigastrium, and cold, clammy skin. In some of the cases described by Graves, intussusception of the intestines was found after death. In other cases uræmia is an active factor.

Diagnosis. The earlier cases in an epidemic may not be recognized, unless the blood be examined, until the occurrence of the characteristic relapse. The diagnosis is based upon the occurrence of an epidemic, the presence of the predisposing factors, the clinical course, and the examination of the blood. It is most likely to be mistaken for *typhus fever*, which occurs under similar conditions. The aspect of the two diseases is very different. In typhus there is a heavy, stupid, sometimes besotted expression, with slight redness of the eyes and a contracted pupil. The patient lies oblivious of his surroundings, with low muttering delirium and ataxic symptoms. In relapsing fever, on the other hand, the sensorium is rarely much disturbed, the spleen and liver are enlarged, and there is hyperæsthesia. Moreover, in typhus there is a spotted eruption, later becoming petechial. In relapsing fever this is absent.

Anthrax.

The next infectious disease, the cause of which can be determined by an examination of the blood, is anthrax. This affection is also of historical importance, and is probably the best worked out of any of the infections common to man and the lower animals. It is also called malignant, pustule, charbon, wool-sorter's disease, splenic fever. It is derived principally from herbivorous animals, and characterized by the development of a pustule or boil, with extensive brawny œdema and subsequent toxæmia; or toxæmia may appear first and metastatic abscesses subsequently. The disease also attacks the gastro-intestinal mucous membrane and the lungs.

Anthrax is caused by the anthrax-bacillus and its toxins. Outside of the body it forms endogenous spores, which are extremely tenacious of life, and to which infection is invariably due. They infect not only the carcasses of animals, but also the soil, all utensils used in the care of the animals or the soil, and they persist with infective power in the hides, hair, hoofs, and wool ("wool-sorter's disease"). It is possible that it may be transmitted to man by stings of insects, particularly flies and mosquitoes.

The period of incubation varies from a few hours to several days. In the form known as *malignant pustule* the patient has a pricking or burning feeling, which may lead him to think he has been stung by an insect at some exposed part of the body, particularly the hand, face, or neck. At the seat of irritation, first a papule, then a vesicle, develops. The vesicle may attain considerable size. The contained fluid quickly passes from clear to bloody, and then escapes, leaving a dark-brown or black scab (anthrax).

The original vesicle may be surrounded by a series of smaller ones. Instead of disappearing, the base of the vesicle becomes inflamed and indurated, the induration extending to surrounding tissue and causing a condition of brawny œdema. A whole arm or one side of the face and neck may be swollen. There may or may not be an associated lymphangitis and adenitis.

The general health does not suffer at first, but in a day or two fever sets in, accompanied by delirium, sweating, great weakness, enlargement of the spleen, severe pains in the limbs, and diarrhœa. Death, preceded by collapse, may occur in from five to eight days (Fagge), or the tissue occupied by the pustule may slough out.

Bollinger and others have called attention to *anthrax œdema*, in which there is no pustule, but only a yellowish or greenish swelling of the tissues. Gangrene may ensue. It is seen most frequently in the eyelids, but may be on the head, hand, or arm.

Intestinal Form. Anthrax of the gastro-intestinal mucous membrane, as described by Bollinger, presents the following symptoms: the patient first complains of malaise, loss of appetite, pains in the limbs, giddiness, and headache. Then vomiting may set in, and a more or less severe diarrhœa, the evacuations often containing blood. There may be pain in the abdomen, which becomes somewhat tumid; the spleen is enlarged. Dyspnœa and lividity appear, with restlessness

and with excitement or stupor. Epileptiform convulsions may occur, the upper limbs may be affected with tetanic spasms, there may be opisthotonos, and the pupils may be widely dilated. The pyrexia is slight, and death is preceded by extreme collapse. The duration of the disease is usually from two to seven days, but sometimes it is scarcely twenty-four hours.

Wool-sorter's Disease. Still another form of anthrax occurs among the wool-sorters of Bradford, England; it is characterized by intense dyspnœa and a feeling of oppression or constriction. Breathing is labored, but not much accelerated. Only a few coarse râles are to be heard on auscultation. The expectoration may be abundant and bloody, or absent. There is a tendency to collapse, with cold, bluish skin, and a subnormal axillary temperature. The rectal temperature, however, is raised two or three degrees. Death may occur in coma and convulsions, or suddenly, the mind being clear. The duration of the disease is from one to five days. Dr. Bell says that those who survive for a week generally recover.

Examination of Blood. THE BACILLUS ANTHRACIS is found in the blood of the patient or the pus of the lesions of anthrax or malignant pustule.

MORPHOLOGY. A bacillus, 2 to 3μ up to 20 to 25μ in length and 1 to 1$\frac{1}{4}\mu$ in breadth. The bacilli are often joined end to end in long threads, and these threads are massed together in bundles. As found in animals they are short rods with square ends. They stain best with Löffler's blue, but also with the basic anilines and by Gram's method. When in the stage of spore-formation the threads look like strings of beads.

FIG. 57.

Bacillus anthracis highly magnified, to show swellings and concavities at extremities of the single cells. (ABBOTT.)

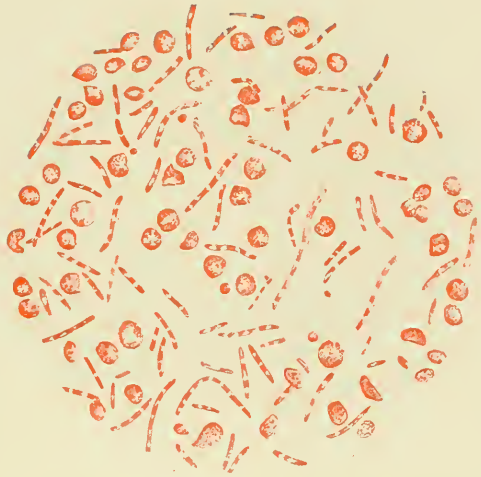
CULTURES. BIOLOGICAL PROPERTIES. It is aërobic, non-motile, and liquefies gelatin. (See Plate III., Fig. 2, A; Plate VI.; Fig. 57.)

It grows best in neutral or slightly alkaline media (gelatin, agar, milk, meat-infusion, etc.) at 20° to 38° C. The growth-limits are 12° and 45° C.

Cultures on agar are quite characteristic, consisting of a dense central mass with twisting and crossing bundles all around it. In gelatin stab-cultures a fine branching threadwork grows out alongside the puncture. The gelatin soon liquefies and the bacilli settle in white masses. The growth is abundant on potato, and is grayish, dry, rough, and irregular. The virulence is attenuated by cultivation. Drying does not kill the spores. Very toxic substances are found in the culture-medium.

PLATE VI.

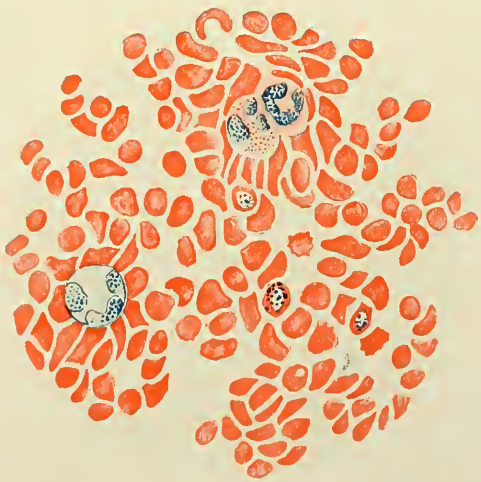
FIG. 1.



Anthrax-bacilli from Rabbit's Spleen.

(Oc. 4, ob. $\frac{1}{12}$ immersion.) Drawn by J. D. Z. Chase.

FIG. 2.



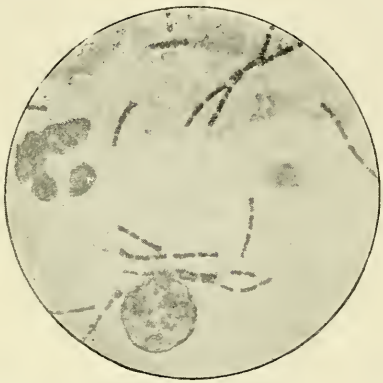
Protozoa of Malaria, Intracellular and Crescentic Forms.

(Oc. 4, ob. $\frac{1}{12}$ immersion.) Drawn by J. D. Z. Chase.

Inoculation. When *inoculated*, the organism produces the pustule of anthrax. If inoculated into the abdominal wall of a guinea-pig or rabbit death follows in forty-eight hours. No reaction is seen at the point of inoculation, but beyond this the tissues are œdematous. Ecchymoses are seen, and the underlying muscles are pale. The spleen is enlarged, dark in color, and soft. Cover-slip preparations confirm the diagnosis.

Anthrax bacilli are not so numerous in human blood as in that of the lower animals. They are most likely to be found in the spleen, which is apt to be much swollen.

Fig. 58.



Bacillus anthracis in the blood of a guinea-pig. × 1040. (Gibbes.)

Diagnosis. In doubtful cases a mouse or guinea-pig should be inoculated with the blood. *Carbuncle* is distinguished by its tendency to develop upon the back or shoulders and other covered portions; anthrax on uncovered portions. In carbuncle there is a series of openings resembling a sieve, filled with pus and plugs of necrotic tissue. In anthrax there is at first a central black crust. The boggy feeling of carbuncle is different from that of the brawny œdema of anthrax. Finally, in carbuncle, anthrax-bacilli are not found in the blood.

The intestinal and thoracic forms are distinguished by the occupation of the patients, the absence of other adequate cause, and the result of the blood-examination, cultures, and inoculation experiments.

Malarial Fevers.

The next infection which we are about to consider is one of the most common the world over. In its various forms it is recognized by direct examination of the blood. Its clinical features are such that often but little difficulty surrounds its recognition, but no case should be unqualifiedly pronounced malaria without an examination of the blood. It comprises a group of fevers associated with the protozoan organism of Laveran, and is characterized by periodic paroxysms of chill, fever, and sweat. They are not contagious, but can be transmitted by inoculation.

Malarial fevers, while most prevalent in tropical and subtropical

regions, are found also throughout the temperate zone, especially in autumn and spring. In Europe their favorite habitat is Italy, and in the United States the Southern and Southwestern States. Conditions that especially favor their development are marshes and swamps, fed partly by sea-water; low ground along streams of slow current, and freshly upturned soil. The poison is carried in the air.

The specific poison in malarial fevers is no doubt organic. The protozoan organism described by Laveran exhibits several different forms, which he regards as stages in the development of one organism, but which may be different species. Golgi maintains that there are several distinct varieties of parasites whose periodicity in development and sporulation corresponds with the different types of fevers.

Intermittent Fever. This is a type of malarial fever in which the temperature remains normal between the paroxysms.

A malarial paroxysm is characterized by (1) chill, (2) fever, and (3) sweating, occurring in the order named and in immediate succession. The time between the beginning of one paroxysm and the beginning of the next is called the "interval," that between the conclusion of a paroxysm and the beginning of the next the "intermission." The interval varies in different forms of intermittent fever: in the quotidian there is a paroxysm every day, with an interval of twenty-four hours; in the tertian there is a paroxysm on alternate days, with an interval of forty-eight hours; in the quartan there is a paroxysm every third day, with an interval of seventy-two hours. In double quotidian there are two paroxysms in the twenty-four hours, but not of the same intensity.

In the double tertian there is a paroxysm every day, the first and third and second and fourth corresponding as to hour and intensity. That is to say, if there be a paroxysm at 10 A.M. Monday there will be another severe paroxysm at 10 A.M. Wednesday, while on Tuesday and Thursday there will be milder paroxysms, but at another hour than 10 A.M.

In the double quartan severe and mild paroxysms succeed each other every other day, but each third day is free from any paroxysm.

While the rule is for malarial fevers to occur periodically at the same hour, the second paroxysm may occur an hour or two earlier (anticipation) if the disease is growing worse, or an hour or two later (postponement) if it is growing better. (See Figs. 30, 31, 32.)

Quotidian intermittents are slightly more common than tertian, while the quartan variety is rare.

The *incubation*-period probably varies widely, depending upon the intensity of the poison. As a rule, repeated exposure is necessary to develop the disease in temperate climates. During this period the patient may suffer with headache, drowsiness, pains and aching in the limbs and back, constipation, a coated tongue, and thirst.

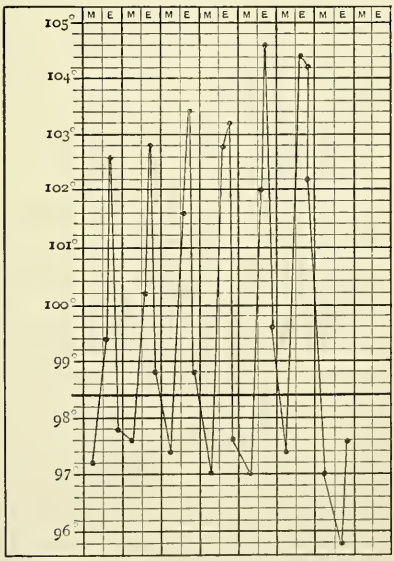
The *onset* of a typical malarial paroxysm is marked by chilly sensations, especially along the spine, accompanied by yawning and the development of "goose-flesh." Then a decided chill sets in, the patient shaking violently. The face is pale and pinched, the lips blue, the nose pointed; as the chill becomes worse the teeth chatter, the whole body feels cold, the skin feeling rough, dry, cold, and harsh. The

finger-nails and toe-nails are blue, the skin being wrinkled upon the palmar and plantar surfaces. The superficial bloodvessels are so contracted that a drop of blood is obtained with difficulty. The voice is thin and weak, almost inaudible.

The volume of blood driven from the surface leads to congestion of the viscera, particularly the spleen, liver, and stomach. Nausea and vomiting are not uncommon. The spleen is perceptibly enlarged, and frequently the liver also.

Although the surface temperature is depressed, the internal *temperature* is rising, and may be two or three degrees above normal. By degrees the severity of the chill abates and the patient asks to have the extra bedclothing removed. Reaction has set in. The surface-bloodvessels dilate and the skin becomes flushed. The temperature continues to rise, often reaching 103° to 106°, pulse and respiration increasing correspondingly in frequency. The patient complains of a throbbing, dizzy headache, and vomiting may recur. The bowels remain constipated. The temperature now begins to fall, and the sweating-stage succeeds. Perspiration appears first upon the forehead, face, and neck, and gradually extends over the rest of the body. The perspiration becomes more and more profuse, until the whole body is drenched with it. All the subjective symptoms vanish with wonder-

FIG. 59.



Intermittent fever. Temperature every six hours. Morning and evening temperature and highest at chill.

ful rapidity, and the patient, with the exception of exhaustion, seems to be restored to complete health. The hot stage lasts from one to two

hours, the cold stage from three to eight hours, and the sweating-stage from two to six hours.

In the interval between paroxysms the patient is free from fever, but is anæmic, weak, and has impaired appetite and constipation. During the entire paroxysm the mind remains clear.

The chief *objective symptom*, apart from the phenomena of chill, fever, and sweat already described, is the occurrence of plasmodia in the blood. (See Plate VI., Fig. 2; and Fig. 61.)

Examination of the Blood. The plasmodia of malaria were first pointed out by Laveran. They have been studied in Italy, especially by Marchiafava and Golgi, and in this country by Councilman, Osler, and Dock. Minute amœboid bodies are found in the red corpuscles. These become pigmented with altered hæmoglobin, and grow until they fill nearly the whole of the cell, the pigment being arranged chiefly in a peripheral ring. Later, the amœboid bodies become spherical and transparent, the pigment collecting in the centre. Sporulation now begins and a fresh crop of small, rounded parasites appears, to begin the same cycle over again in fresh corpuscles. (Plate VI., Fig. 2.)

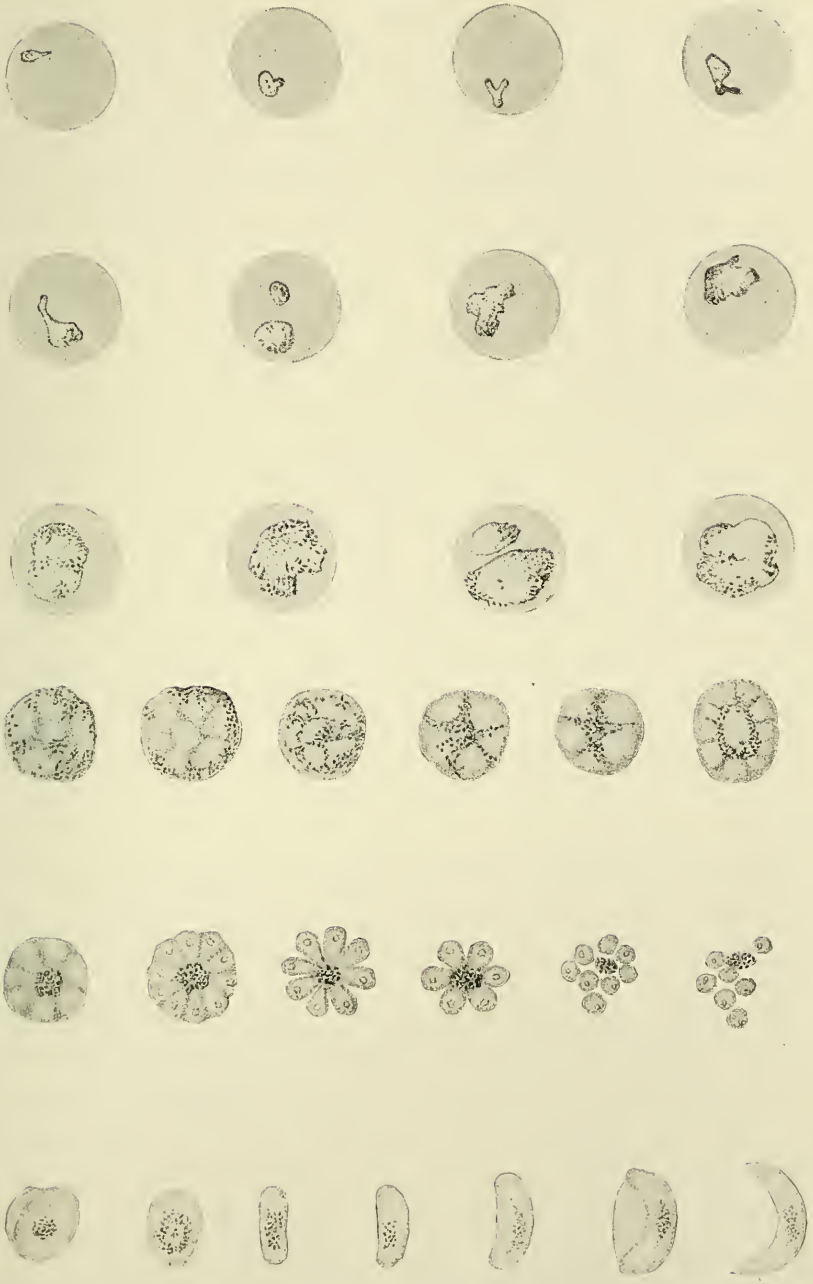
Three forms of parasites are described: 1. The *tertian*, which sporulate at the end of four hours, begin as small amœboid intracorpuscular bodies, gradually enlarge, produce fine brownish pigment-granules, and finally completely fill the corpuscle. In sporulation the segments number fifteen to twenty.

FIG. 60.

Malarial plasmodia. (Reproduced from colored plate.) To the right two normal red blood-cells with central depression. In addition, several others with bluish contained bodies and pigment-sprinkled cells, which show the endogenous development of the plasmodia. Besides, two of Laveran's bodies, one exhibiting a delicate little basket appearance. Near the centre a polynuclear white cell with bluish nuclei and red granulation. (H. RIEDER.)

2. The *quartan*, which sporulate at intervals of seventy-two hours, are smaller; amœboid movement is not so marked; when full grown

FIG. 61.



The first twelve figures show the malarial plasmodium. It is a pale amœboid body inside the red corpuscle. It increases in size at the expense of the corpuscles. In the last four of the twelve it is enlarged and contains pigment-granules derived from the hæmoglobin. The figures of the fourth row show progressive stages in the process of cleavage of the plasmodium and shifting of the pigment-granules. In the fifth row the process of cleavage is seen to be completed, and final isolation of the spores has taken place. The dark granules are pigment-granules. The last row shows oval parasites—Laveran's corpuscles observed in atypical cases of malaria. (From GOLGI, "Studien über Malaria," *Fortschritte der Medicin*, Bd. iv. Tafel., III.)

the parasites are smaller, and the corpuscles tend to shrink about them and to become a deeper greenish color. They sporulate with five to ten segments in a very beautiful characteristic roseate appearance.

3. The æstivo-autumnal are smaller, and contain less pigment. The period of sporulation is still in dispute. They usually form ovoid-crescentic or round bodies with coarse pigment-granules in the centre.

Golgi maintains that in tertian malarial fever the period between invasion of the corpuscles and the sporulation is two days; in quartan, three days, the difference in cycle being due to a difference in the parasites.

The onset of the fever corresponds in time to the division of the parasites.

The crescentic form described by Laveran is said to be more common in the irregular forms of malarial fever. Canalis says that it only makes its appearance several days after the first access of fever. It is somewhat longer than a red blood-cell, and the pigment tends to collect in a focus about the middle of the parasite. Subsequently it becomes oval and divides into eight or more daughter-cells.

Another form with flagella is occasionally found. Councilman says it is most common in blood drawn directly from the spleen.

The plasmodium of malaria may be *stained* as follows: Cover-glass preparations of the blood spread very thinly are dried in the air and fixed by immersion for twenty minutes or half an hour in a mixture of equal parts of alcohol and ether. They are then stained for twenty to thirty minutes in concentrated aqueous solution methylene-blue, 60 parts; ½ per cent. solution eosin in 75 per cent. alcohol, 20 parts; distilled water, 40 parts; 20 per cent. solution potassium hydroxide, 12 drops. The cover-glasses are then washed in water, dried, and are then ready for mounting. The red blood-cells are stained rose, the nuclei of leucocytes a deep dark-blue, and any plasmodia a delicate sky-blue.

Aronson and Phillips' staining method is as follows: Make concentrated aqueous solutions of orange G., acid rubin, and crystallized methyl-green, leave them to settle, then mix in these proportions: Orange G., 55; acid rubin, 50; distilled water, 100; and alcohol, 50. To this add methyl-green, 65; distilled water, 50; and alcohol, 12. Leave the mixture standing for a week. A well-diluted solution should be used for staining purposes; one drop of the mixture should be added to 25 cubic centimetres of water; the stain should be left on for twenty-four hours and the fixing of the preparations carried out at a temperature of 120° C. In the result the red corpuscles are stained orange, nuclei greenish blue, neutrophile corpuscles violet, and eosinophile red.

The examination of the blood discloses the presence of a high degree of anæmia. The hæmoglobin is usually diminished in greater proportion than the corpuscles. There is a marked reduction in the leucocytes. Thus leucopenia is most marked after a paroxysm. There is a relative diminution of the polynuclear forms and a relative increase in the mononuclear forms. In severe post-malarial anæmias, as Thayer points out, the blood is characteristic of pernicious anæmia.

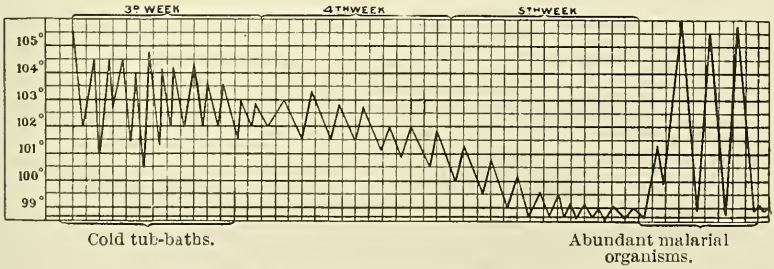
Irregular Form. *Irregular forms* of intermittent fever are more common in Philadelphia than the typical form just described.

In the *mild form* the patient complains of great lassitude, irritability of temper, and drowsiness during the day, but at night tosses upon his bed and gets up in the morning more tired than when he went to bed. The back and limbs ache, and the latter feel as though they would give way under him. There is severe throbbing headache, with some dizziness and faintness. The bowels are constipated; the tongue heavily coated with yellow fur. The temperature is moderately elevated and the patient has great thirst. Nausea and vomiting are absent, though there is little desire for food. There may be a burning feeling referred to the splenic region. The patient is worse on alternate days, and the attacks may be preceded by slight creeping chills. On inquiry the patient will be found to live in a low-lying district near one of the rivers, or in a damp house over an unclean, moist cellar, or adjoining a place where fresh soil has been upturned.

In the form known as "*dumb ague*," there is a periodically great depression, with aching in the head and limbs, a sensation of coldness rather than chilliness, but no marked fever and sweating. Nausea and vomiting may, however, be present. Da Costa says he has seen it manifest itself by excruciating pain over the kidney, and almost entire suppression of urine. There may also be severe paroxysms of gastralgia. It is more common in old residents of malarious districts.

In *masked* malarial fever the poison manifests itself in an attack of neuralgia, especially of the supraorbital nerve and gastric nerves. Malaria may also be latent until some impairment of the resisting power brings it to light. Hence it appears as a complication of pneumonia and dysentery and typhoid fever (Fig. 62), especially in the

FIG. 62.



Malarial fever associated with enteric fever. (THOMPSON.)

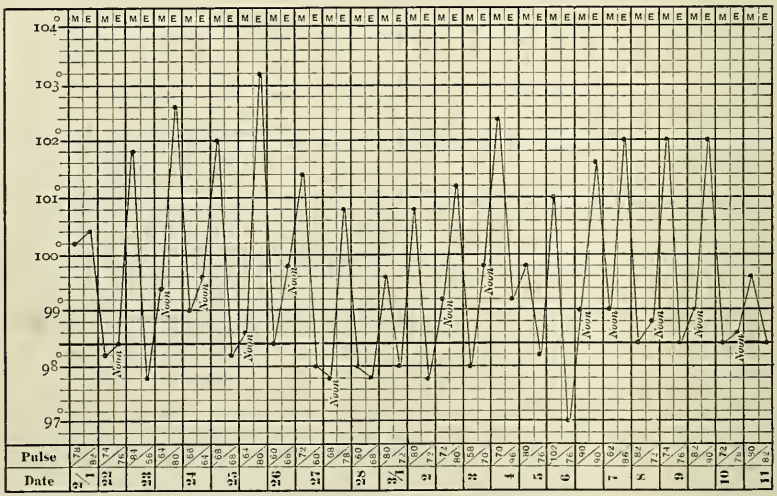
southern and southwestern portions of the United States. Moreover, women who have previously had intermittent fever may suffer a recurrence after confinement.

Diagnosis. The essential points in the diagnosis of intermittent fever are the periodical recurrence of paroxysms of chill, fever, and sweating, or of attacks of dumb ague, or of paroxysms of neuralgia, without organic lesion, associated with the presence in the blood of pigment and plasmodia, and with enlargement of the spleen and possibly of the liver. The so-called therapeutic diagnosis may be made—

an intermittent fever which does not yield to proper doses of quinine in three days is not malarial. A typical malarial intermittent fever is not likely to be mistaken for anything else. (See Fever, pages 205, 206.) It needs, however, to be distinguished from *septicæmic fever*, due to absorption into the blood of pus and the toxins produced by bacteriological growth. Such fever occurs in *tuberculosis*, especially in the stage when cavities form and pus collects; in the puerperal state, in empyema, subphrenic abscess, abscess of the liver, or, indeed, in any form of suppuration. Here also, then, are recurring chills, with fever and sweating, but the attacks are not regularly periodical and intermittent; sometimes the fever is intermittent and sometimes remittent, the chills recur at irregular intervals, and are not so violent as in the malarial attack. The essential difference, however, lies in the fact that a local cause can be found to explain them, tuberculosis either of the lung or of some other viscus, or a collection of pus in an organ or cavity, or a fœtid discharge from the womb, with local tenderness or peritonitis; moreover, the patient loses flesh more or less rapidly, his blood is free from malarial germs and pigment, and quinine does not control the fever. (Plate VI., Fig. 2.)

From the intermittent fever of *hepatic* origin (described elsewhere by the author) the diagnosis is more difficult, in that physical signs of any local trouble may be wanting. But the fever is not regularly intermittent, is not controlled by the quinine, but may be by measures directed to the origin of the trouble, and jaundice may be present.

FIG. 63.



A form of intermittent fever from syphilis. J. D., aged twenty-six years. Secondary period. Mercury and iodide of potassium relieved it. Observe that the pulse-frequency is not increased.

Urethral fever, occurring as the result of operations upon the urethra, or simply from the passage of a catheter or bougie, may be mistaken for malarial fever; but the paroxysm is usually single, and the history

of the operation and the absence of plasmodia from the blood clear up the diagnosis.

Syphilitic fever is distinguished by a tendency for the chill, fever, and sweating to be nocturnal in recurrence, and by evidence of a syphilitic infection coupled with absence of malarial germs from the blood.

Remittent Malarial Fever. Æstiva-autumnal Types. A type of malarial fever characterized by a remission instead of an intermission in the febrile paroxysms. It is due either to a greater intensity of the malarial poison or to a different species of organism. It is much more rare in temperate climates than either quotidian or tertian intermittent, and is attended with more gastric disturbance and a much larger mortality (twelve times greater, according to the statistics of the civil war).

The *onset* is more abrupt than in intermittent fever. Prodromata are not so common, but when they occur they are of the same character. The chill is not usually so violent, nor the cold stage so long as in intermittent fever; on the other hand, nausea and vomiting are common, and in some cases there are bilious vomiting and diarrhœa, tenderness over the stomach and spleen, and sometimes jaundice. The temperature rises rapidly from 103° to 106°, and remains high for a longer time than in intermittent fever, the hot stage lasting in severe cases from six to eighteen or twenty hours.

During this time the patient suffers from headache, pains in the back and limbs, great thirst, and gastric irritability. A remission now succeeds. The temperature falls two or three degrees, but not to normal; free sweating occurs, the nausea and vomiting cease, and the patient becomes much more comfortable. He may fall asleep from exhaustion, but if awake is conscious of weakness, aching in the limbs, and perhaps nausea. In the course of some hours the temperature again rises, often to a higher point than before, but frequently without antecedent chill. The same subjective symptoms are repeated, and another remission follows. Daily paroxysms usually occur, those on alternate days being severe. The temperature often reaches its highest point at the third paroxysm. The disease generally runs its course in from nine to twelve days, but it may last much longer. The type of fever may change to intermittent, which is a favorable sign, or become continued and again remittent, or remain remittent throughout; finally, the fever may subside gradually, or, less commonly, by crisis. The urine is febrile but not albuminous. (See Examination of Blood, page 282.)

Pernicious Malarial Fever. This, as the name implies, is a form of malarial fever with destructive tendency. It is also called malignant and congestive fever. It may be intermittent or remittent. Nearly 24 per cent. of the cases occurring in the U. S. Army from May 1, 1860, to June 20, 1866, proved fatal.

Bemiss[1] divides it into three classes: the algid, or congestive, form; (2) the comatose form; (3) the hemorrhagic form. To this another class, (4) the gastro-enteric form, may be added. It is important to remark that the first paroxysm does not usually, in any of these forms,

[1] Pepper's System of Medicine, 1885, vol. i. 666.

indicate that the type of the disease is pernicious. The first seizure may, however, prove fatal.

1. The *algid form*, according to Bemiss, occurs more frequently than any other, its perniciousness being due to an aggravation of the cold stage of an intermittent attack. The patient is extremely weak, with cold extremities, pinched features, blue lips, and faint voice. Respiration is shallow, the pulse rather slow, feeble, and irregular; he is further exhausted by vomiting and liquid, offensive diarrhœa, the passages sometimes being involuntary. There may be copious perspiration, but the internal temperature is very high. The mind may be clear, or there may be deep stupor. Unless speedy relief can be afforded the attack ends fatally.

2. In the *comatose form* the patient is completely unconscious, the skin hot "and of a muddy, semi-jaundiced hue" (Bemiss). Both pulse and temperature are increased. In other cases coma is preceded by wild delirium, resembling acute meningitis.

The comatose form is most apt to occur in those who continue to reside in a malarious region without proper safeguards against its poisonous influences.

3. In the *hemorrhagic form* there has been, as a rule, previous alteration of the blood, the bloodvessels, and other tissues, by long-continued malarial poisoning or cachexia. Then, when intense congestion of these parts occurs as the result of the surface-chill, hemorrhage follows. In some districts, however, and at certain seasons, there has been a special predilection of the poison for the kidney, with resulting hæmaturia. The prominent symptoms are a prolonged chill with high temperature; nausea and vomiting, sometimes with the expulsion of a greenish-black fluid; œdema of the lower extremities; general anasarca and occasionally œdema of the lungs, and hydrothorax; bloody and albuminous urine, with tube-casts; and intense jaundice. Pain in the right hypochondrium or over the kidneys is common.

Bemiss asserts that uncomplicated malarial fever has not a hemorrhagic tendency.

4. The *gastro-enteric form* has for its prominent symptoms nausea, vomiting, diarrhœa, intense thirst, extreme restlessness, a frequent, feeble pulse, and urgent dyspnœa. "The breathing is deep-drawn; to each expiration succeed two respirations" (Da Costa). The patient is cold and partly collapsed. Reaction may or may not occur.

The patient may have several paroxysms of pernicious malarial fever and succumb in any one of them. Convalescence is slow. The most frequent sequelæ of malarial fevers are anæmia, neuritis, and paralyses, and malarial cachexia.

Typhoid fever is distinguished from pernicious malarial fever by its gradual onset, the absence of chills and vomiting, as a rule, and, on the other hand, the presence of epistaxis, delirium, and ataxic symptoms, tympanites and diarrhœa, with pale-yellow watery stools, and rose-colored spots. The temperature in typhoid is more continuously high, the daily oscillations being of shorter range. A history of exposure to malarial infection and of previous attacks can often be obtained. The urine of typhoid exhibits the diazo reaction; malarial fever does not.

Malarial cachexia occurs especially in those who have lived for a long time in malarious regions. They may or may not have had typical malarial attacks. The patient suffers with dyspepsia and constipation, with occasional bilious attacks; the face is of a pale lemon-yellow color, and may be slightly jaundiced; there is marked anæmia, with pigment and crescentic and flagellate forms of plasmodia in the blood, together with great enlargement of the spleen (ague-cake) and some enlargement of the liver. The patient is weak and languid, and sometimes has considerable mental depression.

Serum Diagnosis.

The infections just described are recognized by an examination of fresh blood or cover-slip preparations. The next group of infections may be recognized by serum diagnosis. Too much stress must not be placed upon this method of diagnosis, yet its value is so great that one is fully justified in giving it a high place in the precise method of diagnosis of infections.

Typhoid Fever.

The first of the infections to which such diagnosis has been applied *in extenso* is typhoid infection or typhoid septicæmia. This infection is caused by the bacillus typhosus. The most common expression of it is seen in a symptom complex which attends a septic process and local intestinal ulceration, which symptom complex we know as *typhoid fever*. This infection, it is stated by some, is unattended in rare instances by fever. More frequently a febrile course, following a definite continued type of a duration of from twenty-one to twenty-eight days, prevails. In mild or abortive forms fever rarely reaches 103°, and declines from the seventh to the fourteenth day. In the grave forms the fever is often very high and attended by cerebro-spinal, renal, pulmonic, or severe gastro-intestinal symptoms.

The most important infection prevailing in the temperate zone is the one we are now about to consider. It is an acute, specific, infectious, and mildly contagious fever, characterized by a gradual onset, a continued fever, an eruption of rose-colored spots, marked nervous and abdominal symptoms, and an average duration of three or four weeks.

It occurs sporadically and epidemically, and in large cities is apt to be epidemic. Its special habitat is in temperate climates, but it may occur anywhere. It is relatively rare in the southern and southwestern portions of the United States. It is more frequent in the latter part of the summer and in the autumn and winter, and following hot and dry summers. Young adults are especially prone to it, but cases have occurred at all ages. Change of residence from the country to the city predisposes to it. Those living in cities often acquire immunity, but they may lose it upon moving elsewhere. The state of previous health does not seem to have any influence.

The period of *incubation* in typhoid fever varies from four or five days to three weeks; more commonly it is from one to two weeks. During this time the patient usually is languid, becomes tired easily

upon exertion, has severe headache, sleeps poorly, and has bad dreams. There is often, even thus early, a dull and listless expression of the face. Toward the close of this period, and in severe cases, there may be colicky pain in the abdomen, a tendency to looseness of the bowels, cough, epistaxis, mental sluggishness, and chilliness. Dr. Pepper says he has been led repeatedly to anticipate the approach of typhoid fever by the unusual dulness of hearing and by the persistent occipital headache coming on after a few days of general malaise.

While the disease may begin abruptly, a gradual onset is so much the rule that it becomes important in the diagnosis from other disease-conditions.

Invasion is not sharply marked. There may be chilliness, but a decided chill is unusual except when pneumonia is part of the initial process. Muscular weakness, headache, and mental sluggishness are more pronounced, and the physician is consulted because these symptoms persist, or because fever is discovered. The beginning of fever is the most constant indication of the onset of the disease, and two very important early symptoms are cough from bronchitis and enlargement of the spleen.

The most prominent and constant subjective symptom during the first week is headache. Other very common symptoms are tenderness, rarely pain, in the iliac region, more or less prostration, and impaired appetite or loss of appetite.

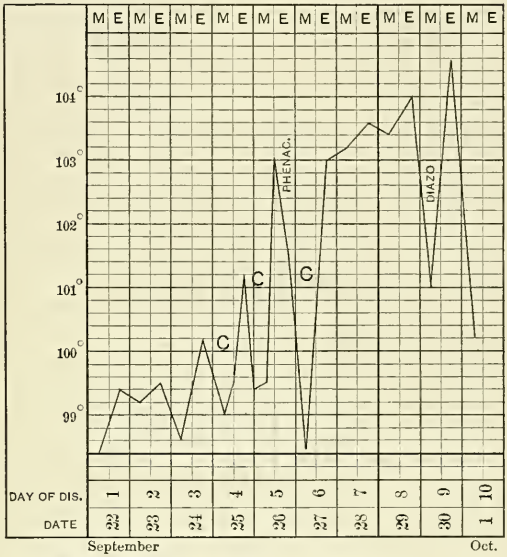
The *objective* symptoms are therefore the most important. The face is pale rather than flushed, and has a dull, listless, apathetic expression. The tongue is heavily coated with a white fur which later becomes yellow. The abdomen is somewhat distended and tympanitic on percussion. There is usually tenderness in the right iliac region, and gurgling upon palpation is pretty constant. Constipation may be present at first, and sometimes persists throughout the disease. A tendency to diarrhœa is, however, characteristic of the disease. Even if constipation exists at first, a laxative is apt to produce an excessive effect. The number of stools varies from two or three to a dozen or more in twenty-four hours. They are light yellow in color (resembling pea-soup), thin, watery, and offensive. The movements are not usually attended with pain, and in severe cases may occur involuntarily.

Enlargement of the spleen is a very constant symptom. It may be detected at the onset, increases up to the height of the fever, subsides during convalescence, but recurs during a relapse. It covers a percussion-area in the left hypochondrium of four to eight finger-breadths.

The *temperature*-curve, when not modified by treatment, shows a gradual ascent during the first four or five days of the disease, with morning remissions. The temperature rises a degree or two in the evening and falls half a degree or a degree in the morning. This "step-ladder" ascent is very characteristic. By the end of a week a temperature of 103°, 104°, or 105° has been reached, and it remains continuously high, with slight morning remissions, during the second and less frequently during the third week. In the third or fourth week the morning fall of temperature gradually becomes greater, and by the end of the week sinks below the normal in the morning.

The temperature in mild cases may never rise above 103° at any time, and most of the time varies between 100° and 102°. Or it may

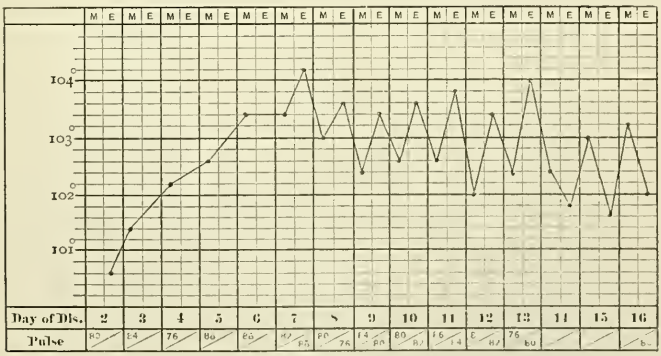
FIG. 64.



Temperature ranges; first week of typhoid fever. (DOCK.)

be 104° from the start; more frequently during the second and third weeks there are marked oscillations of the temperature—a sudden fall from 104° to 101°, or a rise from 103° to 105° or 106°. Hyperpyrexia is a temperature above 105°.

FIG. 65.



Mild typhoid fever. Gradual ascent.

The *pulse* is full, and in favorable cases slower than the pyrexia would lead one to expect. It is more frequently under 110 than over 120. In the second week it is markedly dicrotic.

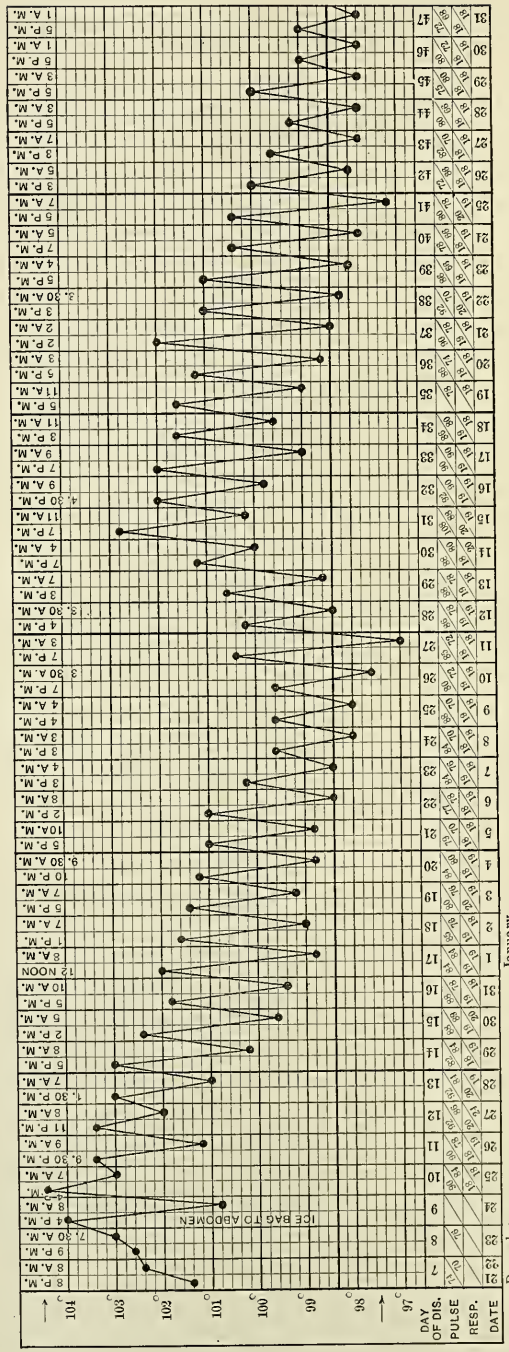
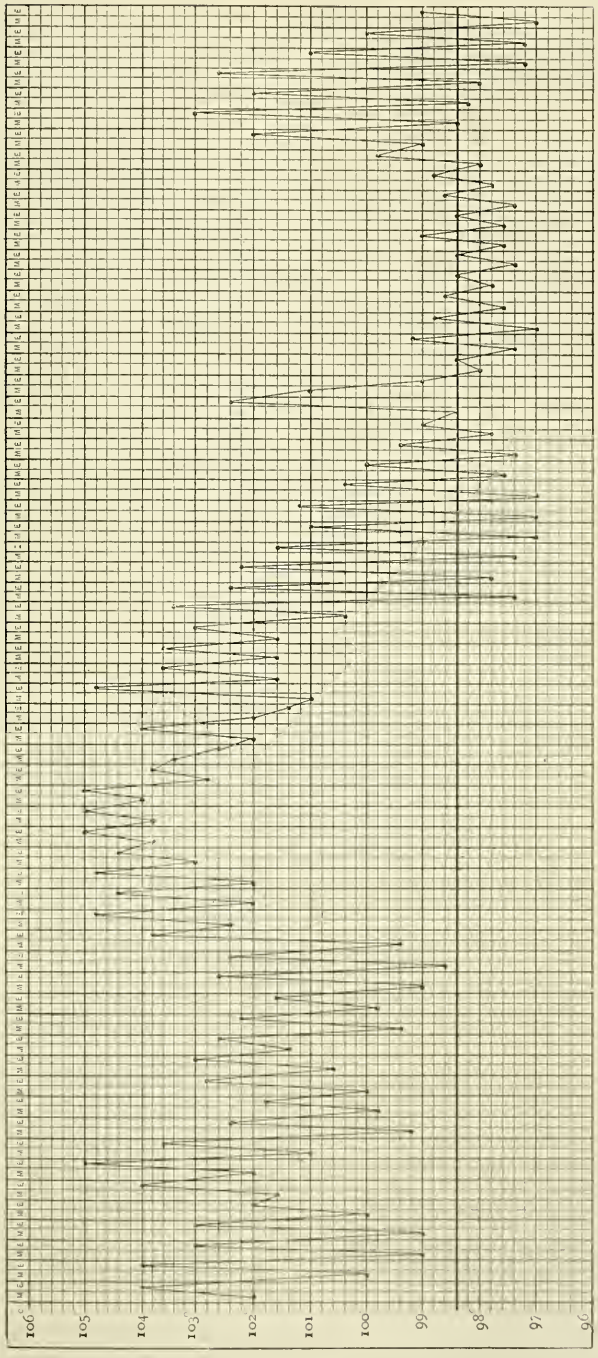


FIG. 66.

Typical chart, mild typhoid fever with relapse.

FIG. 67.



Protracted typhoid fever, not modified by antipyretics. Aberrant temperature during the first twenty days. Duration, 63 days. Recrudescence on the fifty-fifth day.

The *heart sounds* are unchanged apart from complications, but in the second and third weeks the first sounds often are feeble, indicating heart weakness. A pulse of 120 or more is a graver sign in typhoid fever than in other diseases. Therefore, when it becomes very frequent and feeble, the extremities cool and the lips bluish, the outlook is gloomy.

The *urine* is at first scanty and high-colored. A slight degree of febrile albuminuria is not uncommon, and in rare cases the whole force of the poison seems to be spent upon the kidneys, the urine containing, besides the usual blood and casts, biliary coloring-matter. In conditions bordering on coma the patient may have retention of urine, or, on the other hand, he may pass it involuntarily. To obtain the *diazo reaction* of Ehrlich two solutions are necessary. The first (*a*) consists of 2 grams of sulphanilic acid, 50 c.c. hydrochloric acid, and distilled water 1000 c.c. The second (*b*) consists of a $\frac{1}{2}$ per cent. solution of sodium nitrite. These solutions are kept in separate bottles. Fifty parts of solution *a* and one part of solution *b* are poured into a test-tube and an equal volume of urine added. The test-solutions and urine are now thoroughly shaken and then carefully overlaid with 1 c.c. of ammonia. At the junction of the two a pink or ruby ring develops. Upon agitation the foam on the top of the mixture is also colored red. Normal urine gives a light brown ring. This reaction is helpful in diagnosis, but may occur in acute phthisis, tubercular meningitis, and other diseases. According to Pepper, it is rarely absent in measles. The reaction is fairly constant in typhoid fever after the first week.

The *respiration* in uncomplicated cases increases in frequency with the rise in temperature. It usually ranges between 24 and 36. The slight bronchitis present in the beginning in most cases causes no trouble ; sometimes it lasts throughout and contributes to the tendency to hypostatic congestion, which is always present. The physical signs are those described elsewhere in these conditions.

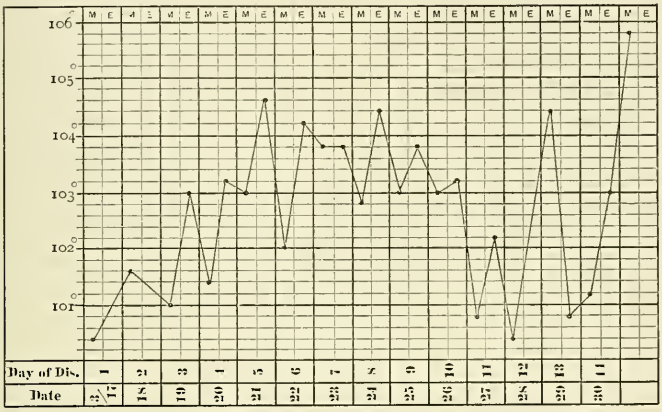
The *nervous symptoms* are often very prominent. In mild cases they consist of hebetude and nocturnal delirium, or they may be absent altogether. Usually, however, by the beginning of the second week, there is some mental confusion, with nocturnal delirium. In more severe cases, and later in the disease, the delirium is of a low, muttering character, with hallucinations of sight and sound more or less continuous. The patient can be roused by a question, and makes an intelligent answer, but speedily lapses into semi-consciousness. Picking at the bedclothes or efforts to catch imaginary objects are very common. Sometimes the delirium is wild and noisy, and the constant presence of some one is needed to keep the patient from getting out of bed. Patients have jumped out of windows, or run long distances before being captured. Rarely the delirium has been so active as to simulate acute mania. Stupor may alternate with delirium. Rarely the patient lies with wide-open eyes, apparently staring fixedly at some object, but really unconscious (coma-vigil).

In ataxic cases the patient has marked twitching of the tendons and jactitation. He is wakeful and restless, wearing himself out. The

hands and lips tremble, and he keeps muttering to himself all the time.

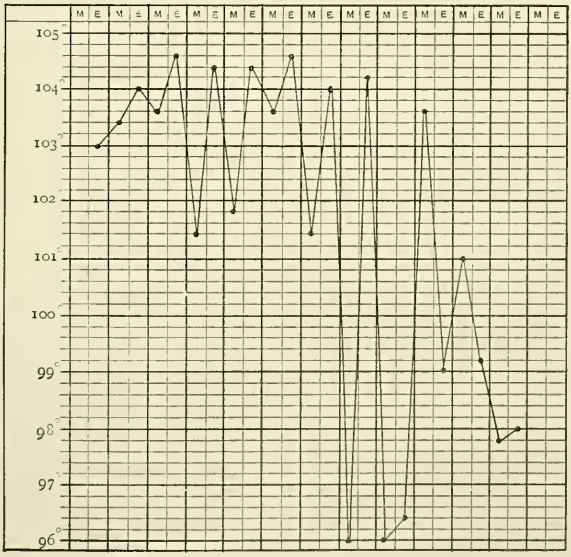
Convulsions are rare, but may occur in children. Sometimes there are considerable hyperæsthesia and tenderness along the spine.

FIG. 68.



Grave typhoid fever. Death. M., aged 22 years. Ataxic symptoms.

FIG. 69.



Typhoid fever in a child aged 12 years. Chart from twelfth to twenty-third day. Repeated crises. (Frequent mode of termination in children.)

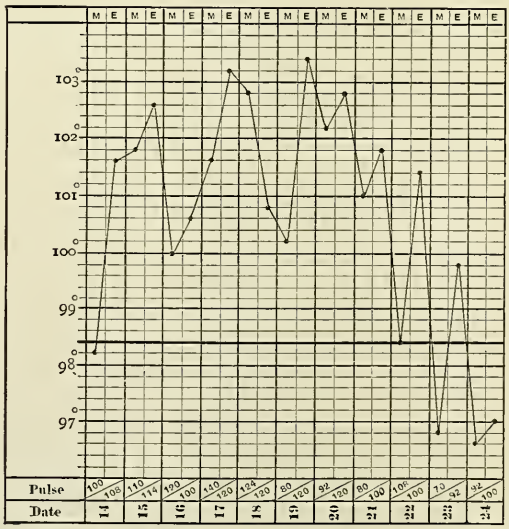
The extent of the nervous symptoms depends upon the habit of the patient as well as upon the height of the temperature and gravity of

the disease. In children and neurotic individuals they may be pronounced, with only moderate fever.

On the seventh or eight day the *eruption* appears. It consists of small, very slightly elevated, rose-colored papules, which disappear upon pressure and come out in successive crops, each papule lasting three or four days. The spots are most common over the abdomen and back, but are occasionally found elsewhere. They are usually few in number, a half-dozen or dozen, but sometimes the eruption is very copious, especially in severe cases. Sometimes it is wholly absent.

During the latter part of the second week, and through the third week, the symptoms are apt to be intensified. The temperature keeps up or even reaches a higher point. Delirium is more decided and constant. The heart grows weak and the pulse increases in frequency. Some degree of hypostatic congestion of the lungs is usual. Diarrhœa may be troublesome; intestinal hemorrhages, announced by sudden fall of temperature and symptoms of collapse, may occur. Tympanites may become so great as to interfere with respiration and circulation. This is the period when ulceration of Peyer's patches in the intestine is deepest, and when perforation is imminent. There is rarely any desire for food, though it is taken and assimilated. Nausea and vomiting are rare. The tongue is dry, brown, sometimes glazed and fissured, and sordes often collect on the teeth.

FIG. 70.



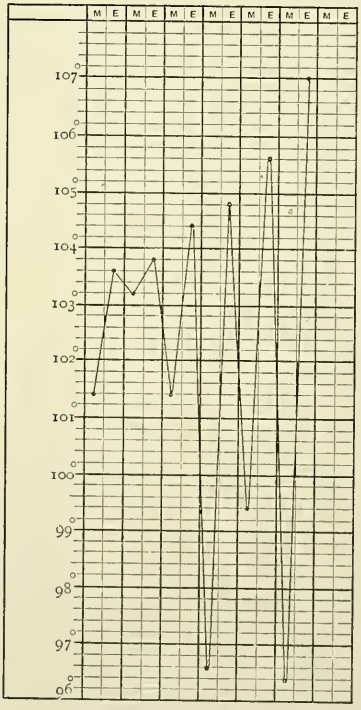
Course of temperature in a relapse beginning on the twenty-sixth day. First attack mild.

In cases ending in recovery the temperature begins to fall in the mornings; delirium grows less; sleep is more refreshing. Diarrhœa ceases, and constipation may even require treatment. The pulse does not usually improve as rapidly as the other symptoms. There is sometimes very marked anæmia without leucocytosis (Osler). When the

temperature sinks to normal or subnormal, convalescence has set in. This is very rapid as far as digestive symptoms are concerned, but strength returns very slowly. It may be interrupted by a relapse, in which the original symptoms are reproduced, with high temperature, but the duration is shorter.

Varieties. It is now well known, as Osler forcibly states, "that typhoid fever is no more primarily intestinal than is smallpox primarily a cutaneous disease." Studies in bacteriology, promoted especially by Chiari, Flexner, Kraus, Nicholls, and others, enables us to divide the infection into three varieties: 1. Typhoid fever with intestinal lesions, as described above. 2. Typhoid fever with general infection or typhoid septicæmia. The symptoms are entirely those of an infection, and the diagnosis must rest upon the serum reaction and culture methods. 3. Typhoid fever with more intense infection of other organs than the intestines. The lungs, the spleen, the kidneys, and the cerebro-spinal meninges are the structures invaded, so that we may have a pneumo-, nephro-, spleno-, or cerebro-spinal typhoid.

FIG. 71.



Grave typhoid fever. Daily rigors. Death on nineteenth day. No complications.

Varieties are also based upon the severity of the disease, hence we have the abortive, grave, and ambulatory forms.

The abortive form is so named because of the abbreviated course of

the disease. The symptoms are sufficiently well marked to make the diagnosis clear, but the type is mild, and in a week or two convalescence is established. In rare instances an afebrile form with intestinal symptoms and eruption is seen.

In the ambulatory form, commonly called "walking typhoid," the patient, from ignorance of the gravity of his ailment or from apparent necessity, keeps at his work until weakness and incessant headache lead him to consult a physician in his office or at a dispensary. He may then be well into the second week of the disease. The majority of such cases prove fatal.

Grave forms are due to especial severity of some symptoms or group of symptoms, such as hyperpyrexia; profound stupor, coma, or intense ataxia; inability to take or retain sufficient nourishment; profuse diarrhœa and intestinal hemorrhage; great adynamia with weak heart and a tendency to cyanosis. In other cases the gravity results from the existence of complications.

In the malignant form there has been a large dose of the poison or a very weak organism, or both, the result being an acute toxæmia; this is not so common as in scarlatina and typhus fever.

In the pulmonary form the onset may be so obscured by severe bronchitis or lobar pneumonia that the primary disease is not suspected at first. Severe bronchitis seems to be more common in children. Chill and initial high temperature are common in these cases.

Typhoid Fever without Intestinal Lesions. This rare form may present the clinical symptoms of typical typhoid, or may be of splenotyphoid type, or of nervous type with extreme intoxication. The first type is rare. The second type, described by Eiselt, is characterized by an excessively large spleen, with local inflammation and remitting fever. In the third class the symptoms of the typhoid state with subcutaneous and visceral hemorrhage occur. Jaundice is more or less common.

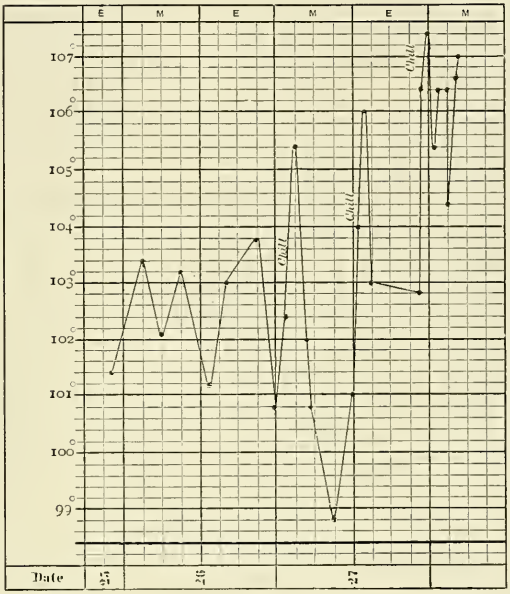
Complications and Sequelæ. Typhoid fever may be accompanied by a number of complications, the most frequent and important being severe bronchitis, hypostatic congestion with œdema, and true lobar pneumonia; bed-sores; parotitis; phlebitis, especially of the femoral vein; peritonitis from perforation of the bowel; meningitis, acute mania, or mental decay; jaundice; myocarditis; periostitis and osteitis. Sequelæ are not frequent. Sometimes, however, the foundation is laid for permanent ill health. There may be impairment of the senses, mental weakness, and even insanity. Paralyses, neuritis, hyperæsthesia, chorea, and epilepsy are occasional sequels.

Examination of the Blood. The infection is due to Eberth's bacillus, the bacillus typhosus. The bacillus is found in colonies in the spleen, liver, mesenteric glands, kidneys, and intestines. It is also found in the feces and rarely in the urine. It may be seen in the blood. It may be recognized by staining methods, although rarely. It has been isolated from the blood successfully, by culture methods, by Gwyn in a small number of cases.

Morphology. A bacillus 1 to 3μ long by 0.5 to 0.8μ broad, with rounded ends. It is motile, facultative anaërobic, does not liquefy

gelatin. It has flagella 3 to 5 times as long as the bacilli. It stains with the anilines, best with Löffler's blue. The flagella are stained by Löffler's special method. (See Plate III., Fig. 6, B.)

FIG. 72.



Renal typhoid. Nephritis on the twenty-fifth day. Course of temperature during three days preceding death.

SERUM DIAGNOSIS. This method of diagnosis has been more successfully employed in typhoid fever than in any other infection. The methods have been previously described. The agglutinative reaction takes place as early as the eighth day, rarely as early as the third day, but sometimes not until the fifteenth or twentieth day, and even may not occur until convalescence is established. By this means typhoid fever can be distinguished from the infection due to the bacillus of Gärtner (bacillus enteritidis). (See *Lancet*, January 15, 1898.)

The paracolon bacillus infection, as shown by Gwyn (*Bulletin of the Johns Hopkins Hospital*, 1898, vol. ix. No. 84), who studied a case which resembled typhoid clinically, does not give this reaction. Influenza and Malta fever and forms of tuberculosis can also be distinguished from typhoid fever by this method.

LEUCOCYTOSIS. A determination of the number of leucocytes is of value in the diagnosis of typhoid fever. It is one of the infections in which leucocytosis does not occur. In a differential count some variation from the normal is seen. The large mononuclear and transitional forms are decreased; the polynuclear neutrophiles are decreased. The absence of leucocytosis aids in distinguishing typhoid fever from various septic fevers and acute inflammations. On the other hand, in a

case of typhoid fever if leucocytosis occurs an inflammatory complication or mixed infection is possible. Perforation and peritonitis are attended by leucocytosis.

In addition to the absence of leucocytosis we find, after the second or third week, gradual reduction of the red cells, and by the time convalescence is established a marked anæmia develops. Both the red cells and the hæmoglobin are reduced.

Culture Methods. The bacillus can be isolated from the blood, the stools, and the urine.

Biological Properties. The bacillus grows readily in acid media as well as in the neutral or alkaline media, best at a temperature of 38° C. Death-point, 60° C.

The colonies develop in twenty-four to forty-eight hours. On gelatin plates they are small and white, nearly spherical ; irregular, granular, and yellowish-brown.

In stab-cultures there is a whitish semi-transparent layer on the surface, with sharply defined irregular edges, and along the puncture a grayish-white growth. (See Plate VII., Fig. 5.) It develops abundantly in milk. On potato it forms an "invisible growth," manifested only by increase in moisture, which is quite characteristic.

Bacteriological Diagnosis. It would be most desirable if a means of diagnosis, that would have no element of uncertainty about it, could be found. Bacteriologists have sought for such means, and at present seem to have found two methods, one of which at least has been brought to such a degree of perfection as to be of value to the clinician. They are Elsner's culture and Pfeiffer's bactericidal serum methods. Elsner's method [1] consists in the preparation of a culture-medium upon which no species of micro-organism can grow except the typhoid bacillus and the bacillus coli communis. For a description of this complicated method the reader is referred to the recent works on bacteriology.

Recently bacteriologists have been successful in isolating the typhoid bacillus from the stools and the urine. Unfortunately, the methods are too complicated for clinical work. P. H. His, Jr., recovered the bacillus typhosus and distinguished it from members of the colon group by a combined plate and tube method.[2]

For differentiation of the typhoid from the colon bacillus the method of Proskauer and Capaldi may be used. They employ two solutions. In solution No. 1 the typhoid bacillus does not grow at all. The colon bacillus grows rapidly, produces a marked acid reaction, and the blue color gives way to red. Solution No. 2 both bacilli grow, but the typhoid bacillus is the only one which gives an acid reaction. Note, the solutions are neutral in reaction and colored with litmus.

Another method is that of Thoinot. He prepares a medium of bouillon, to which he adds $\frac{1}{100}$ per cent. of arsenious acid. On it the typhoid bacilli do not grow, while the colon bacilli multiply rapidly.

[1] Zeit. Hygien. und Infection kr., B. xxi. H. 1.

[2] P. H. His, Jr., "On a Method of Isolating and Identifying Bacillus Typhosus," etc. Journal of Experimental Medicine, vol. ii. No. 6, p. 677.

PLATE VII.

Fig. 1.

Streptococcus—Erysipelas.

Fig. 2.

Streptococcus Septicus.

Fig. 3.

Staphylococcus.

Fig. 4.

Diphtheria-bacilli.

Fig. 5.

Typhoid-bacilli.

Fig. 6.

Tuberculosis-bacilli.

Mark Richardson isolated bacilli in the urine of about 25 per cent. of the cases of typhoid examined. They were present in large numbers and in pure culture. They appeared late in the disease, and persisted into convalescence. The bacilli were always associated with albumin and casts. After disinfection of the meatus the urine is passed in two portions into sterilized test-tubes. The second portion is used. It is immediately plated upon plain agar. At the end of twenty-four hours the characteristic colonies appear. Richardson relies upon the active motility of the bacilli, which are set free in a typhoid colony by scarring with a platinum needle to distinguish them from the colon bacilli. He also used the dry serum reaction test.[1]

Pfeiffer's method, while of interest and full of suggestions as to its future usefulness, cannot be applied with sufficient ease to render it practical for clinical work.

Inoculation. Thus far the results of inoculation have not proved satisfactory, and are certainly not of diagnostic value.

Diagnosis. A typical case of typhoid fever ought not to be mistaken for any other affection, but atypical cases are numerous. The most common sources of error are a hurried diagnosis and a willingness to accept a demonstrable local affection as sufficient to account for the condition. In this way the significance of bronchitis, pneumonia, and diarrhœa is overlooked. In the symptomatic form there will almost always be found a history of gradual onset and a degree of fever and prostration greater than should attend the purely local affection. Moreover, in bronchitis and pneumonia, which are a part of typhoid fever, there may be found tenderness with gurgling in the right iliac region, enlargement of the spleen, and epistaxis, to aid in the diagnosis; while in cases in which the diarrhœa leads to uncertainty, bronchitis, enlargement of the spleen, and epistaxis may coexist. Examination of the blood, extended over a period of several days, is necessary to exclude the *æstivo-autumnal* type of malarial fever, which often resembles typhoid fever.

New Diagnostic Sign of Typhoid Fever. Dr. Simon Baruch writes as follows: "As soon as the patient shows a rectal temperature above 102.5° in the morning and 103° in the evening for three successive days, especially if this be accompanied by headache, dulness, or apathy, he is placed in a full bath at 90°, which is reduced to 80°, with constant friction over the body. In three hours, the temperature still being above 102.5°, he receives another bath 5° cooler. This is repeated until the temperature of the bath is 75°. If one or more of these baths fail to reduce the rectal temperature 2° in half an hour, the diagnosis of typhoid fever is almost certain, and the bath-treatment is continued. The resistance of the rectal temperature to a bath of 75° for fifteen minutes, with friction, is an almost certain test of typhoid fever."[2] Dr. Baruch considers that the diagnosis of this disease should no longer be obscure, even in the first days of its course.

[1] Richardson, M. W., "On the Presence of the Typhoid Bacillus in the Urine." Journal of Experimental Medicine, vol. iii. No. 3, p. 349.

[2] New York Medical Journal, September 2, 1893.

Appendicitis is more likely to be mistaken for typhoid fever than the converse. There is usually a history of constipation, though the occurrence of several inadequate movements a day may conceal the fact that there is a fæcal accumulation. In appendicitis the onset is more abrupt and the local symptoms more pronounced than in typhoid. Pain and tenderness are prominent in appendicitis, and while they may be general over the abdomen at first, they are found to be more acute in the iliac region and loin. Here, in place of gurgling, we find some increase of resistance on palpation, and a relatively dull note—a wooden sort of tympany—or there may be a demonstrable tumor. The patient lies with the right leg drawn up, has moderate fever, and vomiting. In fact, the attack is often introduced by chilliness and vomiting. Headache is not a prominent symptom, while bronchitis and enlargement of the spleen are absent.

Acute right-sided *salpingitis* simulates typhoid fever. It is distinguished by the history of a preceding vaginitis, endometritis, or abortion, by the absence of diarrhœa, of enlargement of the spleen, and of the characteristic eruption. A digital examination through the vagina discovers the womb pressed to one side and fixed, and a tender mass blocking up the pelvis.

Simple continued fever is distinguished from typhoid fever of a mild type principally by the absence of bronchitis, of enlargement of the spleen, of epistaxis, and of the characteristic eruption of typhoid fever. In simple continued fever constipation is more common than looseness of the bowels, and gurgling is absent.

Typhus fever is distinguished by its sudden onset, the besotted expression of the face, with reddened eyelids and small pupils, the absence of abdominal symptoms, and the occurrence on the fourth day of maculæ, which are subsequently converted into petechiæ. It is of shorter duration, and terminates very abruptly by crisis.

Relapsing fever differs from typhoid fever in its sudden onset with chill, pain in the epigastrium, but absence of abdominal symptoms and eruption ; in the absence of marked nervous symptoms, in spite of the high fever ; the short duration and termination by crisis, and the characteristic relapse at the end of a week. The conclusive test is finding spirilla in the blood.

Acute tuberculosis of the lungs, at times, closely resembles typhoid fever. In both the onset is gradual, with cough and fever. In the former, however, the bronchial symptoms are more prominent, there are apt to be recurring chills and sweats, the temperature is remittent and irregular, emaciation is rapid, and constipation instead of diarrhœa is the rule.

In *peritoneal tuberculosis* there is persistent diffused pain in the abdomen ; the belly is swollen. If effusion occurs, the physical signs disclose its presence. The temperature is irregular and may be below normal ; nervous symptoms comparable to those of typhoid are wanting.

Meningitis before the stage of effusion exhibits exaggeration of the reflexes and marked hyperæsthesia. There may also be muscular rigidity. The patient is restless, easily annoyed, and " fussy " about

things that would be unnoticed by a typhoid patient. Vomiting is often present, whereas it is rare in typhoid fever. The temperature does not maintain so high an average range as in typhoid fever, and is subject to greater oscillations. The pulse varies greatly, and may be irregular.

In *septic meningitis* the headache and vomiting are more persistent, the bowels are confined, and the abdominal walls are retracted. There may be double optic neuritis. In *tubercular meningitis* the knee-jerk and other reflexes are variable, irregularly absent or present. In typhoid fever they are always present. In the former choroidal tubercles may be seen with the ophthalmoscope. In *tuberculosis* in all forms leucocytosis is present; in typhoid it is absent. Typhoid fever must not be confounded with *trichiniosis*; the peculiar muscular pain and œdema do not occur in the former. *Uræmia* may simulate typhoid fever when it becomes chronic; but the age, the character of the urine, the cardio-vascular symptoms, are diagnostic, and, with the absence of the specific typhoid symptoms, render the diagnosis easy.

Mountain Fever is an infection which has been described as peculiar to the mountains of our Western States, characterized by a continued fever with intestinal symptoms not unlike those of typhoid fever. Irregularity of the temperature-range and the occurrence usually of constipation rather than diarrhœa make it difficult to classify the infection from typhoid fever on the one hand and from forms of malaria on the other. Recent observations of Woodruff, who studied the serum reaction in a large series of cases, show conclusively that the infection is typhoid fever, confirming the prior observations of Hoff, Smart, and Raymond.

Yellow Fever.

The infection which we are about to consider is the latest of the epidemic and contagious disorders for which a definite causal micro-organism has been discovered. It is an acute, specific, contagious, miasmatic disease, endemic and epidemic on the tropical and subtropical shores of the Atlantic Ocean, characterized by a sudden onset, a duration of a week or less, a characteristic facies, a fall in the pulse-rate preceding a fall in temperature, and by albuminuria, jaundice, and vomiting, with a tendency to hemorrhages. The specific micro-organism is the bacillus icteroides described by Sanarelli.

Yellow fever is endemic in Havana and other seaport cities of Cuba, and in Rio Janeiro, Brazil. From these centres it is liable to become epidemic, and to be carried in ships and by persons and clothing to other places. In this way epidemics have developed in the seaports of the United States, especially in the south around the Gulf of Mexico, but sometimes as far north as Philadelphia and New York. The disease becomes epidemic in the hot season and ceases upon the appearance of frost. The specific germ has not yet been isolated.

In countries in which the disease is endemic it is the custom to regard the native children as immune. Dr. John Guitéras, however, is strongly of the opinion that the disease is kept alive between epidemics by cases among these children. He has also shown that it

prevails among white children before it becomes epidemic among adults.

The period of *incubation* varies from a few hours to two weeks. Guitéras states that the cases in which it extends beyond the seventh day are exceptional.

The *invasion* is abrupt, and occurs usually in the night. It is marked by chilliness oftener than by a decided chill. The temperature rises rapidly to 102° to 103° or 104°, not often higher in favorable cases. The *pulse* is correspondingly increased in frequency at first, but very commonly begins to fall before the temperature, so that later the pulse is relatively slow. The *face* is peculiar and characteristic—it is flushed and somewhat swollen; the eyelids are somewhat swollen, with reddened edges; the eyes are watery, glistening, and slightly but distinctly tinged with *yellow*; the pupil is small and brilliant. Guitéras says:[1] "The appearance of the face is often sufficiently characteristic on the first day of the disease to warrant a positive diagnosis." "The early manifestation of jaundice is undoubtedly the most characteristic feature of the facies of yellow fever." He also says that these phenomena are often better observed at a slight distance than on close inspection.

The *tongue* is large, moist, and coated with white fur. The stomach is irritable and the epigastrium tender. Nausea with repeated vomiting occurs. The fluid is at first of a light greenish-yellow, subsequently becoming decidedly bilious. The bowels are constipated.

The *urine* almost invariably contains albumin at some time during the first three days. Its presence may be very transient. It may be found in the evening and not at other times. The amount of albumin is sometimes very large, and abundant blood and tube-casts are found.

The nephritis subsides rapidly, without leaving traces. The urine is acid in reaction and scanty in amount. It is sometimes suppressed.

During this febrile period the patient complains of headache, pains in the back and limbs, and intense thirst. The mind, however, is usually perfectly clear. Contrary to expectation, Guitéras asserts that the nervous symptoms are, perhaps, more prominent in the adult than in the child. "The loquacity, the short-cut phrases and precipitate speech, the excitement, the show of indifference with unmistakable evidences of fear—all these, that are such prominent features of the disease in the adult, are absent in the young."[2]

In from two to five days the temperature falls to or below normal, headache and pains in the limbs disappear, and the patient is cheerful and thinks himself convalescent. This is the fact in mild cases, but in more severe cases the period of remission or stage of *calm* is followed by a return of symptoms in a few hours or at most a day or two. The jaundice deepens, vomiting becomes more urgent and in adults is accompanied by much retching. It is bilious, streaked with blood, or thick and wholly black ("black vomit"); the temperature may rise again

[1] "Report of the Surgeon-General of the Marine-Hospital Service, 1888;" Keating's Cyclopædia of Diseases of Children, 1889, vol. i.

[2] Keating's Cyclopædia, loc. cit.

as high as, or higher, than in the original paroxysm, or it may remain depressed. In any event the pulse is apt to be slow, often from 40 to 60. The urine contains albumin, blood, and casts, and may be suppressed, adding uræmia to the other toxæmia. Convulsions at this stage are usually uræmic. Hemorrhages may occur from any mucous surface. The gums are tender, swollen, and bleed easily. There may be epistaxis, hemorrhage from the ear, bowel, uterus, or vagina. Pregnant women miscarry. Ecchymoses also may form. Death may take place in coma or convulsions. If the patient lingers beyond the fifth or sixth day he sinks into a typical typhoid state, with diarrhœa and marked adynamia, from which he may or may not emerge.

As in scarlet fever, the patient may be smitten down and die in a few hours from the time he was in apparent health. In other grave cases the temperature remains high, and rises instead of falls on the third or fourth day. The *duration* of the disease is from two to five or six days; if a typhoid state develops, it may last ten days or two weeks.

Complications are not common. Phlebitis and lymphangitis occur, and Guitéras says he has noticed hepatitis, insanity, and paralysis (probably from neuritis). Second attacks are extremely uncommon.

Examination of Blood. The bacillus icteriodes is a slender rod from two to four micromes in length. It is ciliated and motile. By staining a drop of blood with Gram's method it is seen in more than half the cases.

Serum Diagnosis. Woodson and the Archinards have found agglutination to take place in a large proportion of cases of yellow fever. The blood, taken as early as the second day, gave a prompt reaction in from 75 to 80 per cent. of all cases. Dilutions of 1 to 40 were used, but reaction took place in dilutions as low as 1 to 5. Pothier and Lerch report successfully upon this reaction. Cultures from the blood produce an organism which grows on ordinary media; does not coagulate milk, but ferments saccharine fluids.

Inoculation. Inoculation of dogs and monkeys produces a clinical picture similar to the original infection.

Diagnosis. Yellow fever is distinguished from pernicious malarial fever by the slow pulse, the characteristic facies, the early transient albuminuria, the deep jaundice, the absence of diarrhœa, the occurrence of black vomit, the tendency to hemorrhage, and the clear mind.

If it is not practical to make a diagnosis based upon an examination of the blood, the three important characteristics which Guitéras laid stress upon must be borne in mind in addition to the usual data secured for the purpose of determining the presence of an epidemic and contagious disease. The three diagnostic points of Guitéras are the facies, the albuminuria, and the slowing of the pulse, with continuance or increase of the fever. By these means the affection must be distinguished from dengue and from various forms of malarial fever, especially the æstivo-autumnal infections.

Malta Fever.

Malta fever is a remarkable infection which seems to prevail within the limits of the Mediterranean. It is an infection characterized by

gradual onset and by repeated remissions of the *fever*. The alternating febrile and afebrile periods which characterize the disease continue from two months to two years. The most remarkable feature is the

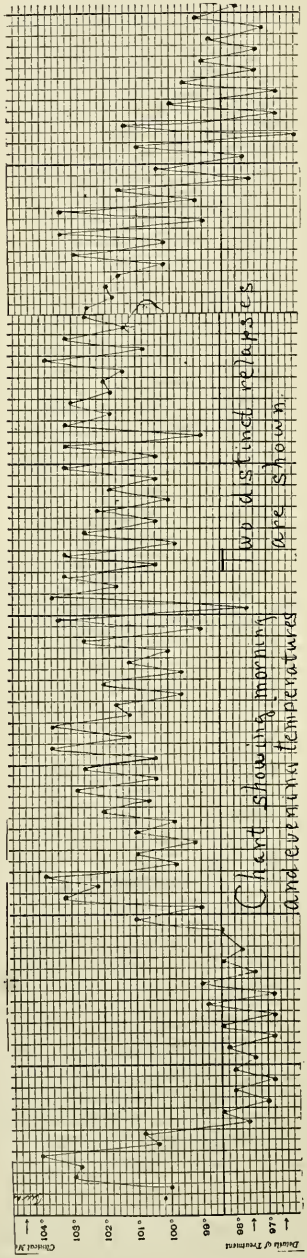
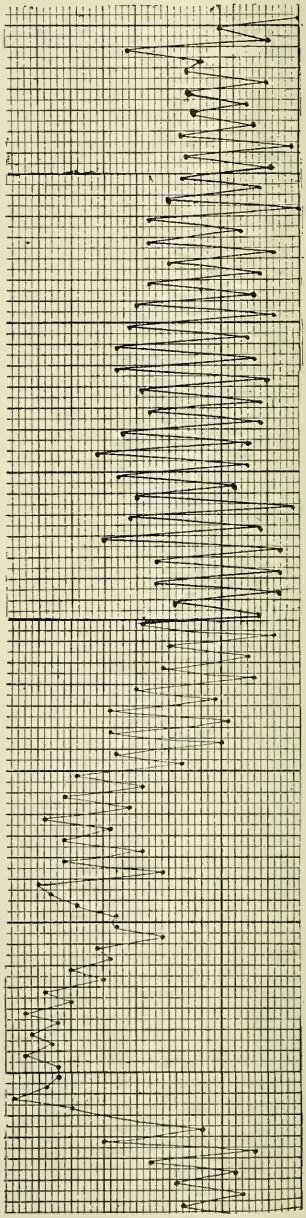


FIG. 73.



Malta fever. (MUSSER and SAILER.)

peculiar character of the temperature-range, which consists of intermitting waves or undulations of fever of a distinctly remittent type. These periods of fever last from one to three weeks, followed by an apyretic period or a period of abatement lasting from two to ten days. The daily temperature-range may be intermittent or remittent. The febrile course may continue six months or more. During this time patients grow more and more prostrated, become anæmic, and usually suffer from constipation. Profuse sweats attend the decline of the daily range, and in many instances we find enlargement of the spleen. Neuralgias occur in various parts of the body; the joints become enlarged, and fibrous tissues may be the seat of inflammation. Hughes —who describes the disease most accurately—describes a malignant type lasting a week or ten days, and an undulatory type continuing for weeks or months. Indeed, the relapses are known to occur over a period of two years. The third is known as the intermittent type, in which there is a daily rise of temperature without other marked symptoms. The undulatory type is the most common variety. The infectious micro-organism is the *micrococcus melitensis*.

Diagnosis. The occurrence of fever described above in the countries bordering upon the Mediterranean, whether epidemic or endemic, should always suggest Malta fever. The possibility of its occurrence in other tropical countries, as in the islands of the Caribbean Sea, must not be forgotten. A positive diagnosis is made by exclusion of all forms of malaria by an examination of the blood, and of typhoid fever by finding the bacillus typhosus in the urine or stools of the suspected patient. The micro-organism has not been isolated from the blood, but the *serum reaction* is a valuable means of diagnosis. (See page 233.) This reaction is performed as in cases of typhoid fever. The culture must be carefully selected. With a 1 to 10 or 1 to 50 dilution agglutination takes place when the serum of a patient with Malta fever is used. The serum of such a patient does not have any effect upon the typhoid bacillus nor upon other organisms. Aldrich states that the first reaction occurs about the fifth day.

Gonorrhœal Infection.

Although the infection is usually limited to the genito-urinary tract, it is well known that the gonococcus may enter the blood and infect tissues elsewhere, causing a local inflammation. We therefore see symptoms due to the primary infection; symptoms due to the infection of the genito-urinary organs by direct continuity, and systemic infection. The primary infection involves the adnexæ of the genital organs in the male and the female. Salpingitis, metritis, and ovaritis in females, with the occurrence occasionally of peritonitis, arise from spreading by continuity. In both sexes cystitis, ureteritis, and pyelitis occur. The infection is usually mixed.

When the gonococcus invades the blood, symptoms of septicæmia or pyæmia arise. The infection may be rapid and fatal, and may terminate ten days after the primary lesion. The occurrence of such general infection is suspected when the history of the primary infection can be

secured, and in addition the micro-organism can be recovered from the blood, as was successfully done by Thayer.

In other infections the joints become involved and we have the phenomena of gonorrhœal rheumatism (see Joints), the course and symptoms of which are discussed elsewhere. Endocarditis may result from gonorrhœal infection, and can only be distinguished from other forms of endocarditis by the history and the finding of micro-organisms in the blood. Myocarditis (Councilman) and pericarditis may also occur.

Diagnosis. Thayer and Blumer and Thayer and Lazear have succeeded in recovering the gonococcus from the blood in this form of septicæmia. The blood is withdrawn from the median basilic vein by a sterilized syringe. A large quantity is secured. It is mixed with melted agar and immediately plated. The medium should contain at least one-third blood. This is practically the medium which Wertheim recommends. After forty-eight hours colonies appear half the size of a pin-head, granular, but with irregular borders. Cover-slip preparations of the colonies, if the case is gonorrhœa, will show the tinctorial and morphological characteristics of the gonococcus. (See Plate III., Fig. 3, B. The diagnosis is further established by finding the gonococcus in any purulent discharge, as of the urethra or vagina. (See Chapter XXI.—Exudations, etc.)

CHAPTER XX.

THE DATA OBTAINED BY OBSERVATION—(*Continued*).

FEVER. THE INFECTIOUS DISEASES.

Infections Recognized by the Examination of Excretions and Secretions or by the Products of the Infectious Inflammation.

THE following infections are disclosed by the examination of the products of the infection found in the inflammatory areas (pus); in the excretions and secretions of the body; in the sputa; in the vomitus; in the fæces or in the urine. The reader should refer to the sections describing the method of the examination of pus, sputum, and secretions bacteriologically. They are as follows: *Erysipelas, pneumonia, tuberculosis, influenza, cerebro-spinal meningitis, diphtheria, septico-pyæmia, glanders, cholera Asiatica, dysentery, bubonic plague, leprosy, actinomycosis, tetanus, trichinosis.*

Erysipelas.

The fever of this infection, particularly in a first attack, is very marked. It rises suddenly to a considerable height and may antedate the eruption. It resembles the course of a pneumococcus infection.

It is an acute, specific, contagious, and infectious disease, characterized by a sudden onset, with a bright-red eruption, which usually begins on the face near the nose or mouth and spreads over the entire face and scalp. It is attended with burning heat of the skin and great disfigurement from swelling.

The specific cause of erysipelas is the streptococcus erysipelatosus. It is carried to a slight extent by the air, and still more in the discharges, especially those of the nose. Repeated attacks occur in persons with chronic nasopharyngeal catarrh, carious teeth, or a sinus. It is apt to attack persons with open wounds (surgical erysipelas), and puerperal women, producing in these cases sloughing and septicæmia.

One attack does not protect against another; on the contrary, if there is any focus in which the streptococci linger, one attack actually predisposes to another.

The period of incubation is usually from three days to a week. On close inquiry a history of sore-throat and some enlargement of the cervical lymphatics is usually found to precede an attack of facial erysipelas. The invasion is sudden and is marked by chill. The temperature rises to 104° or 105°, and in the next two or three days may rise still higher.

Coincidently with the rise in temperature the portion of the *skin* to be affected burns, tingles, is tender to the touch, and may be seen to be

reddened. The redness increases in intensity and extent, while the skin is swollen and slightly œdematous. The part of the face to be affected is usually the cheek in close proximity to the nose, less frequently near the mouth and ear. Vesicles and blebs often form when the inflammation is very intense. The redness disappears upon pressure, but quickly returns; sometimes it has a dusky, purplish hue.

A marked characteristic of the disease is its tendency to spread. In ordinary cases it involves one cheek, eyelid, and ear, and travels across the bridge of the nose to the other side. The inflammation is most intense when it is spreading; the advancing margin is raised, tense, and brawny; the line is thus sharply drawn between healthy and inflamed tissue. The loose tissue about the eyes swells enormously, both eyes are closed, the entire face swollen, red, and disfigured with vesicles and blebs here and there. Curiously the chin escapes. The redness and swelling begin to subside in the part first attacked, before the process has reached its height on the opposite side. As a rule, facial erysipelas does not extend beyond the face, the scalp and neck being spared. The scalp, however, is more frequently affected than the neck; occasionally erysipelas leads to extensive cellulitis of the scalp, with the production of a septic constitutional condition and much local sloughing. The submaxillary glands are more or less enlarged, sometimes so much so as to prevent the taking of solid food.

When on the body the eruption spreads over a greater extent than when primary on the face, hence its name, "the red runner." It may pass from the heel to the thigh, and over the trunk, lasting for weeks.

While the erysipelas is extending the fever continues, and is sometimes alarmingly high. The pulse is frequent and soft. Leucocytosis is present. Nocturnal delirium is not uncommon in severe cases, and sometimes nausea and vomiting are frequent. The bowels are usually constipated. The urine is high-colored, frequently contains a small amount of albumin, and actual nephritis sometimes occurs.

In favorable cases of facial erysipelas the process is at an end in a week or less. It may be prolonged to two weeks, subsiding by crisis or lysis, and convalescence is usually rapid. The vesicles or bullæ dry up into yellowish crusts and the epiderm is shed in large or small pieces according to the intensity of the process.

Pneumonia and nephritis are the most frequent complications. Meningitis, pericarditis, and endocarditis also occur. Erysipelas may extend inward and involve the fauces, pharynx, and larynx, producing œdema and death from suffocation.

Sequelæ. If the scalp has been involved the hair falls out. The cervical adenitis may result in abscess; chronic nephritis may result. Otitis media occurs occasionally, and so do keratitis and abscess of the eyelids.

On the other hand, erysipelas is credited with causing the disappearance of lupus, chronic eczema, and sarcomata.

Diagnosis. Bacteriological Diagnosis. Examination of pus or discharge from the nose or thorax will disclose the presence of the streptococcus. (See Plate VII., Fig. 1, and Chapter XXI.)

Herpes zoster of the face and forehead is distinguished from erysipelas

by the fact that vesicles appear first, followed by erythematous redness, and that they are limited by the median line, and are preceded and accompanied by sharp neuralgic pain, whereas erysipelas affects both sides of the face, and vesicles appear at the height of the disease ; the pain is much less in erysipelas. It is distinguished from *dermatitis* of various kinds mainly by the sharper febrile reaction, the raised border of the eruption, which begins on one side and spreads to the other. Erysipelas is rarely equally intense upon the two sides. Dermatitis frequently is. The latter often exhibits a rough surface, whereas, until vesicles appear, erysipelas is smooth and shiny.

Chronic *erythematous eczema* occurs in the middle-aged and old persons, is afebrile, accompanied by little swelling but a great deal of itching, and runs a slow course.

Lobar Pneumonia.

The Pneumococcus Infections. In typical cases of the infection we are about to consider the course of the fever is of great diagnostic significance. Its sudden rise to a great height, preceded by a rigor, is of itself suggestive. During the succeeding days of the disease the morning and evening temperature varies but little. When associated with hurried respiration and the intoxication symptoms attending this infection, even though no physical signs are present in the lungs, pneumonia can reasonably be suspected. The termination of the febrile course is characteristic of the infection. The sudden fall to normal or a subnormal temperature—known as the crisis—brings to an abrupt end the usually alarming symptoms.

Acute pneumonia, croupous or lobar pneumonia, is an infectious inflammatory disease excited by the micrococcus lanceolatus (diplococcus pneumoniæ, pneumococcus) involving the vesicular structure of the lungs, and followed by choking of the alveoli with the products of inflammation ; it is attended by severe constitutional symptoms due to the toxines of the infecting organism.

Symptoms. *Mode of Onset.* The invasion of pneumonia is usually sudden, and is marked by a *chill.* The temperature rises rapidly, and may reach 104° or 105° in the first twelve hours after the chill. With the fever, the patient complains of severe headache and *pain* in the side, and has a short, quick cough and sometimes vomiting. The pulse is moderately accelerated, and the respiration either is or soon becomes very frequent. The face is apt to be flushed, and there may be a circumscribed red spot on the cheek. The skin is hot and dry. On physical examination, within the first twenty-four hours, a small patch of consolidation is detected, which may subsequently extend over a large area.

While this is the picture of an ordinary pneumonia in its early stage, all cases are by no means so clear. In some the course resembles that of a general fever in which the pulmonary disease is a local manifestation. In such cases there may be prodromata, consisting of headache, general malaise, a slight bronchitis, and digestive disturbance. Then follows the chill. *Central pneumonia.* The fever may be high for

several days before there is any discoverable consolidation of the lungs, and during this time cough may be wholly, or almost wholly, absent. The respirations increase gradually in frequency, and finally a well-marked pneumonia can be made out. It is customary to account for these cases by the supposition that pneumonia developed in the interior of the lung and consolidation gradually extended to the surface. In some cases the patient presents no more definite symptoms for three or four days than high fever, intense headache, and moderately accelerated respiration.

Later Stages. At the end of forty-eight hours, or, at the most, of four days, the patient is found lying in bed in the dorsal position, or on the affected side. The face is flushed, and countenance anxious, the respiration hurried, the alæ nasi play vigorously. The temperature varies little from the first day's rise; the chest pain has subsided, and the short, dry cough is now attended by viscid expectoration. The respiration continues hurried, the pulse full and bounding. During this time the physical signs of consolidation continue and increase.

After a period of five to ten days the termination takes place by crisis, the pain in the chest abates, the cough becomes looser, and the expectoration more free, but the other symptoms persist. In addition, in some cases, delirium occurs, the pulse softens and becomes dicrotic, the urine becomes albuminous.

Respiratory Symptoms. Chest-pain, cough, hurried respiration of a peculiar type, and expectoration are characteristic. The *chest-pain* is sharp and stabbing or lancinating. It is increased by breathing. It is seated about the nipple or in the *axillary region*, at the angle of the scapula or below the diaphragm. Its seat always indicates the side affected. *Cough* is short and dry, smothered and painful; it soon becomes softer and painless as the expectoration becomes free. It may be absent in the feeble, in the aged, in alcoholic subjects, or in persons with brain disease, including insanity.

Characteristic symptoms of pneumonia are the increased frequency and the type of the *respiration*. The rate in adults reaches 40, 50, or even 60 per minute, and in children 80 and 100 are not very uncommon.

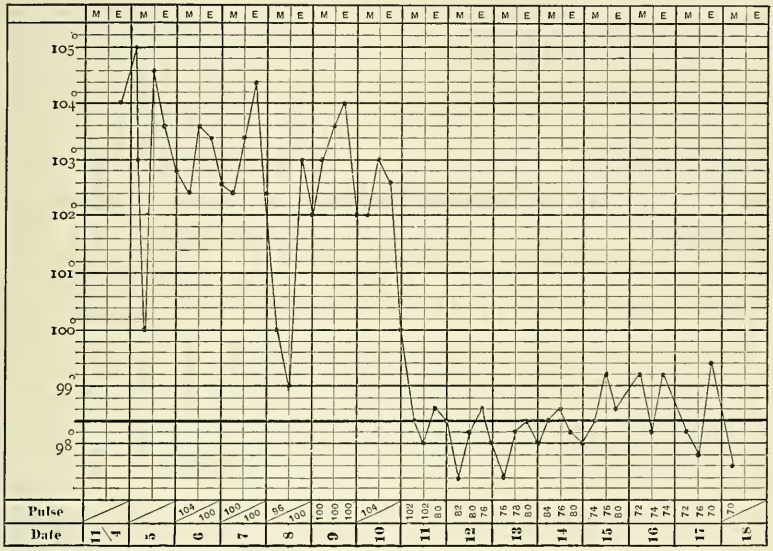
The pulse, on the contrary, does not increase in frequency in the same proportion; hence, the normal ratio of respiration to pulse of 1 to 4 ceases, and becomes 1 to 3 or 1 to 2.

Inspiration is short, expiration quick and often attended by an expiratory noise or grunt. The long pause may take place after inspiration instead of expiration. In children both are so short that unless the epigastrium is inspected it may be difficult to distinguish the two.

In ordinary cases which run a normal course the cough is followed by expectoration, which is at first viscid mucus, but gradually becomes reddish-brown from admixture of blood—the *rusty sputum* of pneumonia. This sputum is characteristic, almost pathognomonic. It is expelled with difficulty from the mouth, clinging to the lips or to the mustache. It cannot be removed from the spit-cup by turning it upside down. It continues to be rusty, and as the crisis approaches becomes purulent and is discharged with ease. In typhoid pneumonia

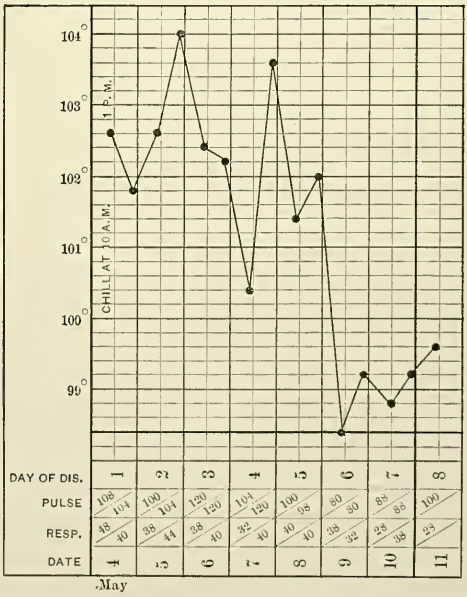
it looks like prune-juice (See Sputum.) It contains blood, alveolar epithelium, the specific micrococcus, and later pus and small fibrinous casts.

FIG. 74.



Pneumonia. Sudden rise; termination by crisis. Pseudo-crisis on eighth day.

FIG. 75.



Pneumonia from first day. Pseudo-crisis on fourth day. Crisis began on fifth.

The Fever. The chill that precedes the fever is pronounced and is always a warning to look for a pulmonic inflammation. In children a convulsion is rarely absent in frank pneumonias. During its occurrence the body-temperature rises. In twelve hours it reaches 104° to 105°. (See Figs. 74 and 75.) It remains at this point, obeying the laws of diurnal variation. The hot, dry skin, the parched lips, the dry tongue, the thirst, the anorexia, the hurried breathing, the occasional delirium, the loaded urine attest its presence. At the end of the third, or more frequently the fifth, seventh, or ninth day, *crisis* takes place; the fall is abrupt, and the normal or a subnormal temperature may be reached in from five to fifteen hours. *Pseudo-crisis*, as the accompanying chart indicates, may precede the true crisis by twenty-four or forty-eight hours. The decline may take place by lysis, however. Protracted fever indicates delayed resolution or the occurrence of a complication.

Cerebral Symptoms. In some cases, especially in children, the onset of the disease may be marked by a convulsion. This is said to occur more frequently in apical pneumonias than in pneumonias of the base. Headache and delirium are so pronounced in some cases as to simulate meningitis. This is most likely to be the case in severe apical pneumonia in children, and in double pneumonia either in children or in adults.

Delirium may occur during the height of the fever, and occasionally is maniacal. Nocturnal delirium may be a constant symptom in very grave cases. In drunkards it may simulate delirium tremens, and may be pronounced, without much fever. In the later stages of grave or fatal cases a low form of delirium, with a tendency to coma, is common.

The Heart and Pulse. The pulse is small at the time of the chill, but becomes full and bounding during the fever; later it may become dicrotic. The pulse-respiration ratio has been referred to. The pulse varies in frequency and in character with the type of the disease. In healthy adults it is rarely over 110. In the debilitated it may be very frequent, small, and feeble; in the aged, frequent and dicrotic. Extensive consolidations reduce the amount of blood in the general circulation, cause rapid action of the heart and a small pulse, and favor death with the heart in asystole.

The heart-sounds are clear. A murmur low in pitch is often heard in the mitral and pulmonary areas. The left ventricle acts forcibly. The pulmonary second sound is accentuated. If dilatation and failure of the right heart takes place, the area of dulness may extend beyond the right edge of the sternum, an epigastric impulse be noted, turgescence of the veins in the neck become marked, but, above all, the previously accentuated pulmonic second sound may become weak or disappear.

Gastro-intestinal Symptoms. Vomiting frequently occurs in children at the onset, and both in them and in adults may persist and mask pulmonary symptoms. The appetite is lost. The tongue is furred. It may become dry and brown. The bowels are constipated except when complications occur. The *spleen* is enlarged. The vomiting and

epigastric pain may be so pronounced as to mask the pulmonary symptoms. The occasional presence of jaundice has caused it to be mistaken for hepatitis, congestion of the liver, and even for gallstones. I saw a case of pneumonia, said to be appendicitis and peritonitis because of the characteristic pain, colic, and vomiting, followed by great abdominal tenderness in the upper abdomen.

The Blood. Leucocytosis is a marked accompaniment of pneumonia, especially in cases ending favorably. The white cells may be increased from 12,000 to 40,000. They fall with the crisis, or probably a day after the termination of the fever. In malignant forms there may be no leucocytosis. Increase in the fibrin network causing the "buffy coat" of older writers is commonly seen.

Cutaneous Symptoms. Herpes on the lips, the nose, or the genitals is of common occurrence. Sweating occurs with the crisis, or if heart failure is imminent.

The Urine. The urine is scanty and high-colored, and may contain a small amount of albumin. In some cases the chlorides are found to be absent. This is determined by acidulating the urine with a drop or two of nitric acid, and then adding one or two drops of a 10 per cent. solution of silver nitrate. If chlorides are present a heavy white cloud of chloride of silver is thrown down. The chlorides are not invariably absent, or even diminished in pneumonia, hence their reappearance, which is said to indicate beginning convalescence, loses its value as a prognostic sign.

Physical Signs. (See Diseases of the Lungs, Plate XIX.) CONSOLIDATION. Diminution in the amount of air, increase of solid contents. On *inspection*, diminished movement. If extensive consolidation, enlargement of the affected side. On *palpation*, inspection confirmed and increased vocal fremitus discovered. Both are more marked at the height of consolidation. *Percussion.* In first stage, impaired resonance or Skodaic resonance. In stage of hepatization, dulness or flatness, but without any wooden quality or marked resistance.

Auscultation. In the early stage, that of congestion, the respiratory murmur is suppressed and crepitant râles are heard at the end of inspiration. On full inspiration or after cough a broncho-vesicular respiration is brought out. When consolidation has taken place the respiratory murmur is bronchial. Râles, if present, are moist subcrepitant râles from associated bronchitis, or a few crepitant râles may still persist, and a friction-sound be heard.

When resolution sets in the crepitant râle reappears, quickly followed by moist subcrepitant râles, heard both on inspiration and expiration, while dulness gradually yields to impaired resonance. The respiration loses its bronchial character and again acquires a vesicular element before becoming completely normal. It may be a week or two, or many months, even in uncomplicated cases, before the percussion-note becomes perfectly clear, and râles wholly disappear.

The physical signs are modified by the intensity of the inflammation in the lung structure and by the pleural complications. In *massive pneumonia*, for instance, the auscultatory signs are absent. On percussion, the lung is absolutely flat. There is no fremitus or tubular breath-

ing. The physical signs resemble those of pleurisy with effusion. In the *central pneumonia* the physical signs may be delayed until the third or fourth day. A few râles or febrile breathing over a small area may be the only indication of a possible lung process. In the *aged* the physical signs are obscure. In patients with laryngeal disease or marked obstruction in the nasopharynx the physical signs may be indefinite. Bronchial breathing may not be heard unless the patient takes a full breath or coughs. In this class of cases, as well as those with feeble respiratory movement, as the aged, the weak, and those suffering from some other disease, as tuberculosis, the physical signs are not made out because of the deficiency of respiratory movements. The indefiniteness of the physical signs makes the diagnosis all the more difficult, because it is this class of subjects in which the general symptoms of infection are very slight. Increased respiration may be the most suggestive sign. Slight elevation of the temperature and more or less stupor may be the only other clinical symptoms.

Duration and Course. The duration of the disease is from one to two weeks. It may subside by crisis on the third, fifth, seventh, or ninth day, or gradually by lysis. Crisis is marked by a critical sweat, a copious discharge of limpid urine, or sometimes by a few loose movements of the bowels, accompanying a fall of temperature to or below normal.

Instead of clearing up, the pneumonia may progress to suppuration, abscess, or gangrene. These conditions can be made out by the character and range of temperature, the general condition of the patient, the sputum, and the physical signs. Termination in abscess or gangrene is rare.

In cases proceeding to a fatal issue the strength fails, respiration becomes more labored, and expectoration increasingly difficult. The number of respirations often diminishes, but the pulse continues frequent and often becomes small and irregular. Physical examination shows diffuse bronchitis with œdema. The heart's action is irregular and rapid. The sounds are weak and feeble ; the first becomes short and snappy like the second, and later both are weak or indistinct. Death may occur abruptly from convulsion, or more frequently from asphyxia, due to œdema of the lungs, which in turn sets in on account of weakness of the heart or the development of heart-clot from cardiac asystole.

Varieties. *Migratory pneumonia.* Sometimes, with the reappearance of abundant râles and increased expectoration, the fever continues high, or, if the temperature has fallen to normal, again rises, the patient is disinclined to take food, has a dry, brown tongue, and is often delirious. In such cases the pneumonia is probably extending in the lung already involved, or has attacked the other lung.

Typhoid pneumonia is an unfortunate name for an adynamic form of the disease with typhoid symptoms. If it arises in the course of or complicates low fevers, it is usually of the typhoid type ; but it occurs also in those much exhausted, in depraved health, or exposed to unhygienic surroundings. It is found also in cases of septicæmia, in Bright's disease, in drunkards, and in the negroes in the southern part of the United States.

The characteristic features of this form of pneumonia are the great physical prostration and the weak heart-action. The fever is high, the respiration and pulse frequent, and delirium and vomiting are more frequent than in the ordinary form. The skin sometimes has a dusky hue; the tongue is heavily coated, or may be dry and brown, and sordes collect on the teeth. The sputa may be rusty, and sometimes pure blood is expectorated. The disease may prove fatal rapidly, or may linger for a long time, the patient only gradually coming out of a low typhoid state. It is always dangerous.

Bilious pneumonia is the name given to a type of pneumonia occurring in persons who are already suffering from malarial poisoning. The initial chill lasts longer, and the pain in the side, from coincident pleurisy, is more marked that in ordinary pneumonia. The fever is more remittent, and jaundice and vomiting are present.

Pneumonia in infants is characterized by nervous symptoms. Repeated convulsions and active delirium may be most pronounced, followed by torpor and coma. There is no sputa and but little cough. The apex of the lung is affected.

Pneumonia in the aged is characterized by latency of symptoms. There is but little cough and expectoration. A tendency to the typhoid state, however, is pronounced. The physical signs are obscure.

Pneumonia in alcoholic subjects also develops insidiously and may be masked by the symptoms of delirium tremens. The temperature may be the only indication of infection, as there is no pain, no cough, no expectoration, and no dyspnœa.

Pneumonia with Other Infections. The staphylococcus and streptococcus pyogenes, the colon bacillus, and the bacillus pneumoniæ (Friedländer) are often found with the pneumococcus, and may predominate, inducing a *mixed infection.* The micro-organisms which cause diphtheria, typhoid fever, influenza, and the plague may cause a pneumonia which resembles that of lobar pneumonia in the extent of the consolidation. The micrococcus lanceolatus is found in increased numbers in the sputum of these cases. There is not the same intensity of pulmonary symptoms, however. The respirations are not so hurried. The physical signs, while extensive, are obscure, and indicate rather a heavy lung (congested) than one greatly consolidated. There is impaired resonance, feeble breathing, and a few râles in a large number of cases.

It is this form of lobar pneumonia which it is difficult to distinguish from bronchopneumonia or catarrhal pneumonia—an infection which usually begins in the upper air passages. This form of local infection is considered in the chapter on diseases of the lungs.

Diagnosis. The diagnosis is based upon the aggregation of special symptoms. The mode of onset, the chill, the course of the fever, the pain in the chest, the cough, the peculiar expectoration, the dyspnœa, the abnormal pulse-respiration ratio, the peculiar character of breathing, the physical signs, and leucocytosis are the phenomena of the symptom-complex. It must be remembered that in children, in the aged, in drunkards, in cases of chronic disease, the type is different. In drunkards cerebral symptoms are more marked. In children the cerebral symptoms are more prominent, the expectoration often absent. In the

aged, the cough, the expectoration, and the fever are not pronounced; the former may be absent; the onset is insidious. The same onset and course occur in wasting diseases, as cancer, phthisis, Bright's disease, diabetes, and organic heart disease. In this class of cases a small patch of pneumonia, difficult to determine on physical examination, may be attended by the gravest general symptoms. In all of the above cases, if there is *fever* without cause, although no pulmonary symptoms are present, the lungs must be examined repeatedly. In many such cases the physical signs are obscured because respiratory action is enfeebled by the primary condition.

Pneumonia must be distinguished from other acute inflammatory affections of the lung and pleura and from acute tuberculo-pneumonic phthisis. The evidence for each is considered in the respective sections. The presence of leucocytosis serves to distinguish it from acute tuberculosis and from typhoid fever, meningitis, and influenza. To distinguish pneumonia from pleurisy with effusion, the aspirator may be used.

Bacteriological Diagnosis. Staining and microscopical examination of the sputum reveal the characteristic micro-organism. Care must be taken to secure the sputum from the lung. By inoculation of rabbits with the sputum the disease is readily reproduced. The organism is not readily found in the blood. (See the Sputum.)

Pneumonia may be distinguished from *cerebro-spinal meningitis* by the results of spinal puncture alone; from acute tuberculous pneumonia by the examination of the sputum. The diagnosis in the latter instance may be postponed, as tubercle bacilli are sometimes not found until the tenth or twelfth day. (See Tuberculosis.) Typhoid fever sometimes resembles pneumonia, and must be distinguished after the first week by the results of serum diagnosis.

Pneumococcus Septicæmia. The account we have just given of pneumonia represents but one phase of the pneumococcus infection. This infection may be attended by very grave symptoms, especially those of a toxic nature, with but little if any involvement of the lung tissue. It is well known that we may see the chill, fever, rapid pulse, and hurried respiration with but little evidence of consolidation in the lung, but with nervous symptoms paramount. Delirium, stupor, coma with the phenomena of the ataxic or the typhoid state may prevail. (See pages 199 and 200.) In the ataxic state the symptoms resemble those of mania. In the typhoid form they are not unlike those of uræmia. In either instance death ensues in coma or from heart failure with its attending symptoms. Preceding the cardiac failure the urine is diminished in amount and the secretions generally suppressed.

In other forms of this infection the localization of the process is in the pleura, as in empyema, in the pericardium, in the endocardium, and in the cerebral meninges. Pneumococcus inflammation of these structures is very common. It may develop at the same time that the lungs are affected, independently of the process in the lungs, or subsequent to it. These forms will be considered in a discussion of the various local inflammations just referred to.

It is important to remember that in pleural, pericardial, and cerebro-spinal infections the nature of the infection can be determined by *aspi-*

ration and bacteriological examination of the fluid removed from the respective serous cavity. The pneumococcus infection can be positively diagnosticated in this manner.

These complications, which occur in the course of the disease, modify the clinical picture and obscure the diagnosis.

Tuberculosis.

The infection discussed in this section prevails to a greater degree than that of all the others combined. In some forms, as pointed out in the clinical description, fever is one of the gravest symptoms. In other forms the febrile process may not be pronounced. It must be remembered that the fever may be due to the specific micro-organism or its toxin, or it may be due to a mixed infection. Staphylococcus and streptococcus infections are common attendants upon the tuberculous infection. This secondary infection may disappear or may become the most prominent infection. In many instances a terminal infection ensues, causing mortal symptoms. Infection by the pneumococcus is the most common of these terminal infections. (See page 228.)

Tuberculosis is an infectious disease, the course of which may be acute or chronic. It is caused by the bacillus tuberculosis. This micro-organism sets up a specific inflammation characterized by the development of nodules or tubercles, or by a diffuse growth of tuberculous tissue. Either anatomical product may undergo caseation or sclerosis, and in either instance ulceration or calcareous degeneration.

Invasion of the body by the micro-organism may give rise to general infection, with an eruption of miliary tubercles in most of the organs and structures of the body, or to a local infection. General tuberculosis is acute; local tuberculosis may be acute or chronic. In acute tuberculosis the serous membranes, the lungs, liver, kidneys, lymphatic glands and spleen, the bone-marrow, and choroid coat of the eye may be invaded in whole or in part. In chronic tuberculosis the lymph-glands, the lungs, the serous membranes, the tissues and organs of the alimentary canal, the liver, the organs of the genito-urinary system, and the brain and cord are individually invaded.

Diagnosis. The diagnosis of any form of tuberculosis is aided by the determination of the chief factors in its etiology, where this is possible.

Bacteriological Diagnosis. *First.* The discovery of the bacillus tuberculosis in any inflammatory area, or any product of inflammation, as serum, blood, pus, or the secretion from any gland or mucous membrane invaded by the disease, establishes at once the diagnosis of this condition. The method of determining the presence of this micro-organism is fully detailed in the various descriptions of tuberculosis in the discussion of local diseases, and in the accounts of the examination of the sputum and of exudations and transudations. *Inoculation* of inflammatory products, as of a gland or of fluid which has been sedimented, is a positive mode of diagnosis. Guinea-pigs are selected for this purpose.

Second. As tuberculosis is an infectious disease, discovery of the infection is an aid in the diagnosis. Infection takes place by means of

the inhalation of the sputum or other secretions, which when dry float about in the air. It implies in a measure more or less contact with individuals previously infected. In rare cases such contact is productive of the disease by means of direct contagion. The second source of infection is the food-supply. This may occur from the consumption of milk secured from a cow infected with tuberculosis. The eating of meat of tuberculous animals may possibly lead to infection. Direct inoculation is another but rarer source of infection. This usually occurs accidentally only.

Third. It is possible that tuberculosis may be inherited. A more prominent etiological factor, which aids in the diagnosis of the disease, is the presence of a certain type of structure which is a marked hereditary characteristic in families, on account of which feeble resistance is offered to the invasion of the tubercle-bacillus. The phthisical and phthisinoid chest which belongs to this type has been described elsewhere, and the tuberculous and scrofulous states have been outlined. (See page 67 and Part II., Chapter II.) These anatomical conditions, which are inherited, undoubtedly favor the development of tuberculosis.

It is a mistake to lay much stress in the diagnosis of tuberculosis upon the age or the occupation of the individual. Tuberculosis may occur at any age. It is true, however, that at certain periods of life the tubercles are distributed more commonly in one group of organs, while in other periods they affect another group. Lymphatic, joint, and meningeal tuberculosis is most common in the first decade of life. The mesenteric glands are particularly open to invasion at this period.

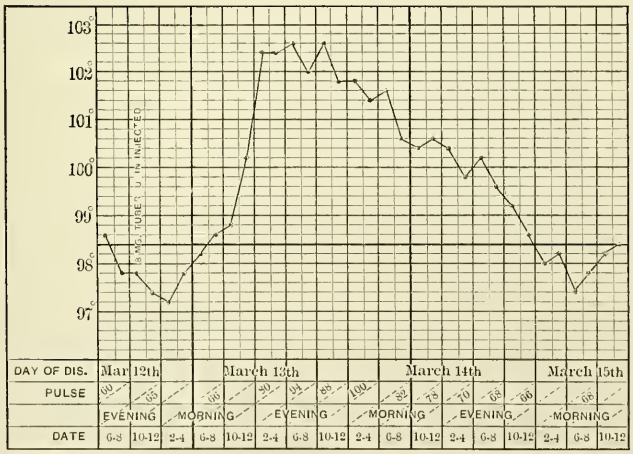
The *diagnosis* of tuberculosis, whether local or general, is further aided by a complete knowledge of the phenomena that attend the entrance of the virus into the body and the mode of diffusion throughout the body after infection has taken place. The phenomena at the point of entrance of the micro-organism are nearly always distinct. The general invasion is associated with symptoms like those of specific fevers. The local secondary effects upon the tissues are always decided. It must be borne in mind that after the exposure, which may lead to infection, either an acute form of tuberculosis of a general character may be set up, with or without marked local symptoms, or acute local tuberculosis alone may arise. In local tuberculosis the disease is confined to one organ or to the lymphatic glands and the organs in the lymphatic distribution, as the bronchial glands, which are primarily affected, and to the lungs. In these structures the entire process of nodular formation, caseation or sclerosis, ulceration or calcification, may take place. The disease remains primarily local. On the other hand, it may be spread by continuity of structure through the lymphatics throughout the remainder of the organ affected, leading to its ultimate destruction and the death of the patient; or general infection of the system may take place from the primary local area. The primary seat of infection may be the lungs, the larynx, the alimentary tract, or the genito-urinary organs. Primary tuberculosis of the serous membranes, of the lymph-glands, of the bones and joints, may take place.

The symptomatology and diagnosis of the various forms of tuberculosis are detailed in the section devoted to the special diseases of the various organs of the body.

The Tuberculin Test. The physical signs and clinical symptoms may point to an inflammatory process in one of the many structures of the body which may be invaded by tubercle bacilli. On the other hand, failure in health, loss of weight, anæmia, and moderate fever may alone occur. The nature of the inflammatory process may be obscure. To determine more accurately whether the inflammation is tuberculous or not, or the "decline" due to tuberculosis, we can resort to the use of tuberculin. Since the researches of Koch, who introduced tuberculin as a remedy in tuberculosis, he himself as well as a number of other observers, has employed this preparation to determine the presence of tuberculosis in the body. In this country Trudeau has been the earliest and most earnest exponent of this means of diagnosis. After the injection of tuberculin a group of phenomena follows, known as the tuberculin *reaction*, if tuberculosis existed anywhere in the body. It was thought the occurrence of this reaction was necessary to bring about a cure. As a therapeutic measure its value has not been upheld by experience. The invariable production of the reaction has led it to be used as a diagnostic medium.

Phenomena of Reaction. About twelve hours after the injection of tuberculin the temperature rises rapidly. In the course of a few hours it has risen two or three degrees. This elevation of tem-

Fig. 76.



Typical reaction with tuberculin.

perature is attended by malaise, pains in the head, back, and legs, and sometimes nausea or vomiting. The maximum temperature is maintained for two or three hours, and then a gradual decline to the normal takes place. The normal temperature is reached in from twenty-four

to thirty-six hours. The whole period of the reaction, from the time of the injection until the termination of the fever, is about forty-eight hours. With the fall of temperature to normal the constitutional symptoms subside. The accompanying chart (Fig. 76) shows the course of the fever in a typical reaction.

Method. Twenty-four to forty-eight hours preceding the test the patient's temperature should be taken every two hours to determine the range at this period of the disease. The injection should be made at a time when the reaction could be observed—*i. e.*, during the period of normal or subnormal temperature. This, of course, can only be selected if the temperature of the disease is intermittent. The hour of day selected to inject the tuberculin should be such that the reaction may be conveniently observed during the waking hours of the patient. Bedtime or the early morning hours are the most convenient.

The site of the injection is not material. Usually the interscapular space is selected. The amount of tuberculin employed is of the greatest importance. The initial dose should never exceed five milligrammes, and it is better to use less than this, and an increasing quantity injected every second or third day. The maximum dose should not exceed ten milligrammes. For children one-twentieth to one-tenth of a milligramme may be the initial dose. The crude tuberculin should be diluted at the time it is used with 1 to 2 per cent. solution of carbolic acid.

At the point of injection a little redness and infiltration, with tenderness to the touch, is observed. This local reaction may also be seen at the site of former negative injections when the larger dose produces reaction. In pulmonary tuberculosis in which physical signs are obscure some auscultatory phenomena which were previously absent may be found during the period of a reaction. This test also enables one to detect tuberculosis in the pleura, pericardium, peritoneum, genito-urinary tract, and lymphatic glands, the meninges, bones, and the skin. The test is of special value in cervical adenitis.

It must be remembered that a negative result with large doses of tuberculin is of more value than a positive one. In the former instance one can affirm that tuberculosis is absent, as well as that there is no old focus in any of these organs. It must also be remembered that the test should only be employed after all other means have failed to make a positive diagnosis.

Acute miliary tuberculosis has been spoken of elsewhere. (See Part II., Chapter II.) Its course may resemble typhoid fever, septicæmia, or malignant endocarditis. It usually develops in the course of tuberculosis in some other organ of the body. The typhoid form has been described in the section indicated. It must not be forgotten that the diagnosis is rendered positive by the demonstration of the presence of tubercle-bacilli in the blood, or of the occurrence of choroidal tubercles in the eye-ground. Another form is attended by marked pulmonary symptoms. This is the type seen in the bronchial pneumonia that occurs in children following measles and whooping cough. (See Catarrhal Pneumonia.) Of the pulmonary symptoms dyspnœa is the most prominent. Cyanosis is marked. The physical signs are not prominent, and may be those of bronchitis alone. Although there is impaired resonance

at the base of the lungs, areas of hyper-resonance are observed above and in front of the chest. Collapse of the lung may cause tubular breathing. The temperature rises to 102° or 103°. An inverse type may be seen.

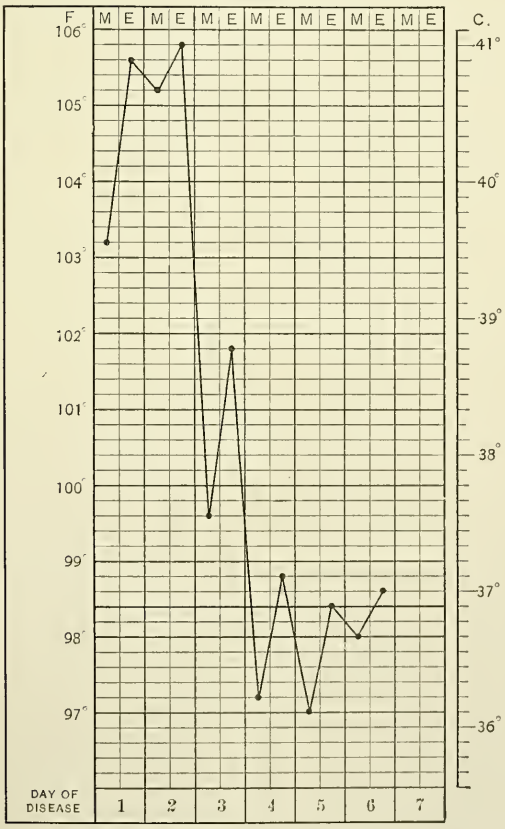
The diagnosis of acute tuberculosis is determined by the history of infection from extraneous sources or from local tuberculosis in some portion of the body, and by the presence of bacilli.

The following conditions should point to the possibility of chronic tuberculosis in some portion of the body : (1) Emaciation, not otherwise explained ; (2) chlorosis or anæmia ; (3) weakness without cause ; (4) fever—the temperature should be taken every two hours during night and day ; (5) causeless sweats ; (6) gastro-intestinal catarrh ; (7) morning nausea ; (8) signs of local inflammation in some organ of the body.

Influenza.

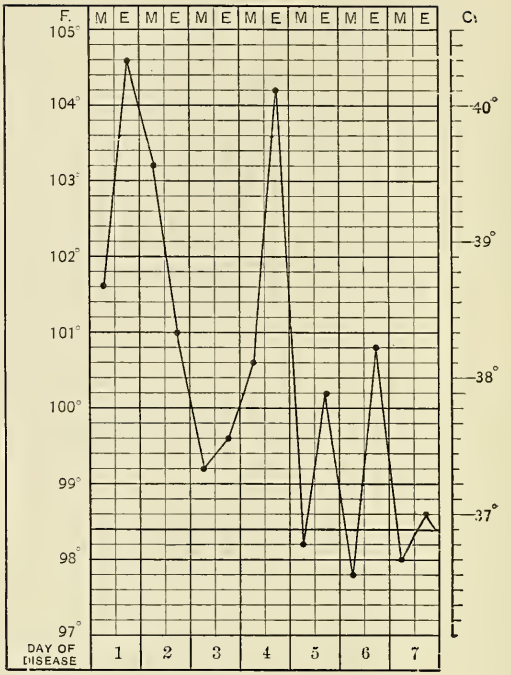
High temperature out of proportion to the local signs of inflammation in the lungs or other structures characterizes this infection. The fever may be continuous, remittent, or intermittent.

FIG. 77.



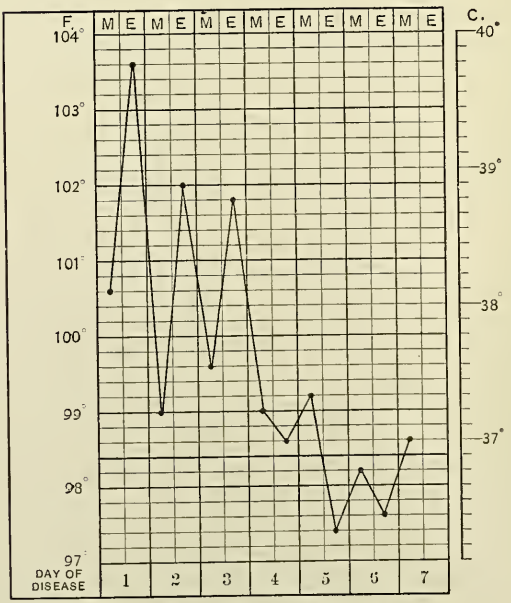
Temperature in influenza—interrupted crisis. (WILSON.)

FIG. 78.



Influenza—intermittent type. (WILSON.)

FIG. 79.



Influenza—remittent type. (WILSON.)

Influenza is a specific contagious febrile disease, occurring in widespread epidemics, having a very short period of incubation, and characterized by great prostration, marked nervous symptoms, and catarrhal inflammation of the respiratory or gastro-intestinal tracts, or both. There is great liability to relapse, and to complications, which are generally pulmonary or nervous.

The disease generally begins with the ordinary symptoms of coryza; but the headache over the eyes and root of the nose is more severe, and may be so agonizing as to mask all other symptoms. The lacrymation, rhinitis, and tormenting cough are all usually worse than in ordinary coryza. Physical weakness, weariness, and depression of spirits are almost invariably present, and they sometimes reach an extraordinary degree. Fever is usually moderate (100° to 102°), but may be 104° to 105° for several days, and then gradually subside. It may terminate by crisis (Fig. 77), or may assume an intermittent or remittent type (Figs. 78 and 79). In ordinary cases the patient seeks relief first for the headache, severe aching pain in back and limbs, and extreme weakness; if these are relieved he is apt to complain most of incessant racking cough, often due more to a tracheitis than to bronchitis. Nausea and vomiting are not uncommon, especially in the morning, at which time also the patient frequently feels worse than he does later in the day. Sleep is broken and restless, and may be accompanied by drenching perspirations. Severe neuralgic pains are common.

In some cases the disease attacks the stomach and bowels especially, and vomiting with diarrhœa are the prominent symptoms. In others the predominant symptoms are nervous, and great pain with prostration masks any catarrhal symptoms. Torpor and delirium may be present. Sometimes a prolonged and severe attack of asthma marks infection in susceptible persons.

The duration of the disease is from a few days to a few weeks. Convalescence is remarkably tedious, and is characterized by persistent weakness. Sweats are often annoying during this time. The heart often continues for some time to beat too frequently and to be easily excited by exertion. Relapses are common.

Diagnosis. Bacteriological Diagnosis. This is possible when the characteristic bacilli are detected by the means described in the section on sputum. Influenza in the great majority of cases is easily recognized. In certain cases, however, it is to be differentiated from *pneumonia*, *typhoid fever*, and *cerebro-spinal meningitis*.

Cases in which the disease sets in with high fever and marked chest-symptoms are very apt to be mistaken for *pneumonia;* but the headache and prostration are more intense, while the respiration is not so frequent. Sweats are common, and albumin and casts in the urine are by no means rare. Physical exploration shows that both lungs are involved, though often not to the same degree. Resonance is impaired, and auscultation shows moist crepitant and subcrepitant râles, which seem to be due to an œdematous condition of the lung-tissue, associated with a diffuse bronchitis. A true lobar pneumonia is rarely present even as a complication.

If diarrhœa is one of the symptoms, *typhoid fever* has to be excluded.

This is extremely difficult in the first two or three days. As a rule, headache, backache, nausea, and sleeplessness are at this time greater in influenza, the spleen is not so much, if at all, enlarged, the diarrhœa can be checked, and tenderness and pain in the right iliac fossa are absent.

It can be distinguished from *cerebro-spinal meningitis* by noting the fact that it begins with coryza, whereas cerebro-spinal meningitis often sets in with chill, vomiting, and faintness; the headache in the former is usually frontal, in the latter occipital, and accompanied by stiffness of the back of the neck. Further, in cerebro-spinal meningitis there are often swellings of the joints, delirium alternating with coma, and in young subjects convulsions are common.

Finally, it may be said that the pronounced diagnostic feature is the preponderance of general symptoms over local inflammations. The occurrence of undue exhaustion, extreme general neuralgias and myalgias, high fever, and profuse sweats, without intense catarrh or inflammation to account for or co-ordinating with them, is of the highest diagnostic significance. The presence of an epidemic, the contagious nature of the affection, the sudden onset, and the bacteriological diagnosis, all point to influenza.

Epidemic Cerebro-spinal Meningitis.

In this infection more than all others the course of the temperature is without diagnostic significance unless it be that this want of a characteristic course is significant. Its extraordinary irregularity is most striking when a large number of charts are examined. The fever may have the course and exacerbation of a typhoid temperature, but it is more similar to that of tuberculosis. It is often of very short duration, followed by a prolonged subnormal temperature. It may be high from the immediate onset of the disease, or remain below 100° for several days, and then suddenly rise to a great height. Remissions and exacerbations may attend many of the cases. The most marked feature, apart from the irregularity of the temperature, is the inequality between the pulse and the temperature. In some instances the pulse is rapid, and the temperature is normal or subnormal, while later in the disease the pulse may be slow when the temperature rises to a considerable height.

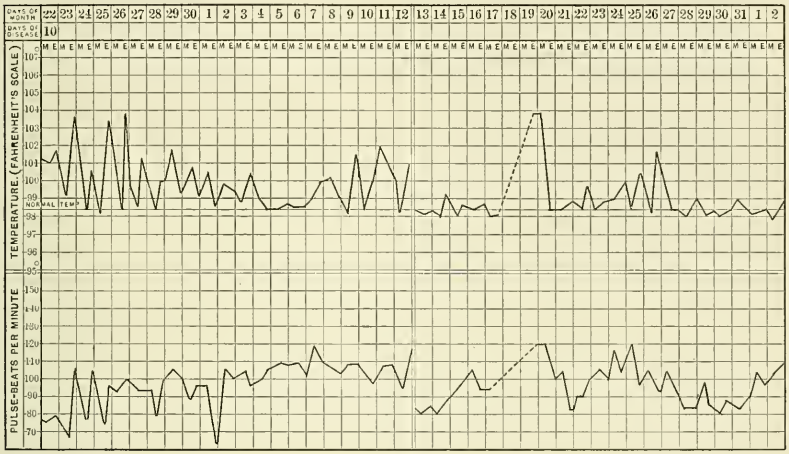
Concerning the temperature, then, it may be said that it may be intermittent, remittent, or continuous; it may be intermittent at one period, continuous at another; it may be afebrile; it may be afebrile at one period and continuous at another.

Cerebro-spinal meningitis, also known as spotted fever, is an acute, specific, infectious, and mildly contagious disease, endemic and epidemic, characterized by evidences of systemic infection, and generally also by symptoms depending upon inflammation of the cerebral and spinal meninges—particularly intense pain in the back and head, hyperæsthesia, retraction of head and neck, delirium, coma, convulsions, and vomiting.

It is most common in cold weather, and in children under fifteen years of age. None of the epidemics show a continuous extension. The

period of incubation is unknown, but is probably short. It is free from symptoms. The invasion of the disease is abrupt, although in some instances the patient may complain of rheumatoid pains in the limbs or a joint, and headache and weakness. Usually the first

FIG. 80.



Cerebro-spinal meningitis, showing irregularity of pulse and temperature. (COUNCILMAN.)

symptom is a severe chill, which may awaken the patient from sleep. In other cases the initial symptom is a convulsion. Then quickly follow repeated vomiting, intense headache, sometimes accompanied by backache, retraction of the head, delirium, and extreme prostration.

The rise in temperature is moderate, and the pulse is as often slow as frequent. The face is pale and livid, expressing suffering, and the patient may toss from one side of the bed to the other, begging some relief for his headache. Simple stiffness of the muscles of the neck may prevail. The pain in the head may be occipital or frontal. The pain in the back becomes more severe, and root-pains dart in all directions, but especially into the limbs or joints, which may be swollen and tender to the touch; in fact, the whole skin is hyperæsthetic and the reflexes are increased. The spinal muscles become rigid, and the head is often retracted. Less frequently the back is arched and trismus occurs. Delirium is common at night. It may develop very early or appear at a late period of the disease. It is sometimes violent or low and muttering. It is often of a sportive type, the patient making absurd remarks, cracking jokes, or singing snatches of a comic song. Delirium may alternate with tonic or clonic convulsions and with stupor. The appetite is poor, the bowels constipated. A remission may occur on the third day, with temporary improvement of the symptoms.

As the attack progresses there may be strabismus, which is usually divergent, inequality of the pupils, nystagmus, ptosis, and optic neuritis. Vertigo, tinnitus, anosmia, and photophobia are common.

Hyperæsthesia and delirium persist. Facial paralysis, a monoplegia, a hemiplegia, or a paraplegia may occur. The pulse becomes more frequent and the fever continues. In favorable cases improvement now begins, the headache and root-pains abating, and delirium and spasms becoming less frequent. In unfavorable cases the convulsions may become more severe and end in fatal coma, or the patient may sink into a typhoid condition, with nephritis as a complication. Coma may come on in the beginning and continue until death.

The skin eruptions, which explain the name "spotted fever," are not always present and exhibit no constant character. Herpes and petechiæ are the most frequent; in other cases the eruption is a purplish mottling, or is macular, or the eruption resembles that of measles. Herpes is most common on the nose and mouth, then on the cheek, forehead, eyes, and ears. The blood shows a leucocytosis, the increase being due to the polynuclear leucocytes.

In the malignant (fulminating) form of the disease death occurs in a few hours, or two or three days. Such cases are apt to arise early in an epidemic. The patient has a violent chill; delirium occurs early; the headache is less intense, or at any rate gives way rapidly to stupor and coma. The pulse is frequent and feeble; there may be no rise of temperature, the skin being cool, clammy, and cyanotic. Local or general convulsions may occur. The eruption may be purpuric, and ecchymoses may even occur. The urine is scanty and contains albumin and casts.

Mild cases usually occur late in epidemics. They are characterized by severe aching in the head, back, and limbs, nausea, vomiting, vertigo, and prostration. They closely resemble the nervous type of influenza, and would escape recognition except during an epidemic.

An abortive form, ending in recovery in two or three days, and an intermittent form, with exacerbations on alternate days, have been described.

The duration of the disease is from a few hours to two or three months. In ordinary favorable cases there is decided improvement toward the end of the first week, and convalescence is established in two weeks. It may become chronic and last for weeks, and, as already stated, may be fatal in a few hours. Relapses are common in some epidemics.

The most frequent *complications* are on the part of the lungs and heart, particularly pneumonia and endocarditis or pericarditis. Pneumonia often occurs so early that it is difficult to decide whether it is primary with marked nervous symptoms, or is only a complication of the cerebro-spinal fever. Nephritis also occurs.

The most frequent *sequels* are deafness, blindness, headache, and local palsies.

Diagnosis. The diagnosis in the presence of an epidemic is not difficult, although an absolute diagnosis can only be made by *lumbar puncture*. The fluid withdrawn is more or less cloudy if the patient has meningitis. If it is the epidemic form, microscopical examination of stained cover-slips and cultures will expose the diplococcus. In some cases fluid cannot be secured, either because the spinal canal is filled with membrane or the fluid is retained in the lateral ventricles.

The fluid is turbid in the early part of the disease. In some cases a purulent sediment forms in the bottom of the test-tube at once. In others, the fluid is simply turbid, and after standing contains considerable fibrin and many cells. The fluid secured at the first puncture may be more turbid than that secured later, although the symptoms may be more severe than at first. If the acute symptoms subside the fluid may be clear, and no cells may be found. In the intermittent cases the fluid may be clear during the interval that the patient is without symptoms. In chronic cases there may be no turbidity.

The *cells* in the spinal fluid are chiefly polymorphonuclear leucocytes—"pus corpuscles." Small lymphoid cells and large endothelial cells may be present. The latter are phagocytic, and have large oval or round nuclei. They may contain leucocytes and blood-corpuscles. In the pus corpuscles or leucocytes the diplococci are found; they are rarely found outside of the cells. Late in the disease the pus corpuscles do not stain sharply and are degenerated. In chronic cases the fluid contains a few pus corpuscles which are smaller than usual, and like lymphoid cells.

Bacteriological Diagnosis. This disease is due to the *diplococcus intracellularis*. This micrococcus appears in diplococcus form as two hemispheres the size of the ordinary micrococcus. It stains with the ordinary stains for bacteria. It is decolorized by the Gram method. The staining is sometimes irregular, some being brightly stained, others faintly. There is some variation in the size of the organisms. Both variation in size and staining are apparently due to degeneration. The two organisms are sharply separated usually, though sometimes they seem to be united. (Figs. 81 and 82.)

Fig. 81.

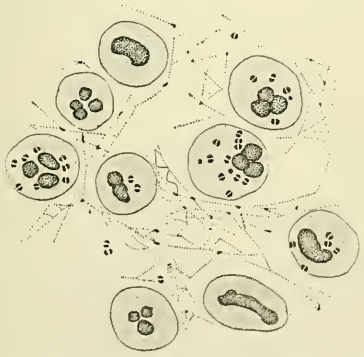


Fig. 82.

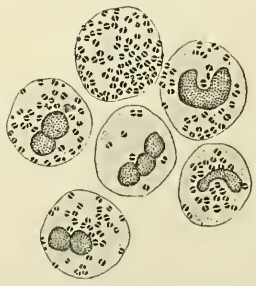


Fig. 81. Pus cells containing diplococci from the meninges. A few diplococci are in the exudate outside of the pus cells. Between the pus cells there are delicate fibrillæ of fibrin. The drawing is an accurate representation of a group of cells in the field of the microscope. (Councilman.)

Fig. 82. Pus cells from an alveolus of the lung in a case of diplococcus pneumonia. The cells are swollen and contain immense numbers of diplococci. Both figures from stained cover-slips.

The organisms do not grow profusely. The blood-serum mixture of Löffler as prepared by Mallory is the best medium. It is often difficult to make cultures unless a large quantity of material is used.

Transfers must be made daily to keep cultures going. The growth on the serum mixture forms round, white, shiny viscid-like colonies with smooth outlines. They do not liquefy the blood-serum. In the tissues the diplococcus is found in the interior of the polynuclear leucocytes.

Cultures. Cultures should be made at the time of puncture. In the majority of cases a growth of the diplococcus is found, although even in acute cases rarely they may not grow. In chronic cases a growth is only rarely obtained. (Plate VIII.)

This form of meningitis must be excluded from pneumococcus meningitis, tuberculous meningitis, and streptococcus meningitis. In the pneumococcus form the symptoms are comparatively slight and are usually preceded by pneumonia. In the streptococcus form the clinical history is like that of ordinary forms of meningitis. The evidence of an infection elsewhere is usually present. Tuberculous meningitis is recognized by the methods employed to detect tuberculosis elsewhere in a patient suffering from the usual symptoms of cerebro-spinal meningitis. The most positive method of distinction of the various forms is by lumbar puncture. (See Chapter XXI.)

Kernig's Sign (Kernig, 1884; Netter, 1898). This sign is of value in the diagnosis of meningitis, but is present in any form. It is determined by placing the patient in the dorsal decubitus, with the legs relaxed and fully extended at the knees. When the child is raised in a sitting posture the knees are flexed, and cannot be extended on account of contracture of the posterior muscles of the thigh. In adults, if the patient is propped up, or seated on the side of the bed, and an attempt made to extend the leg on the thigh, there is contraction of the flexures. The test can be equally well performed by flexing the thigh on the abdomen until it makes a right angle. When an attempt is made to extend the leg it will be found that the limb cannot be fully stretched out if meningitis is present.

Diphtheria.

In this infection the temperature-range is variable. The infection may be intense, and yet the temperature remain subnormal, especially if the fever is due to the toxin, and not, as is frequently the case, to a mixed infection.

Diphtheria is an acute, specific, infectious, and contagious disease, sporadic and epidemic, occurring especially in children from one to six years of age, and characterized by insidious or abrupt onset, with moderate fever, and the development upon the fauces or upon any abraded surface of a grayish-white false membrane, which has a tendency to extend, especially to the larynx. The subsequent phenomena are those of stenosis of the larynx, or toxæmia, with or without superadded uræmia or marked cardiac weakness; it is further characterized by the liability to paralysis as a sequel.

Diphtheria is spread by inhaling the expired breath of a diphtheritic patient, or breathing air which has been contaminated by the clothing of the patient or the discharges from his nose and throat. It may also

PLATE VIII.

Fig. 1.

Fig. 2.

Cerebro-Spinal Meningitis. (Councilman.)

Fig. 1. Forty-eight-hour culture of diplococcus intracellularis on Loeffler's blood-serum mixture.

Fig. 2. Abundant growth in twenty-four-hour culture on fresh blood-serum. The colonies are minute, very numerous, and somewhat resemble similar cultures of the pneumococcus.

be transmitted directly, as when a fragment of membrane is ejected by coughing and infects the mouth or eye of physician or attendant. Moreover, it is contained in the sewers of large cities where the disease is endemic, and it persists in damp cellars if they have once been infected. Hence sewer-gas and cellar-air may carry the disease. There is reason also for believing that a similar disease affects birds, fowls, and cats at times, and from them may be transmitted to man. These facts must be borne in mind in making the diagnosis.

The specific poison is the Klebs-Löffler bacillus and its toxin.

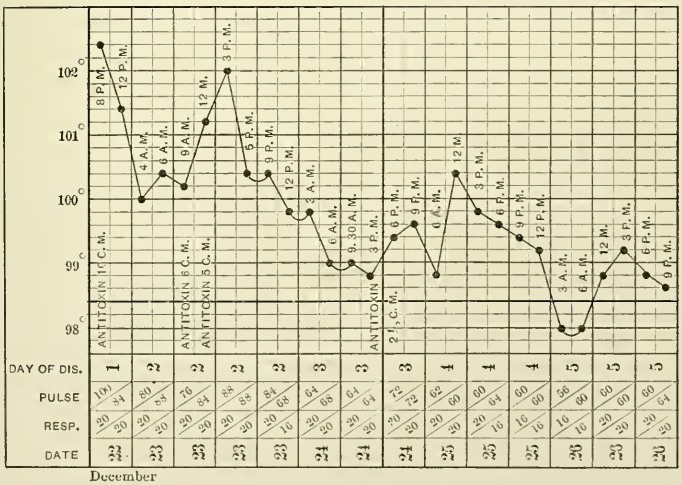
While children from one to six years of age are especially liable to it, no age is exempt—neither the newborn babe nor the very aged.

One attack does not protect a person completely against a subsequent attack.

The period of *incubation* varies from a few days to two weeks, or perhaps longer in exceptional cases. As a rule, it is less than a week. It is shorter when the poison is virulent, and when infection has been upon abraded surfaces.

The *onset* in mild cases is deceptively free from positive symptoms. The child is languid, perhaps slightly chilly, and has a little fever, with thirst, impaired appetite, and discomfort in swallowing. Unless the nature of the trouble is suspected the child is not thought ill enough to be kept in-doors. The throat is slightly inflamed, especially about the tonsils. The child may protest that there is no pain on swallowing. In from twelve to twenty-four hours from the onset, sometimes later, a grayish pellicle will be found upon the tonsils, and the cervical glands will be swollen.

FIG. 83.



In more severe cases the disease begins with chill or chilliness, followed by a rise in the temperature to 102° or 104°, sore-throat, and sometimes vomiting, though this is not so common as in scarlatina.

Convulsions and delirium may occur if the fever be high or the case malignant, but they are not common. Disgust for food makes it difficult to nourish the patient. Headache, thirst, and aching in the back and limbs may be complained of. Prostration is often very pronounced from the first.

Objective Symptoms. The characteristic false membrane appears first as a grayish pellicle upon one or both tonsils, and spreads thence to the soft palate and pharynx. The membrane soon becomes thicker and whitish in color; when fully developed it appears like white or grayish-white parchment, not lying loosely upon the surface, but embedded in the mucous membrane, the inflamed swollen edges of which rise above the false membrane, surrounding it "as the crystal of a watch is surrounded by the rim" (J. Lewis Smith[1]). As the membrane becomes older it may be brownish, or even blackish in color, if tincture of iron has been given. If it is forcibly torn from the underlying surface hemorrhage is excited and the membrane is reformed. As the membrane loosens spontaneously there is often marked inflammatory reaction at the edges of the surrounding mucous membrane, and in the tonsils there may be decided sloughing, with a dark, gangrenous appearance.

The temperature usually falls on the second or third day, but this does not indicate either a favorable or an unfavorable end. A temperature but little above normal is not uncommon in profound toxæmia.

Albumin is usually present early, and often tube-casts and renal epithelium also can be found. The submaxillary and cervical glands are swollen, and it may be difficult to open the mouth sufficiently to inspect the throat.

As pointed out by Buzzard and McDonnell, the patellar tendon reflexes are often abolished as early as the first day.

In *favorable cases* the membrane ceases to extend after three or four days; there is no extension to the larynx; the urine is free from albumin, or only slightly albuminous; and the pulse is not more than 100 to 120 and of good force.

In *unfavorable cases* the membrane shows a tendency to extend, either upward into the nasal fossæ, producing a thin, irritating, excoriating discharge from the nostrils, and rendering mouth-breathing necessary; or it may extend also to the ears through the Eustachian tube, or into the maxillary sinus; or the extension may be downward into the larynx, producing laryngeal stenosis. This is announced by hoarseness, with rapidly increasing difficulty in breathing. Inspiration is high-pitched, noisy, and difficult; the patient brings all the accessory muscles of respiration into play, the alæ of the nose play, the ribs are sucked in, and still he pants for breath. Every now and then a paroxysm of coughing produces cyanosis.

In other unfavorable cases the throat-symptoms are not dangerous, but uræmia develops. The urine is scanty, contains a large amount of albumin, considerable blood, and numerous blood, epithelial, and granular casts. There are œdema of the feet and puffiness of the eyelids. There is apt to be repeated vomiting; convulsions, followed by

[1] Keating's Cyclopædia of Diseases of Children, 1889, vol. i. 606.

coma, and death may end the scene, or the patient may slowly emerge from the dark valley.

In still other cases the diphtheritic poison affects the heart. The pulse becomes feeble and very frequent, the first sound very faint; acute dilatation of the right heart may occur. There may be faintness and a tendency to cyanosis on the slightest provocation, or attacks of sinking and faintness may come without warning; in still other cases sudden exertion induces paralysis of the heart, and death.

In some malignant cases the patient is overwhelmed by a large dose of the poison, and dies in from one to three days in collapse from acute toxæmia, without any special local symptoms to account for it. In others the false membrane extends rapidly over the fauces, pharynx, and nasal cavities to the larynx; death occurs from early obstruction, or, if it is postponed, there is extensive sloughing, with death from secondary blood-poisoning or septic pneumonia.

In exceptional cases the membrane is primary in the nares or larynx, or develops upon some abraded surface, as a burn, or in the vagina of a puerperal woman. It may also attack the mucous membrane of the eye or the seat of a recent operation. Diphtheria also occurs as a complication of other diseases, particularly scarlet fever.

The most frequent sequelæ are anæmia, albuminuria, and paralysis. The latter comes on in from one to two weeks after convalescence has set in, but it may appear much earlier, and in exceptional cases later. It may be marked simply by loss of the knee-jerk, which has been alluded to already in the symptomatology, or involve the palatal and pharyngeal muscles, causing nasal voice, difficulty in swallowing, and regurgitation of food through the nose, or there may be multiple peripheral neuritis.

Löffler's or the Klebs-Löffler Bacillus. This is found in diphtheritic pseudomembranes, especially in the deeper portions. It is not found in the blood.

Morphology. A bacillus 2 to 3μ long by 0.5 to 0.8μ broad, straight or slightly curved, with very many irregular forms. (See Fig. 84.)

The pseudo-diphtheritic bacillus resembles the genuine in all respects, except that it is not pathogenic. It seems to be an attenuated form of the former.

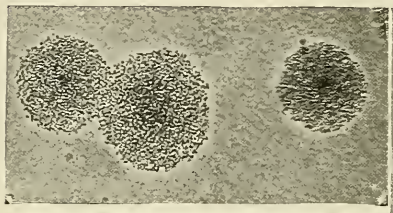
Biological Properties. It is facultative anaërobic, non-motile, and does not liquefy gelatin. It multiplies by fission. Stains with Löffler's blue. Certain points are stained intensely, almost black. It grows in nutrient gelatin, nutrient agar, or bouillon, but best of all in Löffler's blood-serum mixture (see page 243) at 35°. (Death-point, 58°, ten minutes' exposure.) It forms large, round, elevated colonies, grayish-white in color and moist. There is no visible growth on potato. Milk is a good soil. (See Plate VII., Fig. 4.)

On *inoculation* it causes a diphtheritic pseudomembranous inflammation. It generates a very poisonous toxin.

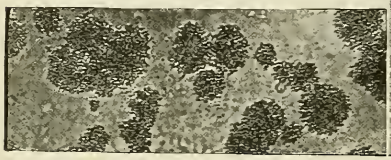
Diagnosis. Diphtheria is distinguished from ordinary *pharyngitis* by the presence of membrane. From *follicular tonsillitis* by the projecting mouths of the follicles containing a creamy-white exudate. Later

the exudate may cover the entire surface of each tonsil and be difficult to distinguish from false membrane. The points of distinction are that in the former the exudate lies upon the surface and can be brushed

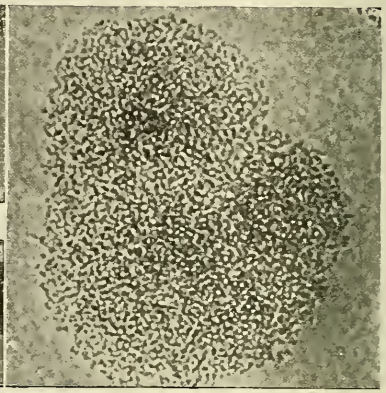
FIG. 84.



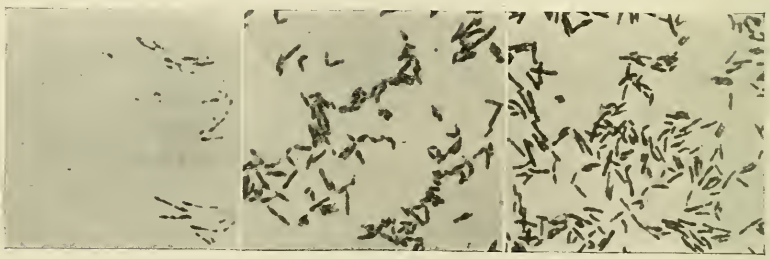
a. Colonies of diphtheria bacilli, × 160.



b. Colonies of pseudo-diphtheria bacilli, × 160.



c. Colonies of diphtheria bacilli, × 249.



d. Diphtheria bacilli, × 1000.

off without force and without leaving a bleeding surface; whereas in diphtheria the membrane is embedded in the mucous membrane and cannot be torn from it without force. A raised, red inflammatory border of mucous membrane at the junction of the patch is strongly suggestive of diphtheria. In tonsillitis there is no appearance of membrane upon the soft palate or pharynx. Furthermore, in tonsillitis the onset is attended with more fever and pain in swallowing than is true in simple tonsillar diphtheria. The existence of albuminuria and swelling of the cervical glands indicates diphtheria, and the absence of knee-jerk is an important but not constant diagnostic sign of diphtheria. The presence of the Klebs-Löffler bacilli in a culture from a suspected throat is proof of the existence of diphtheria.

Septico-pyæmia.

The clinical course of this infection and the bacterial causes have been considered in Chapter XVI. (Class III. of infections). It will be recalled that the phenomena may attend a number of the infections described in this and in previous chapters. When occurring in the

course of pneumonia, diphtheria, typhoid fever, etc., its causal origin is recognized by the methods discussed in the chapter referring to these infections. Septico-pyæmia caused by pyogenic organisms, the so-called "cryptogenetic sepsis," is recognized by bacteriological examination of the blood; by an examination of the morbid secretions, or by an examination of the products of inflammation. Bacteriological examination of the blood has its limitations. Usually only late in the course of the disease and in the more intense infections can the bacteria be found. Examination of the pus from foci of suppuration in the bones (osteomyelitis), in the joints (pyæmia), in the serous cavities (empyema, pericarditis, peritonitis), in the lungs (see Sputum), in the genito-urinary tract (see Urine), will show the infective micro-organism.

The causal micro-organism is detected by cover-slip preparations and cultures. (See Chapter XXI.)

Glanders.

A general febrile disturbance which attends this infection is similar to that of the infective granulomata (Class IV. of infections). In severe cases the symptoms are like those of an acute septicæmia. It is an infectious, constitutional disease, transmitted from horses to man, appearing in an acute and chronic form, and characterized by an eruption, ozæna, small tumors, ulcerations, cough, and death in coma or collapse in from one to four weeks in the acute form, or in three or four months in the chronic form, the symptoms in the latter resembling at times syphilis and at times tuberculosis.

The disease is rare in man. It may be acquired by direct inoculation of an open wound with the pus from a glanderous ulcer or nasal mucous membrane, or indirectly from infected straw or other material. The raw meat of a glandered animal also has infective power.

In *acute glanders* the onset is marked by headache, slight fever, and pains in the limbs. If a wound has been infected this becomes painful, swollen, and behaves like any poisoned wound. Sometimes a diffuse redness, resembling erysipelas, spreads from the infected point. Fagge refers to a case in which the first complaint was of pain in the side and dyspnœa, so that acute pleuropneumonia was suspected.

An eruption, consisting first of papules, which rapidly become flat vesicles and then pustules or bullæ, appears in the first day or two, or sometimes not for a week or even longer (Fagge). The bullæ or pustules rupture and give vent to a thin purulent discharge.

There may be hard, painful lumps in the muscles, with subsequent suppuration (farcy).

Ozæna is not always present. It appears in the second or third week of the disease. It consists of a mucopurulent, then purulent, fœtid discharge from the nose. The latter subsequently swells and becomes red and very painful. Ulcers and even necrosis of the septum are the lesions; the same catarrhal condition may exist in the throat, eye, larynx, and mouth, accompanied at times by ulcers and false membrane. The patient gradually sinks into a septicæmic condition, with irregular fever, dry brown tongue, albuminuria, delirium, coma, and collapse.

The duration of the acute form is from one to four weeks. Only one in thirty-eight cases collected by Bollinger ended in recovery.

In the *chronic form* there are ulcers upon the hand, face, forehead, or elsewhere. In other cases the lesions are abscesses in connection with joints which are followed by persistent fistulæ. In still other cases there is pustular eruption. Ozæna may or may not exist. In still other cases the prominent symptoms are cough, bloody expectoration, hoarseness, fever, and emaciation. Bollinger reports seventeen recoveries in a total of thirty-four cases of chronic glanders.

Diagnosis. Acute glanders is distinguished from rheumatism by the history of the case, the occupation of the patient, the existence of an open, irritable sore, and the fact that while the joints may be painful, they are rarely red and swollen, as in rheumatism. Subsequently the appearance of pustules, bullæ, and ozæna makes the case clear.

The same peculiar features serve to distinguish it from pyæmia, malignant pustule, and other infectious diseases.

In a suspected case of chronic glanders a correct diagnosis might be arrived at by inoculating a mule or a horse with the nasal mucus or pus from a farcy.

Bacteriological Diagnosis. The specific germ is the bacillus mallei. This is a short, non-motile micro-organism resembling the tubercle bacillus. It is 2 to 3μ long, and 0.3 to 0.4μ broad, frequently having spores on the ends. It stains readily with all the basic aniline dyes, although taking up the dyes irregularly.

The diagnosis is readily made by the method of Strauss. A portion of the suspected tissue or a culture from the lesions is inoculated into the peritoneal cavity of a male guinea-pig. If the case is one of glanders the testicles begin to swell in about thirty hours, and an orchitis with abscess develops. The diagnostic sign is the tumefaction of the testicles.

The Mallein Test. Mallein is the filtered products of the growth of the bacillus on fluid media. It is allied to tuberculin. The injection of it in a suspected case produces a reaction similar in its course to the tuberculin reaction if the case is one of glanders.

Cholera.

An acute, specific, infectious disease, endemic in parts of India, but occurring in epidemics elsewhere, characterized by the outpouring into the stomach and bowels of large quantities of a serous fluid resembling rice-water, which fluid is usually vomited and discharged from the intestines. It is further characterized by an algid state of collapse and by painful muscular cramps.

The specific poison of cholera is believed to be the comma-bacillus of Koch and its ptomaïne.

The native habitat of cholera is India, particularly the neighborhood of Calcutta ; here it is endemic, and thence it is liable to spread in successive epidemic waves along the lines of travel by sea and land, over the whole world. It is scarcely, if at all, contagious ; the poison is

contained in the vomit and dejections, which contaminate the drinking-water, food, and clothing. The cholera-bacillus preserves its vitality for long periods of time in water, especially if the water is slightly alkaline and contains vegetable matter, and in moist clothing, as rags.

The period of *incubation* is probably short in the majority of cases, lasting only a few days. Occasionally it is two weeks. There are usually no definite symptoms during this time, but there may be a sense of weakness, with loss of appetite and dyspeptic symptoms.

First Stage. The first stage, that of premonitory diarrhœa, is better regarded as the beginning of true cholera. It is characterized by profuse watery stools of a yellow or light-yellow color, and alkaline in reaction. They are accompanied by a rumbling noise in the bowels, but are passed without pain. From six to a dozen of these passages occur in twenty-four hours. The patient feels faint and exhausted after them, and may suffer with nausea, but vomiting is not usual. In severe cases there may be cramps in the calves of the legs. The voice is faint and husky, thirst intense, the tongue white and moist. The temperature is normal or slightly depressed.

This stage may last from two days to a week, depending upon treatment. In some cases it is wholly absent, and the patient is ushered abruptly into the second stage.

Second Stage. This usually comes on during the night. The patient is seized with vomiting, which is at first bilious, but the fluids rapidly lose all color and become like rice-water. The stools likewise resemble water in which meal has been stirred, or in which rice has been soaked—a semi-transparent fluid, with particles of epithelium resembling rice floating in it. This fluid seems to well up and regurgitate rather than to be vomited from the stomach, and to gush in quantities of a quart or two from the anus. Sometimes vomiting and diarrhœa occur at once. The patient has unquenchable thirst, and is tortured with painful cramps of the toes, legs, belly, and diaphragm. As the discharges continue the patient becomes more and more exhausted; the nose is pinched and twisted, the eyes sunken, the lips bluish, and the whole body may shrink beyond recognizable proportions.

The skin is cold and moist, the breath icy, and the temperature under the tongue is sometimes as low as 78° to 80° F. In the vagina and rectum it may be normal or slightly above normal. The patient, however, often has a sensation of heat. The urine is very scanty, containing albumin and sugar, or it may be suppressed. The pulse is very small and feeble, 100 to 120. The mind is clear, but the patient is listless, answering questions in an extremely faint voice and with manifest effort.

Third Stage. From this collapsed and algid condition the patient may slowly emerge, the skin becoming less cold, the cramps less severe.

A return of the secretion of urine is a hopeful sign. The reaction, however, may simply introduce a low typhoid condition, with fever, dry brown tongue, subsultus, low muttering delirium, and coma.

In some cases serum is poured out into the stomach and intestines and is retained there. The patient may be seized while walking with dizziness, faintness, extreme prostration, and early collapse.

In other cases the patient is smitten down with profuse vomiting and purging, dying algid and collapsed in a few hours, no reaction appearing.

In favorable cases the vomiting ceases, the stools become less frequent, and are tinged with bile and have a fæcal odor. The urine increases in volume, while the albumin diminishes. Convalescence is very protracted. Anæmia, great debility, feeble digestion, and sometimes obstinate diarrhœa delay complete recovery. Relapses are frequent.

In other cases reaction brings improvement in the gastro-intestinal symptoms, but uræmia develops, death following in convulsions or coma.

The most frequent *complications and sequelæ* are eruptions, chiefly erythematous, ulcerations and bed-sores, parotitis, and a painful tetanic spasm of the flexor muscles of the hands, forearms, legs, and feet, occurring between the tenth and fifteenth days of convalescence (Stillé).

Diagnosis. The chief points in the diagnosis from other affections are the knowledge of exposure to cholera; the character of the vomit and dejecta, which contain the comma-bacillus (for its detection see under Bacteriology); the cyanosis; the rapid development of collapse, with cold skin, icy breath, torturing cramps, and greatly shrunken visage and body.

Cholera morbus differs in that the stools remain turbid with bile or fæcal matter, or contain blood; they never present the rice-water appearance. Moreover, the passages are frequently preceded by colicky pains. Cyanosis and collapse are extremely rare. The stools do not contain the cholera-bacillus.

Other forms of acute *toxic gastro-enteritis*, whether from ptomaïne-poisoning or from corrosive poison, are to be distinguished by the history, the difference in the character of the stools, and the comparative absence of painful cramps in the legs, of cyanosis, and of collapse.

Bacteriological Diagnosis. Koch remarks:[1] "As cholera resembles in clinical symptoms cholera nostras, infantile cholera, certain forms of peritonitis, certain organic poisons, and poisoning by arsenic, it is important to attain some means of making a definite diagnosis."

Spirillum Choleræ Asiaticæ. The *Comma-bacillus.* The comma-bacillus of Koch is the specific causative agent of cholera. In a disease so wide-spread in times of epidemics, and so fatal, it is of great importance to be able to recognize the bacterium that produces it. Works on bacteriology give a fuller study than is permitted here, and should be consulted. This is more especially true because, while the bacilli, as found in the stools, can be stained quite easily, and may be recognized by expert microscopists, in the great majority of cases their recognition is only effected by bacteriological examination. They have no specific relation to dyes, as have tubercle bacilli.

Microscopical Examination. The cholera bacillus is a short, more or less bent rod, both shorter and thicker than the tubercle bacillus, and generally shaped like a comma. They are often found

[1] Zeitschrift für Hygiene und Infektionskranheiten, 1893, vol. xiv., No. 2.

placed end to end, and thus form a curve like a spiral. They are always present in the stools of cholera patients and sometimes in the vomit. They are particularly abundant in the mucous floccules of the rice-water discharges, and can be obtained from the linen soiled by the same. Cover-slip preparations are made from these portions by placing a uniform film on the slip, drying it in the air, and then passing it through the flame of a Bunsen burner or spirit-lamp.

The spirillum, or so-called "comma-bacillus," consists of a slightly curved rod, with rounded ends, 0.8 to 2μ long by 0.3 to 0.4μ broad. It is usually slightly curved like a comma, but may form a half-circle, or two may be joined like an S. Under certain circumstances they grow out into long spiral threads. By Löffler's method a single flagellum is found on the rods. It stains with anilines, but slowly. An aqueous solution of fuchsin (Zeihl's red) is the best. (See Plate III., Fig. 3, A; and Fig. 85). In addition to the cholera-bacilli, the bacillus coli communis and other intestinal bacteria are found. The cholera-bacilli lie in groups in the thread-like strands of mucus. They form in heaps, the bacilli lying in the same direction. Koch holds that this mode of grouping is characteristic and diagnostic. He further holds that if bacilli coli are in close proximity to numerous scattered bacteria resembling the cholera bacilli the case is one of Asiatic cholera.

FIG. 85.

Cholera spirilla grown on moist linen. × 600. (After KOCH.) Cultivated from the dejections after two days.

The bacillus of cholera nostras and one found in cheese by Deneke resemble the comma-bacillus in shape, though somewhat larger, but they have bacteriological peculiarities by which they can be differentiated.

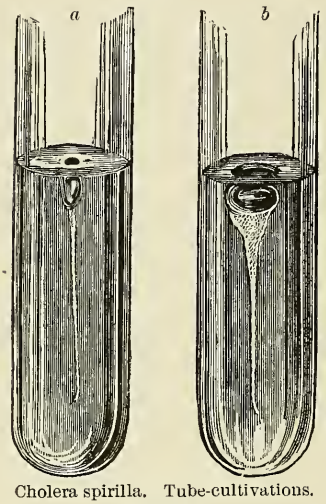
Biological Properties. Aërobic (fac. anaërobic), motile, liquefying.

CULTURES. *Growth.* Grows in ordinary media at room temperature; faster in oven. Does not grow except between 14° to 42° C. *Gelatin plates:* At the end of twenty-four hours small white colonies appear deep in the gelatin. These grow toward the surface and liquefy the gelatin in a funnel-form, which gradually deepens, and at the bottom of the colony is seen as a small white mass. Under low power the colony is white or pale yellow, margins uneven, texture granular, surface looks as if covered with bits of glass. When liquefaction begins a dim halo forms about the colony, which by transmitted light is roseate in hue.

Stab-culture in Nutrient Gelatin. Develops all along the puncture, liquefaction beginning near the surface, forming a funnel which enlarges, and finally the gelatin almost entirely liquefies. (See Fig. 87.) On potato a thin, transparent grayish-brown layer. Milk, bouillon,

blood-serum, are all favorable. In media with other bacteria it soon dies. Death-point, 52° 5′. In moisture it retains vitality for months, but is killed by drying.

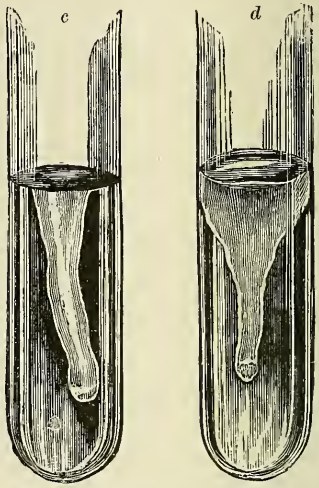
Fig. 86.



Cholera spirilla. Tube-cultivations. (Flügge.)

a, after two days; *b*, after four days.

Fig. 87.



Finkler and Prior's comma-bacillus. Cultivation in gelatin.

c, two days; *d*, four days old.

Peptone-cultivation. A small quantity of the dejection of some flake of mucus is inserted with a platinum loop into a sterilized 1 per cent. peptone solution. The solution is maintained at 37° C. The cholera bacteria are aërobic, and develop on the surface of the peptone, while the fæcal bacteria remain in the deeper layers. As soon as the peptone is cloudy a drop from the surface is examined microscopically. Within six hours the surface is overwhelmed with a pure culture of cholera bacilli. Later they are mixed with bacteria coli. The examination should be made from six to twelve hours after the peptone solution is inoculated. The peptone solution should be strongly alkaline, and a 1 per cent. solution of common salt should be added. Care must be taken to see that the solution contains sufficient soda. In plate cultivations the cholera-bacilli are overwhelmed by the fecal bacteria.

Agar-plate Cultivation. The growth is not so characteristic as it is in gelatin. The cholera-bacilli form large colonies of light gray-brown transparent appearance. Colonies of other bacteria are less transparent. The colonies can be obtained in from eight to ten hours after exposure to a temperature of 37° C. Microscopical examination of the colonies must be made.

Cholera-red Reaction. Cholera-cultivations contain indol and nitrous acid, and produce a red purplish color if sulphuric acid is added. This color is produced by other bacteria also, but by none other of the bacteria that are curved. Care must be taken to make the cultivations with suitable peptone and to have the sulphuric acid free from nitrous acid.

To determine its presence in the shortest time, inoculate diluted bouillon. After ten to twelve hours a wrinkled film has formed. Make another culture in the same way from this, then inoculate gelatin plates and use color-test on these.

Inoculation. The agar-cultivations are employed. They must be introduced into the abdominal cavity of the guinea-pig. The injection must not be made into the intestine, a matter which requires considerable practice. No other spirillum or curved bacillus produces the symptoms of cholera.

Acute Dysentery.

The fever which attends this infection is, from a clinical stand-point, the least characteristic symptom. It varies in part with the age of the patient. In the aged it is subnormal, normal, or moderate. In the young it is usually very high. It differs with the character of the infection. If a mixed infection prevails the temperature is not unusual.

The term dysentery is applied to an inflammation of the intestinal tract, chiefly the colon, which is attended by the symptoms of intestinal catarrh in intense degree, with mucus and bloody discharges and the general symptoms of *fever* and prostration, followed by extreme exhaustion, and at times the occurrence of abscesses in the portal circulation, or of paralysis, arthritis, nephritis, or profound anæmia. It was formerly thought to be an epidemic, mildly contagious disease. Although of frequent occurrence sporadically, it is especially common in jails and institutions, in camps, or where people are crowded together, when at the same time hygienic conditions are most unfavorable. It usually occurs in the summer or fall, and is attributed to the drinking of impure water. A form most common in the tropics is called tropical dysentery. Recent investigations have shown that catarrhal dysentery due to the above-mentioned circumstances may occur, and that in addition "tropical" dysentery, which is not confined to the tropics, is associated with inflammation and ulceration of the bowel, attended by the amœba dysenteriæ or A. coli.

Catarrhal Dysentery may be limited to simple inflammation of the intestine, or may be followed by ulceration. Its first symptoms are those of intestinal catarrh. There is indigestion, with loss of appetite, perhaps vomiting, and slight diarrhœa. These symptoms may be the immediate effect of the diarrhœa. At the end of three or four days a chill may take place, showing the setting in of an infection. The diarrhœa is attended by pain, at first seated around the umbilicus; it then becomes marked in the course of the colon. The movements are frequent, preceded by constant desire, and attended by extreme tenesmus. The stools, which were first fæcal and fluid, soon become scanty, and consist almost entirely of mucus and blood. The symptoms of local proctitis are severe; there is a sensation of a hot mass in the rectum. There may be strangury, and prolapse of the anus may ensue.

With the continuance of acute pain and frequent evacuations the skin becomes hot and dry; thirst, nausea, and occasionally vomiting occur. The temperature continues at about 103°; the pulse is rapid. The patient is weak and restless; the tongue is red and raw.

If the disease is severe from the start, or the course is unfavorable, the stools may contain pure blood, or they may be dark in color, and contain shreds of membrane. Pain and tenesmus disappear, and the evacuations become constant or involuntary. Restlessness is aggravated; the extremities become cold; mild delirium sets in. The tossing and restlessness are quite characteristic, and are attended by sighing and some dyspnœa. The pulse is rapid and feeble; the heart-sounds are weakened; the tongue becomes dry and brown, the mouth is parched, and thirst is intense; ulcers develop in the mouth and sordes collect around the teeth. The delirium increases to stupor, and from that to coma. The urine, at first high-colored and scanty, becomes bloody, and contains albumin and casts. Although the fever continues during this stage, the extremities become cool, perspiration breaks out over the forehead, and, instead of typhoid symptoms, the symptoms of collapse may ensue. If the disease is prolonged and the bowels are controlled, the symptoms of pyæmia may develop.

The anæmia that ensues is extreme, and there is great wasting. Convalescence is slow and may be attended by chronic diarrhœa. Before it is established ulcers of the skin may form on various parts of the surface of the body. Arthritis is of common occurrence, and paralysis may occur during convalescence on account of peripheral neuritis. Chronic dysentery may succeed the acute. It is thus seen that the attacks may be of moderate severity or extremely grave; during the course of the latter gangrene of the lower bowel may take place.

Amœbic Dysentery; Tropical Dysentery. This differs from catarrhal forms of dysentery in many respects. The onset may be abrupt or gradual, as in the previous form, with symptoms of intestinal catarrh. In most of the cases a frequent and painless diarrhœa follows a period of slight ill health. The diarrhœa alternates with short periods of constipation; the stools are watery and contain mucus, but no blood. The course of the disease is irregular. There may be intermissions and exacerbations of the diarrhœa without obvious cause. It may rapidly pass from one grade to another, or become chronic. One form is the gangrenous, which may scarcely be appreciated by the symptoms until the autopsy shows it to have been present. True relapses are common, and the tendency to chronicity is very great. The milder cases are attended by weakness, emaciation, and pallor; the expression is dull; the skin is dry and sallow; the tongue pale, flabby, and moist, slightly furred; the abdomen is normal or retracted; the temperature does not rise above 100°, and the pulse ranges from 70 to 90. Sleep is disturbed by frequent evacuations of the bowels. In the grave form the face is drawn, or cyanosed or flushed, the expression anxious; the mind is clear. Anorexia, intense thirst, and sleeplessness are present. The abdomen is greatly retracted, and there may be free sweating. The temperature is normal or subnormal, the pulse small and rapid. Progressive anæmia and loss of flesh are prominent and dominate the intestinal symptoms. The skin is dry and harsh, and of a dull greenish-yellow color if the cases are protracted.

The special features of amœbic dysentery are: 1. *The anæmia.* This is due to diminution of the red cells and the hæmoglobin, first, because of the action of the amœbæ upon the red blood-corpuscles, which they destroy; second, the direct loss of blood; and, third, malnutrition. The first is the most prominent.

2. *Diarrhœa* may be the only feature of the disease. It is characterized by great variation in character and frequency in all grades and during different periods of the disease. Intermissions and exacerbations may be observed at any time. The latter begin suddenly, and subside in the same manner. They last from two to ten days. The intermissions continue from one day to three weeks, during which the fæces are soft, but contain mucus. Councilman and Lafleur have observed this periodicity to be most marked in cases complicated with hepatic abscess.

3. *The Stools.* The stools are extremely variable according to the severity of the ulceration, and also vary in number and character from day to day in individual cases. In the gangrenous form they number thirty or forty in twenty-four hours at first, then decline, so that toward the end of fatal cases but three or four take place. At first the movements are small, and consist of mucus with more or less bright blood and small fæcal masses. As ulceration advances the stools change, they become more copious and watery, fæces are absent, blood is not so frequent. Shreddy masses of grayish or yellow color, mixed with mucus, appear. If there is sloughing, they become greenish or grayish, resembling spinach, or reddish-brown and very liquid or pultaceous. The odor is penetrating and offensive. Shreddy masses of necrotic tissue are discharged. Gray liquid movements, somewhat slimy, contain more pus than the others. Small opaque, or translucent, gelatinous grayish masses, one to three cubic millimetres in diameter, are found in the stools.

In the more moderate types the stools at the outset are like those of gangrenous dysentery if the attack is abrupt. If gradual, the stools are fæcal, liquid, containing mucus and streaks of blood and many of the gelatinous grayish masses. Stools of this character number from four to ten in twenty-four hours; this may continue for weeks. During the exacerbations the stools resemble those of the second period of the gangrenous form. In chronic dysentery there is not so much mucus or blood, except in exacerbations. The stools are of the consistence of thin gruel and have an earthy or dull-yellow color. Mucus is persistently present, however, in the intermissions, when the stools are soft and fæcal.

The *reaction* of dysenteric stools is generally alkaline.

Microscopical Examination. In the mucoid and bloody stools of the acute stage red blood-corpuscles, leucocytes, and large, round, or oval epithelioid cells are seen. The latter are often in groups of three or more. The nucleus is about the size of the red blood-corpuscle, the protoplasm granular. Their outline is sharp. They may be taken for amœbæ. They are non-motile and refract light less strongly. *Cercomonas intestinalis* is present, but bacteria are not abundant. In the later periods the cell-elements are less numerous; shreddy and

muscular detritus and bacteria are observed, with elastic-tissue fibres. Charcot's crystals and phosphates are seen. In chronic dysentery the cell-elements are still fewer and amœbæ are easily detected.

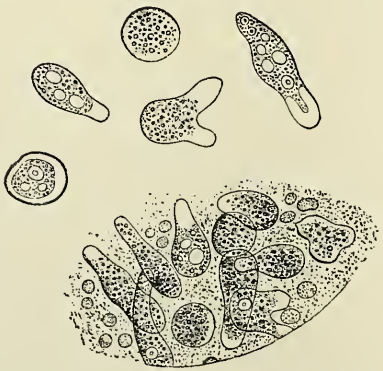
Amœba Dysenteræ. Amœbæ are found at all periods of the disease. They vary in different cases and at different periods in proportion to the severity of the intestinal ulceration. (See section on the Fæces.)

They are most abundant in the grayish-yellow gelatinous masses, next in the particles of clear or opaque mucus, and least in the fluid portions of the stools. In chronic dysentery they are found in all portions. In the intermission of the diarrhœa they may be found in the particles of mucus adherent to the fæces. They disappear as recovery proceeds, although they may be seen after the evacuations become normal. They vary in size and activity. They are more common in the alkaline and neutral stools. They are scarce and are rarely motile in acid stools. In the more active forms of the disease red corpuscles are seen.

For the detection of amœbæ the following should be observed: First, the stools should be passed in a warm bed-pan and kept at a temperature of 30° to 35° C. until an examination is made. Second, the stools must be examined before they become acid. Third, the gelatinous masses in the stools should be selected for examination. They contain amœbæ in greatest abundance. A magnifying power of four hundred diameters is required, although they may be seen with less. A $\frac{1}{12}$ oil immersion lens is the best.

Description of the Amœbæ. When *inactive* they are round or slightly oblong, highly refractive, and contain vacuoles of greater or

Fig. 88.



Amœbæ coli. (Hallopeau.)

less size. The latter are clear, and vary from small points to one-third of the diameter of the areola. The ectosarc and endosarc may or may not be sharply divided. If they are, the outer is hyaline or homogeneous, the inner is more refractive and contains vacuoles. They are difficult to recognize in this condition, being mistaken for swollen connective-tissue cells. The amœbæ frequently enclose red

corpuscles, pus-cells, blood-pigment, bacilli, and micrococci. In a fresh state the nuclei cannot be made out because they resemble vacuoles. The endosarc is not granular, is composed of a dense substance, and is highly refracting. When *active* the movement is characteristic. It may be slow or rapid, and is of two kinds, a progressive movement and one limited to the throwing out of pseudopodia. The movements appear to be rhythmical in some cases, occurring at regular intervals. The movement is sudden and characterized by change in form of the pseudopodia. The ectosarc and endosarc are clearly defined usually. The pseudopodia are hyaline and homogeneous, like the ectosarc. The amœba changes its position sometimes by enlargement of the pseudopodia, into which the inner contents of the older part follow. The movements are increased when the examinations are made on the warm stage. These amœbæ may be stained with various aniline dyes.

In *catarrhal dysentery* the stools are uniform in character, quantity, and frequency. The onset is sudden, and evacuations consist of bright blood and viscid, clear mucus mixed with fæcal matter. Soon they are composed entirely of mucus and a little blood. The mucus is viscid. In a week or ten days the mucus changes and becomes grayish-white in color—is less blood-stained and brown ; pultaceous or fluid fæcal matter appears in the stools. As the blood and mucus disappear formed fæces return. In the prolonged cases there are soft, yellowish-brown, or greenish stools in addition to the bloody mucoid stools. The frequency is greatest at the onset, and progressively diminishes until convalescence is established. The more frequent the evacuations the smaller the size of the stools. The mucoid stools are small, pultaceous, more bulky. On microscopical examination red and white corpuscles, cylindrical, epithelial, and oval epithelioid cells are seen. The latter are very characteristic, and occur singly or in groups. Bacteria are more common as improvement sets in. In the pultaceous stools the cell-elements are scarce. In *diphtheritic dysentery* the stools are watery. They resemble wheat-washings—evacuations such as are described in cases of *gangrenous dysentery*. They are grayish-green or reddish-brown and very offensive. Mucus is present in small amounts. At first unclotted blood is present, afterward minute dark-red clots are seen. Shreddy and finely divided material, gray or reddish-brown in color, is present, but there are no sloughs. The stools are not numerous at first, and average from seven to fifteen daily during the course of the illness. The quantity passed is small. Cylindrical epithelial cells are most abundant on microscopical examination. Red blood-corpuscles and leucocytes are observed, but fibrin constitute the larger portion of the stool. In all the stools bacteria are present in great numbers.

Other Symptoms of Amœbic Dysentery. Abdominal *pain* is constant ; it occurs in the early stages of both forms and in acute exacerbations. As the movements diminish the pain decreases. In the gangrenous form pain also disappears, although the intensity of the process is increasing. In chronic cases the colic is complained of during the exacerbations ; during the intervals a dull, aching, or burning pain is complained of in the upper quadrants. In all cases the

pain is cramp-like, boring or burning in character, and usually precedes and accompanies movements of the bowels. When severe it is general; but it is usually localized in the lower abdominal zone. Moderate tenderness on pressure is present in most cases along some part of the course of the large bowel. In catarrhal dysentery tenesmus is common; in the amœbic form it is infrequent. A burning sensation in the rectum and at the anus during and after the passage of fæces is generally complained of. Nausea and vomiting occur at the outset or at irregular intervals, being caused by improper food, or due to complications. Hiccough occurs in the terminal stages.

Fever. In amœbic dysentery fever is not a prominent feature, although there is usually a moderate rise in temperature. In the gangrenous form it is normal, or may be subnormal for days. Chronic dysentery is afebrile. In exacerbations of diarrhœa slight fever may occur. Complications cause a higher temperature. If fever is present it may be remittent or intermittent in character, or, if the illness is prolonged, first continuous, then remittent, and then intermittent. If the latter, the usual morning fall is observed, although an inverse temperature may be present. Rigors occur with the complications. Sweating is observed, with subnormal temperature, in the gangrenous form. In cases of abscess the fever is intermittent or remittent.

In *chronic dysentery* the skin is excessively dry. The circulation and respiration are influenced by the pyrexia. Anæmia is pronounced. When exhaustion ensues the pulse becomes more feeble, compressible, and rapid. The urine is albuminous, and often contains casts. In the gangrenous form there may be retention of urine.

The complications of amœbic dysentery are: 1. Hepatic abscess, or hepato-pulmonary abscess. 2. Peritonitis. 3. Hemorrhage from the bowels.

Hepatic Abscess. This complication may develop at any period of the disease. The time of the disease when it occurs cannot be determined definitely. In the subacute cases it is liable to develop from the fourth to the twelfth week. The abscess may develop on the convex surface of the right lobe of the liver near the coronary ligament. In these cases the lung also becomes involved. Councilman and Lafleur suggest that infection takes place by the peritoneum. (See Abscess of the Liver.) While the symptoms of abscess of the liver will be treated under the section devoted to liver disease, it is important to note that hepatic symptoms may occur in cases in which, on account of the mildness of the disease, the local bowel trouble may be overlooked entirely. (See Amœbic Abscess of Liver: Musser and Willard, Phil. Co. Med. Soc.) If the association of hepatic pain with fever and discharge of mucus from the bowels is observed, it is barely possible, even if an examination of the fæces cannot be made, that a hepatic abscess is present. If, in addition, cough and expectoration occur, involvement of the lungs is possible.

Hepato-pulmonary Abscess. The character of the expectoration points conclusively to the nature of the lung complication. After a period of dry, hacking cough, sudden expectoration of mucopurulent or bloody sputum takes place. It is of a dirty-red or brownish color,

not unlike anchovy sauce. From this time on this material is expectorated in varying quantities after a paroxysm of coughing. The expectoration is diffluent, tenacious, and frothy. It varies in color from bright red to russet-brown; it may be bile-stained. The sputa are alkaline; the odor is not putrid. At a later period they become more purulent, and contain less blood. The sputum separates into three layers: an upper frothy layer, a middle layer of turbid fluid, a thin layer of mucopus below. Large amounts may be coughed up in twenty-four hours; the sputa contain, on examination, blood-corpuscles, leucocytes, round alveolar epithelial cells and polyhedral, fatty degenerated cells, which look like liver-cells. Elastic-tissue fibres from the lungs are found with crystals of hæmatoidin and tyrosin, and Charcot's crystals. Bacteria are present. Amœbæ are constantly present. They vary in size and activity, but are larger than those seen in the stools. The sputum should be kept warm and examined as soon as possible.

Peritonitis. Peritonitis from perforation is not a common complication of amœbic dysentery, but takes place occasionally in the gangrenous form. Peritonitis without perforation may occur. The symptoms do not differ from peritonitis under other circumstances *Hemorrhage* from the bowel occurs and may be sufficiently profuse to cause death. This accident may occur in the course of amœbic abscess of the liver, as in a case reported by the author, in which there were no intestinal symptoms. Other complications which have been described under catarrhal and croupous dysentery are likely to occur in this affection.

The Diagnosis. The diagnosis of amœbic dysentery is made absolute by finding the amœbæ in the stools. The history and the course of the illness must also be taken into consideration, the characteristics of which have been previously detailed. The irregularity, and the intermittency of the diarrhœa, the infrequency of tenesmus, the moderate fever, the reaction of the stools, and their comparative freedom from bacteria, are further corroborative points.

The Plague.

This infection is seen in two forms: One, *pestis major*, is characterized by inflammation of the glands of the body, known also as *malignant adenitis*. Another, *pestis siderans*, is attended by intense septicæmia, with or without hemorrhages. Unlike the first variety, the glands are not enlarged. It is divided, in accordance with its special features, into septicæmic, pneumonic, gastro-intestinal, nephritic, and cerebral forms.

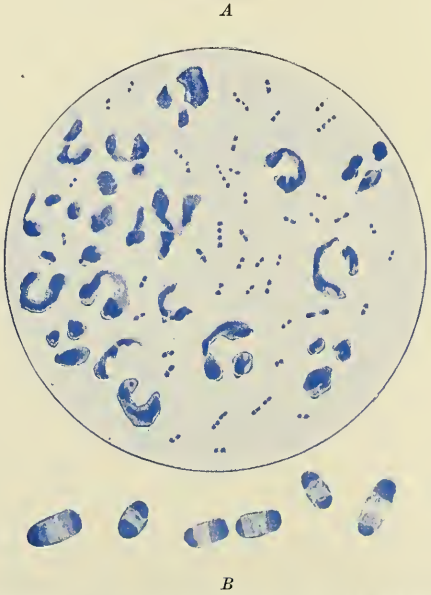
It is an acute, specific, infectious, and contagious disease, occurring in epidemics, characterized by high fever, sometimes by petechiæ and other hemorrhages, and, in cases which last long enough, by buboes. The death-rate is extremely high.

The plague is a disease of the East, being endemic in some parts of India, but epidemics have occurred in Italy, Russia, China, Turkey, England, and other parts of Europe.

The period of *incubation* is from two to seven days. The invasion is marked by lassitude, languor, headache, and dizziness. The stupid aspect and staggering gait may lead to the belief that the patient is drunk. Chill or chilliness soon supervenes, followed by fever, which often rises to hyperpyrexia, and is accompanied by unquenchable thirst, and sometimes nausea and vomiting. Delirium and a typhoid condition follow, with a marked tendency to failure of the circulation and collapse. If the patient survive until the second or third day, glandular swellings develop in the groin, or axilla, or angle of the jaw. Often they have to be sought for to be found. Sometimes they are prominent and are followed by suppuration and even ulceration. Carbuncles are much rarer manifestations than buboes. Petechiæ, vibices, hemorrhages into the kidney, and bloody vomit, occur in the worst cases.

Diagnosis. The diagnosis is based upon the history, the clinical course, and the results of bacteriological examination. The following description from Abbott enables the diagnosis to be readily made:

FIG. 89.



Bacillus of bubonic plague: *A*, in pus from suppurating bubo; *B*, the bacilli very much enlarged, to show peculiar polar staining. (ABBOTT.)

"This organism is described as a short, oval bacillus, usually seen single, sometimes joined end to end in pairs or threes, less commonly as longer threads. It stains more readily at its ends than at its centre. It is sometimes capsulated; is non-spore-forming; is aërobic, and is non-motile. It is found in large numbers in the suppurating glands, and in much smaller numbers in the circulating blood. (See Fig. 89.)

"It is demonstrable in cover-slip preparations made from the pus

and in sections of the glands by the ordinary staining methods. Yersin states that it retains its color when treated by the method of Gram, while Kitasato says that it at one time stains by this method and at another it becomes decolorized. Aoyama observed that those bacilli within the suppurating glands were decolorized, while those in the blood retained the stain when treated by Gram's method."

The duration is from six to ten days. If there is much suppuration, convalescence is prolonged.

Leprosy.

A chronic, specific, infectious disease, characterized by the development of tubercles, anæsthetic patches, and neuritis, and followed by ulceration and destruction of tissue. The disease occurs especially from puberty to the thirtieth year, and oftener in men than in women. It develops slowly and insidiously. Sometimes the first skin lesion is a crop of bullæ, suggestive of pemphigus. More commonly there appear reddish or violet-colored patches, varying in size from a quarter of an inch to two or three inches in diameter, and becoming of a darker hue later. The next step is the formation of nodules, which are characteristic of the disease. These may develop upon the patches already described, or in other places. They vary in size from a pea to a bird's egg or larger. They are most common upon the face and extensor surfaces of the arms, legs, fingers, and toes. The tubercles consist of an infiltration into the true skin ; they are raised, firm, relatively painless, and vary in color from red to copper. The face is characteristically distorted into a fierce expression (leontiasis). The tubercles may become absorbed and leave atrophic areas, but generally they break down into eroding ulcers, which slowly burrow and increase in extent, eating off a portion of the nose, fingers, hands, and feet, and exposing muscles, tendons, nerves, bloodvessels, and bone. Tubercles form also upon nerve-trunks, and ulcers upon the mucous membranes. (See the Nose and Larynx.)

In other cases, or in combination with the tubercles, especially upon the limbs and trunk, there are anæsthetic areas. Ulcers may follow without the previous occurrence of tubercles. With the anæsthetic patches are associated crops of bullæ, and neuritis.

The further peculiarities of the disease are: its long duration, its slow progress interrupted by apparent healing of some of the ulcers ; its afebrile course (the temperature is generally subnormal) ; its comparative painlessness, and the slight impairment of the general health.

Death results from gradual wasting, or is hastened by some intercurrent affection.

Diagnosis. The specific cause of the disease is probably the bacillus lepræ of Hansen. It is found in the thin pus of the ulcers and in the lesions themselves. It consists of rods 4 to 6μ long and 1μ broad, closely resembling tubercle-bacilli. They stain in alkaline fluids, but do not bleach after exposure to acids. Staining cover-slip preparations with the Ziehl-Neelsen fluid and decolorizing in acid and alcohol bring them out. They may be distinguished by yielding their color more

readily, and by taking easily aniline-dyes in simple watery solution (Von Jaksch). (See Plate III., Fig. 4, B.)

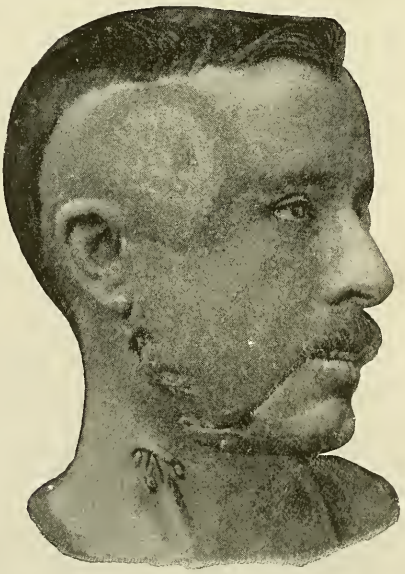
The *diagnosis* from a tubercular *syphilide* is made by the history of the case, the possibility of infection, the bacteriological examination, the slow progress, and the inadequacy of specific treatment. The presence of anæsthesia and of neuritis points to leprosy.

Actinomycosis.

The general symptoms attending this infection are like those due to suppurative infections. The fever is irregular, often *intermitting*. It is a specific infectious disease of cattle, occurring occasionally in man, attacking especially the lower jaw, lungs, and intestines, and characterized by a long duration, by the development of tumors and metastatic growths, and by *pyæmic* symptoms.

It is due to the actinomyces, or ray-fungus (see Fig. 91), which produces in cattle the disease known as big or lumpy jaw and swelled head. The fungus is conveyed in the food or drink, and gains entrance to the body through abrasions in the mouth or a decayed tooth, or is inspired into the lungs. Israel, Ponfick, and Boström have given us the greatest amount of information in regard to this parasite. It was discovered in 1845, in human beings, by B. v. Langenbeck, and in 1877, in cattle, by Bollinger.

FIG. 90.



Case of actinomycosis.

At the seat of invasion a slowly growing, slightly painful tumor develops. Bones are affected as well as soft tissues. These become swollen and suppurate, the fungus being at all times obtainable. The

fungous masses appear to the unaided eye as particles of yellow sand, and are greasy to the touch.

Pulmonic Form. Actinomycosis of the lung may be divided into three stages: a latent stage, when the lung proper is affected; an active stage, when extension to the pleura and chest wall takes place; and a final or chronic stage, when perforation and the formation of a thoracic fistula occur and the adjoining organs become affected. The symptoms of the first stage are those of chronic bronchial catarrh, with later the occurrence of the physical signs of consolidation, especially in the mamillary and axillary regions of the chest, in the middle zone of the thorax. The apices and bases are rarely affected primarily. The symptoms of the second stage are those of pleurisy, with adhesions and with or without effusion. At this time the disease may extend downward to the liver and peritoneum, or the pericardium may become infected. Fever and pain accompany these processes. On physical examination, in addition to the signs of the pulmonary and pleural conditions above mentioned, swelling of the thoracic wall will be observed, not unlike that of an empyema which is about to perforate. The swelling, which is at first dense, and hard, and red, becomes softer in small areas, and may fluctuate. Fluid, which is mucopurulent and shows the parasite, may be removed by aspiration. Repeated dry taps may occur before the needle secures the serous or sanguino-serous exudation in the pleura. The sputa at this time may accidentally show the parasite, although this is rare. The expectoration is mucopurulent, but it is said to never contain elastic fibres. The course of the disease at this time may extend over many months, in contradistinction to empyema on the one hand or carcinoma on the other. In the final stage ulceration of the swelling is seen in many places, fistula forms, and the disease extends to adjacent structures. Secondary infection may occur and symptoms of pyæmia develop.

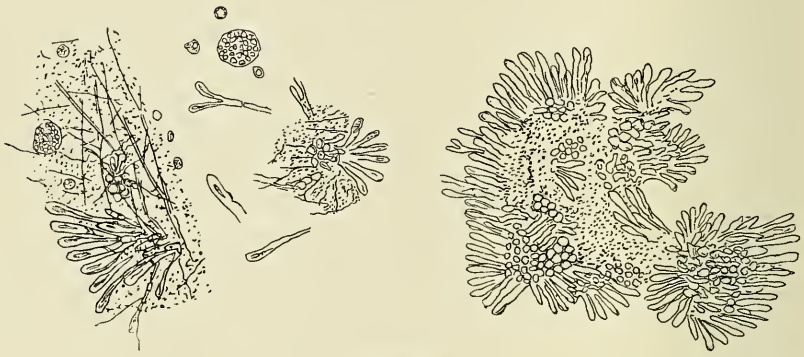
The masses which form upon the intestinal mucous membrane may lead to suppuration and perforation of the intestine. Metastasis to any organ may occur, with resulting local symptoms. The duration depends upon the organs involved in metastases. If metastases do not lead to early death, that result is brought about at the end of months or years by slow pyæmia, with resulting amyloid degeneration and its consequences.

It is usually associated with chronic inflammation and the production of pus. The pus is peculiar. It is thin and viscid. Small nodules of gray or yellow color, the size of a poppy-seed, can be seen by the naked eye when it is spread out on a glass. With a low power these particles are aggregations of spherules, which with a higher power are seen to be arranged in masses radiating from a common centre. Each separate spherule is pear-shaped. They have high refractive power. In the centre of the masses a network of fibres is seen. If the mass be broken up numerous club-shaped forms in the periphery are seen, while at the centre a sort of detritus alone is observed. The micro-organism belongs to the class of fission-fungi, and the club-shaped bodies are the degenerated forms. (See Fig. 91.)

Gram's method of staining brings out the threads of the network most distinctly. The centre is made up of a network of minute spheri-

cal organisms, with converging constituent threads. The whole is surrounded by a delicate envelope. The pear-shaped bodies may be defined by Weigert's process. Make a solution of 20 c.c. of absolute alcohol, 5 c.c. of concentrated acetic acid, 40 c.c. of distilled water, and sufficient French extract of litmus to color it ruby-red after repeated filtering. In this solution the cover-glass preparations are allowed to remain for an hour, and then rinsed with alcohol rapidly and placed

FIG. 91.



Actinomyces.

in a 2 per cent. gentian-violet solution for three minutes. The fluid should be boiled before use and filtered after cooling. The fungous threads are stained a ruby-red, while the central mass of actinomyces is colorless.

Diagnosis. Simple microscopical examination is usually sufficient to determine the nature of the fungus. The recognition is more positive if we bear in mind the peculiar character of the pus in which the nodules and the club-shaped forms are seen. It must not be mistaken for the radiating leptothrix threads found in the mouth. Pure cultures have been obtained resembling macroscopically the cultivation of the tubercle bacillus.

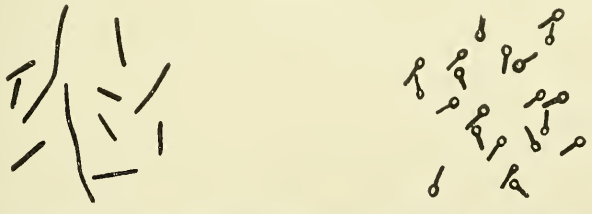
Tetanus.

Tetanus is an acute, infectious disease of the nervous system, the essential characteristic of which is persistent tonic spasm of the muscles of the jaws (*lockjaw*) and of the spinal and trunk muscles. The disease begins with the stiffness of the jaw, which steadily increases until, within a few hours, there is complete tonic spasm of the jaw. The neck-muscles, and then those of the spine and trunk, become rigid, so that the body is arched backward and may rest upon the heels and head (opisthotonos). The facial muscles share in the spasm, and by their contraction produce a horrid, grinning countenance (*risus sardonicus*). The contracted muscles become painful, and there is also epigastric pain. The rigidity is persistent, but is interrupted by exacerbations in which the phenomena already described are exaggerated, and, in addition, respiration is embarrassed, the face becomes livid, the skin bathed in sweat, and the patient is further distressed by increased

pain in the affected muscles. The body may be bent forward (emprosthotonos) or laterally (pleurosthotonos). The temperature is not constant. It may remain normal, be moderately elevated, or hyperpyrexia may be present, especially toward and after the end in fatal cases. The spasm ceases during sleep, but subsequently returns.

The cause of the disease is the bacillus of tetanus, which produces the convulsive poison *tetanin*. The bacillus is seen as a delicate, slender rod, with a terminal spore. It stains with aniline dyes and Gram's fluid. Cultivations may be made with the pus. It should be smeared over the surface of slanted agar-agar or blood-serum in a sterilized tube, placed at 37° C., for twenty-four hours, then heated to 80° C. in a water-bath from forty-five to sixty minutes. At the end of this time gelatin plates or Esmarch tubes are to be made from the growth in the heated tube; these are to be kept in an atmosphere of pure hydrogen at 20° to 22° C. Growth is favored by the addition to the gelatin of 2 per cent. of glucose. If the inoculation be made as a stab in a tube about three-quarters filled with gelatin, growth is seen only to within about 2 cm. of the surface of the media. Faint radiating striæ or thorn-like processes are seen. The development is rapid in agar-agar. After an exposure of thirty hours to a temperature of 37° C. the spores make their appearance. On gelatin the colonies are dense at the centre, with a more delicate periphery. The preparation becomes fluid, and gas is evolved. It is strictly anaërobic. The accompanying illustration from Abbott's work on *Bacteriology* shows its appearance.

FIG. 92.



A B

Tetanus bacillus. A. Vegetative stage, from gelatin culture. B. Spore-stage, showing pin-shape. (ABBOTT.)

Tetanus frequently follows an injury. Trismus neonatorum and puerperal tetanus are names given to special varieties which occur in new-born children and in puerperal women. Tetanus is much more common in men than in women, and Gowers states that three-fourths of the cases occur between the ages of ten and forty. It is much more common in hot than in cold countries, though cold is an exciting cause.

In traumatic and puerperal cases the disease usually develops in from a few days to two weeks from the time of injury or childbirth or abortion. In new-born children it occurs usually during the first week. It lasts from two to six weeks, but may be fatal much earlier, or, in rare cases, last even longer.

Tetanus must be distinguished from strychnine-poisoning. In the latter the jaw-muscles are never involved early, if at all, and the mus-

cles are relaxed between the paroxysms. It is distinguished from tetany by the history and the distribution of the spasm, which in tetany is confined to the extremities. Bacteriological methods should be resorted to.

Trichinosis.

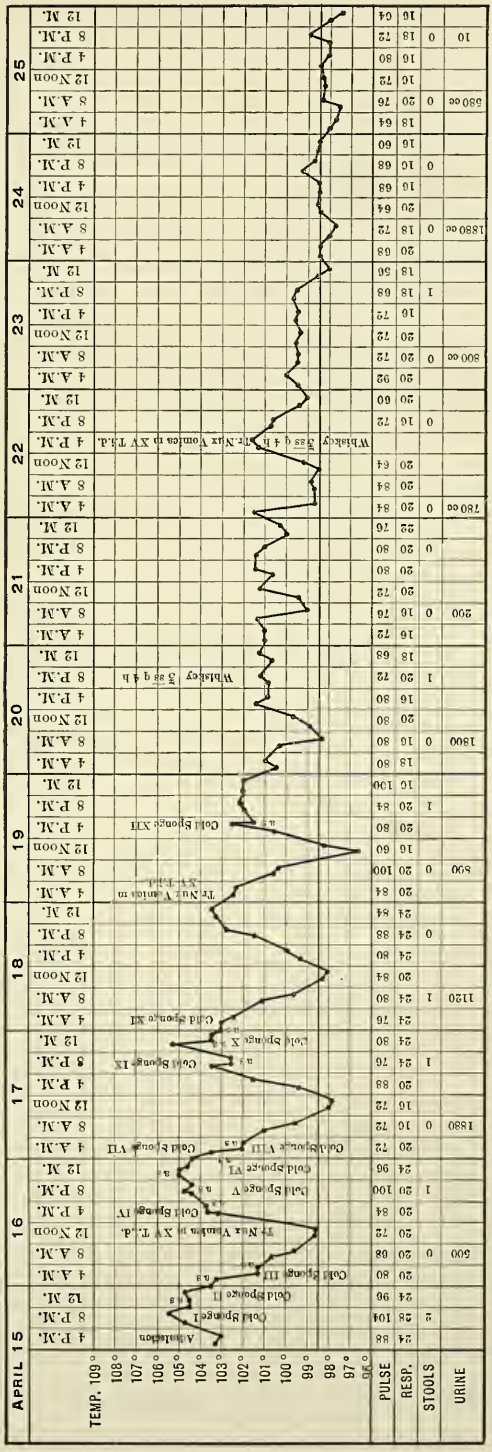
Until recently *fever* was not looked upon as an attendant of the gross parasitic invasion which is considered below. The study of a large number of cases shows that fever is present in various forms. In not a few, it is true, it may be very slight for a few days, and then fall to normal, and even, especially in convalescents, be strikingly subnormal. In other instances the temperature curve may be markedly intermittent. The chart from Osler's monograph shows this peculiarity. (See Fig. 93.) Finally, the fever-range is not unlike that of typhoid fever in many instances. Strümpell observes that the fever is seldom continuous for any length of time, and that its course is interrupted by frequent and prolonged intermissions. Niemeyer compares the curve to that of typhus, and Eichhorst to that of typhoid fever.

The infection is acute, caused by absorption of trichinæ spiralis, and characterized by fever, gastric and intestinal irritation, followed by pain and stiffness in voluntary muscles, œdema of the eyelids, face, and feet, by profuse sweating, and by death or tardy convalescence.

The trichinæ are absorbed by human beings through raw or imperfectly cooked food, often in the form of sausage. The trichinæ are encysted when absorbed, but within forty-eight hours they are liberated in the intestine and can be found adherent to the mucous membrane. In the course of six or seven days each liberated female worm produces about 180 embryos, which immediately penetrate the walls of the intestine and travel or are carried to all parts of the body, becoming in turn encysted.

Swallowing of trichinous flesh does not necessarily produce symptoms; the trichinæ may be destroyed in the stomach, or, if calcified, may pass through the intestine unchanged. When symptoms result the severity depends upon the number of trichinæ which become liberated. The symptoms are sleeplessness, lassitude, anorexia, nausea, vomiting, tenderness over the abdomen, and diarrhœa. Headache is a constant and marked symptom of invasion. Colicky pains attend the gastro-intestinal symptoms. These symptoms may not be marked in the beginning of the disease; or they may be so severe as to cause death in two or three days. If the patient survive, toward the end of the week the voluntary muscles become stiff, painful, and contracted. The muscles feel hard and swollen. The eyelids, face, and sometimes the feet become œdematous. Depending upon the muscles involved, there are interferences with the eye-movements, contractions of the jaw-muscles, difficulty in breathing or in swallowing, etc. The calves of the legs are especially involved. Recurrent œdema over the affected muscles, eyelids, and face is very common and characteristic. Profuse sweating also is very common, and at times there are severe neuralgic pains.

The fever is usually moderate, but it may be high. It follows the types described above. It is accompanied by malaise, with pains in


APRIL 15
16
17
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20
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22
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24
25
TEMP.
PULSE
RESP.
STOOLS
URINE
Intermittent fever in trichinosis. (Osler.)

FIG. 93.

the joints and muscles, preceding the true local muscle pain. The pulse is very frequent if trichinæ reach the heart. The later stages in fatal cases are marked by insomnia, delirium, stupor, and coma.

The duration varies from a few days to four or five weeks, or even longer. Muscular pains may persist for months after recovery. Death results from exhaustion, or from some complication, as pneumonia or ulceration of the large intestine.

The Blood. Brown, in studying Dr. Osler's cases, found an increase in the leucocytes, and on a differential count a great increase of the eosinophiles. The leucocytes were increased to 17,000 per c.mm. The eosinophiles increased from 2 per cent., the normal, to 37 per cent., and at one time to 68.2 per cent. In subsequent cases their average increase was as high as 48 per cent.

Diagnosis. The diagnosis is based upon the history, the peculiar muscular pains and swellings, the localization of the œdema, and the leucocytosis and eosinophilia. The muscles are swollen and hard, painful on pressure, and contracted. There is no involvement of the joints, an important point in the diagnosis. The œdema (see Chapter XI.) is seen in the eyelids and over the eyebrows. It is of common occurrence over the swollen and tender muscles. It is distinguished from *typhoid fever* by the presence of vomiting, and œdema of the face and eyelids, the development of muscular troubles, by the absence of hebetude, delirium, and other typhoid symptoms, and absence of the characteristic eruption and enlargement of the spleen.

Muscular rheumatism is distinguished by being limited to one part, as the lumbar region, arm, or chest; by its appearance following exposure to draught; and by the fact that it is not preceded by nausea, vomiting, and diarrhœa, nor accompanied by œdema.

CHAPTER XXI.

THE DATA OBTAINED BY OBSERVATION—(*Continued*).

Exploratory puncture or aspiration for diagnosis: Instruments. Preparation of instruments. Preparation of skin. Point of puncture.—*Exudations* (Pus. Seropus. Gangrenous débris. Blood. Serum. Chyle): Pus. Blood-corpuscles Bacteria. Protozoa. Vermes. Crystals.—*Chemical examination:* Seropurulent exudations. Putrid exudations. Hemorrhagic exudations. Serous exudations. Chylous exudations. Pleural effusions. Transudations.—*The contents of cysts:* Hydatid, ovarian, renal, pancreatic.

THE EXAMINATION OF EXUDATIONS, TRANSUDATIONS, AND CYSTIC FLUIDS.

Exploratory Puncture or Aspiration for Diagnosis. The presence or absence of fluids in the natural cavities of the body, as the pericardium, the pleura, or the abdomen, or in the gall-bladder, must frequently be ascertained by means of puncture or aspiration. The fluid is secured at the same time by the puncture for examination. The fluid of tumors or cysts is likewise withdrawn to complete a diagnosis by determining its chemical, microscopical, or bacteriological character. Certain rules of procedure are necessary, and, as they are common to the method in whatsoever situation employed, may be considered in this section.

The Instruments. If it is the desire of the observer to determine the presence of fluid, an ordinary grooved needle may be used. If, however, fluid is to be obtained for examination, a syringe or aspirator must be used. An ordinary hypodermatic syringe, or the syringe of Pravaz, may be used if the needles are long enough. A special aspirator made for diagnosis by instrument-makers is the best. The needles are sufficiently long, the barrel large enough to hold sufficient fluid for any method of examination. If the diagnosis is to be followed by treatment by aspiration, the apparatus of Dieulafoy, or any equally perfect apparatus, may be used at once.

Preparation of Instruments. The instruments should be sterilized in a steam sterilizer, or boiled. This does not apply to the needles alone, but every portion of the instrument should be cleansed, because, for instance, the contents of the barrel of the syringe pass through the needle. After sterilization they should be carried to the patient in sterilized test-tubes plugged with cotton-wool. When not in use the needles should be kept in absolute alcohol and the syringe in carbolic acid solution, 1 : 20. Before using, the carbolic acid should be washed from the syringe and needle with boiling water; they are then to be sterilized as described. Unless the carbolic acid is removed from the syringe its presence may serve as an antiseptic or disinfectant,

and thus interfere with the culture-tests, to which the material drawn is to be subjected.

Preparation of Skin. The skin should first be cleansed with soap and water, then with alcohol, then with a solution of carbolic acid, 1 : 20, or of the bichloride of mercury, 1 : 1000. After thorough cleansing the parts should be kept covered with a towel soaked in bichloride solution until the time of operation. At the time of puncture the surface should be made anæsthetic by ethylene chloride, the rhigolene spray, by ice and salt, or, in adults, by the Schleich method of subcutaneous anæsthesia. Care must be taken, if the patient is aged or poorly nourished, or the skin œdematous, not to freeze the skin too much, on account of the danger of local gangrene.

The Point of Puncture. The points selected for aspiration depend upon the cavity to be explored or the situation of the cyst.

The Pleura. To withdraw the fluid within the pleura it is best to select a point for aspiration in one of the lower interspaces of the chest, because the fluid is more likely to accumulate in this position and because complete aspiration can there be performed if necessary. The sixth or seventh interspace in the anterior axillary line, or the eighth or ninth interspace in the posterior axillary or scapular line, may be selected. On the right side the upper interspace of the two should be chosen on account of the position of the liver. If the contents tend to point or break out at any particular spot on the surface of the chest the puncture may be made in this area. In suspected loculated empyema or effusions the point of puncture should be at the site of greatest dulness and least fremitus.

The Pericardium. For aspiration of the pericardium three points of election have been recommended : First, the usual position of the apex-beat, in the fifth interspace, inside of the midclavicular line ; second, the space between the ensiform cartilage and the left seventh cartilage, the point advised by Roberts ; third, Rotch has tapped the fifth right interspace a number of times on the cadaver, and thinks that this situation is a proper one on the living subject. The writer has aspirated the pericardium in several instances inside of the normal position of the apex. Care must be taken to insert the needle slowly and with the point directed downward and toward the left axilla when this position is selected.

The Abdomen. It should be remembered that no attempts at puncturing the abdomen should be made if pus is suspected, unless preparations have been made to perform laparotomy at once. Indeed, this exploratory operation is performed with so little detriment to the patient by modern surgeons that, on the whole, it should be advocated instead of puncture. There are times, however, when the latter must be resorted to. The writer has performed it in a number of instances —always refusing to do so in cases in which pus was probably present in the peritoneal cavity, or in tumors, or in organs the seat of suppuration—without any danger having ever arisen. Explorations of this character are probably more feasible in connection with diseases of the *liver*. It does not appear to be harmful to insert needles into that organ, and valuable information is often gained thereby.

In aspiration of the *abdomen*, to determine the character of peritoneal contents, the median line should be selected for the puncture. The bladder must be emptied and a point midway between the umbilicus and pubes selected.

The Vertebral Canal. *Spinal* or *Lumbar Puncture.* Proposed by Quincke, the procedure has been carried out by many clinicians and has proved to be a means of corroborating and even establishing a diagnosis. Cerebral lesions are diagnosed and intracranial pressure relieved because of the continuity of the spaces in the brain and the spinal canal. (See Cerebro-spinal Meningitis.)

Method. The patient should lie on the right side, with the knees drawn up and the left shoulder turned forward. The puncture is made by an antitoxin needle or the needle of a large hypodermic syringe, which may then be used to withdraw the fluid. The syringe itself may be removed and the fluid allowed to ooze through the needle drop by drop. A needle 4 cm. in length and 1 mm. in diameter is suitable for infants; a longer needle for children over ten and adults. The point selected for puncture is midway between the third and fourth or fourth and fifth lumbar vertebræ, below the spinous process, a little to one side of the median line. The thumb of the left hand of the operator placed between the spinous process may be used as a guide. If the needle is inserted to the right of the median line, preferably on this side, it should enter 1 cm. from the median line, on a level with the thumb, and be directed slightly upward and inward. At a depth of 3 or 4 cm. in children and 7 or 8 cm. in adults the canal is entered. The fluid oozes drop by drop, and should be collected in a sterilized test-tube. It should not run down the sides of the tube. Five to fifteen cubic centimetres should be withdrawn.

The fluid is examined chemically, bacteriologically, and microscopically. Sugar has been found in brain-tumor and not in meningitis; albumin is said to be less in the former than in the latter. In tuberculous meningitis the fluid is usually clear and limpid; in other forms cloudy and turbid. Pus has been withdrawn in leptomeningitis. Blood may be found in hemorrhage into the lateral ventricles. The respective affection is distinguished by the results of bacteriological examination.

Cover-glass preparations are made of the fluid and cultivations taken at once. In purulent meningitis streptococci, staphylococci, the pneumococcus, and the meningococcus (diplococcus intercellularis) may be detected. In tubercular meningitis tubercle bacilli have been found, especially after sedimentation. After the fluid has been twenty-four hours in a conical glass the fine clot which forms should be examined for bacilli. The absence of bacilli does not exclude tuberculosis. The positive result, however, is diagnostic.

Inoculation, as in a case by Lafleur, will cause tuberculosis in a guinea-pig, and is diagnostic. A clear fluid does not exclude purulent meningitis; usually, however, the fluid is purulent, turbid, or rich in leucocytes.

Sometimes, although the canal is entered, fluid is not secured, because the needle enters pseudomembrane, thick pus, or gelatinous fluid, or because fluid is retained in the lateral ventricles.

Cysts or tumors, with fluid contents, should be punctured over the point which presents externally, at which place it is evidently in closer proximity to the external wall.

The Spleen. The *spleen* has been punctured for therapeutic and diagnostic purposes. If the organ is hard, as in chronic malaria, it may be done without danger; but if it is enlarged and soft, as in infectious diseases, such as typhoid fever, it is hardly justifiable to puncture it, because of the danger of subsequent rupture. Risks attend the puncture of other organs, as the kidney. The writer has seen a serious hemorrhage follow such puncture, and, of course, septic inflammation may arise. Exploratory operation is more suitable for determining its condition.

The Examination of Fluids and Discharges. While the fluids to be examined can be obtained by the above-mentioned method, it sometimes happens that they are discharged spontaneously, as in the case of an empyema.

The following general methods apply to the examination, in whatever way material is obtained. When derived from the natural cavities they are known as exudations or transudations. Fluids are also obtained from cysts, but do not require different methods of examination.

The naked-eye appearances are first noted; then microscopical examination with and without staining is resorted to. Chemical examination is also required. Often culture-preparations and inoculations must be resorted to, as in the case of pus or of serous exudation.

The Exudations.

They may be composed of pus, seropus, gangrenous débris, blood, or pure serum or chyle. When pus, seropus, or putrid fluid is withdrawn, it implies absolutely an inflammatory origin. Blood and serum may be associated with inflammation, simple or infectious; but may also point to impediments in the general or lymphatic circulation. Blood or bloody serum is thought to be of tuberculous or cancerous origin. Its absence does not imply the absence of either disease. A chylous exudation is usually due to obstruction of the lymph-channels.

Purulent Exudations.

Pus ranges in color from gray to greenish-yellow. It is turbid, of high specific gravity, and alkaline. It varies in consistence. When standing after removal it separates into two layers; the upper layer is light yellow and transparent, and the lower opaque. Pus may be mixed with blood, and is then reddish-brown. (See Abscess of the Liver.) When it has undergone decomposition it is thin, green, or brownish-red, of a penetrating odor.

Microscopical Examination: *White Corpuscles.* If the specimen is fresh the cells exhibit the movements that are common in leucocytes. If a solution of iodine and iodide of potassium is added to them they change to mahogany color. If the pus is old and the

cells are dead, they are shrunken and granular. Enormous giant-cells and cells loaded with fat are seen in pus.

Red Corpuscles. In fresh pus red corpuscles are also seen along with blood-pigment or hæmatoidin-crystals.

In addition to the corpuscles free *fat-globules* and fat-particles are seen. *Epithelium* is rarely seen. In the pus from the pleural cavity, if cancer is present, the vacuolated epithelial and endothelial cells sometimes seen in cancer may be observed.

Bacteria. Micro-organisms are always detected with the aid of staining-methods. (See Chapter XVII., Bacteriological Diagnosis.) The micro-organisms are usually the determining cause of the suppuration. Suppuration, however, may be caused by chemical substances, although this is at least of rare clinical occurrence. Of the various fungi found the micrococci and bacilli are the most numerous. The commonest of these are the *staphylococcus pyogenes aureus* and *streptococcus pyogenes;* the *amœba dysenterica,* in abscess of the liver and secondary abscess of the pleura and lung. It was found in an abscess of the jaw by Flexner. For further description of the pyogenic micro-organisms, see below and Chapter XVI., The Infections.

The Pyogenic Bacteria. 1. Staphylococcus Pyogenes Aureus. This micro-organism is found in acute abscesses and boils, sometimes also in infectious osteomyelitis and ulcerative endocarditis. In addition to other portals it may enter the tissue through abrasions or the hair-follicles.

Morphology. In cover-glass preparations they appear as small round bodies scattered among the pus-cells, rarely within them, single, in pairs or in clusters. They stain readily with the basic aniline dyes. (See Fig. 94.)

Biological Properties. It is aërobic, facultative anaërobic, grows in milk, meat-infusions, gelatin, or agar at 18° C. Death-point is 56°

Fig. 94.

Pus with staphylococcus. × 800. (Flügge.)

to 58° C. after ten minutes' exposure. *Growth.* Make plate-cultures on agar-agar. After twenty-four hours in the incubator the plate will be studded with yellow or orange-colored colonies, round, moist, and glistening. In a gelatin stab-culture liquefaction occurs in thirty-six to forty-eight hours along the puncture, forming a funnel. The whole mass gradually liquefies. At the bottom of the funnel the microbes

collect as an orange-colored mass. On potato it grows as a brilliant, orange-colored, somewhat lobulated layer. The growth gives off an odor of sour paste. (See Plate VII., Fig. 3, and Plate III., Fig. 2, *b*.)

2. Staphylococcus Pyogenes Albus. It is also found in acute abscesses, but less often than the "aureus," and is less virulent.

It is morphologically identical with the "aureus," but develops no pigment. The surface-cultures are milk-white, and the mass at the bottom of the liquefying gelatin is white.

3. Staphylococcus Epidermidis Albus (Welch) closely simulates the staphylococcus pyogenes albus. It is the most common micro-organism on the surface of the body, and is often present in parts of the epidermis too deep for disinfection, save by heat. It is supposed to be the usual cause of "stitch-abscess."

4. Streptococcus Pyogenes. It is found in acute abscesses, *erysipelas*, otitis media, puerperal metritis, *infectious endocarditis*, pseudo-diphtheria, scarlatinal angina, and most purulent inflammations of a phlegmonous character. It is the organism most commonly found in inflammations having a spreading tendency.

Morphology. Cover-glass preparations show spherical cocci of varying sizes, which form chains of four to twenty elements, the chains often forming tangled masses. It is stained by the basic anilines or by Gram's method. (See Fig. 95.)

Biological Properties. Grows in most media at a temperature of 16° to 37° C. (best 30° to 37°), but not on potato. It is facultative anaërobic, and does not liquefy gelatin. On plates it forms a flat, transparent disk of about one-half millimetre diameter. In stab-cultures it grows all along the puncture and forms a white opaque granular column. The death-point is 52° to 54°, ten minutes' exposure. (See Plate VII., Figs. 1 and 2.)

Fig. 95.

Streptococcus pyogenes in pus. × 800. (Flügge.)

Inoculated, it causes erysipelatous or phlegmonous inflammation.

5. The Tubercle Bacillus. This is seen at times in pus removed from phthisical cavities, and the pus of abscesses, particularly about glands. It may be detected by methods of staining adopted in the examination of the sputum. Pus may be of tubercular origin, and the micro-organisms may not be detected by the usual microscopical methods. Its absence, therefore, does not imply the absence of tuberculosis. Culture-methods and inoculation should be resorted to, particularly the latter.

6. The Bacillus of Syphilis. The pus under these circumstances is usually derived from ulcers or inflammations, or from secretions about the vulva or prepuce. The actual relationship to syphilis has not been demonstrated.

Lustgarten's method is as follows: After immersion for twenty-four hours at the ordinary temperature in the gentian-violet fluid of Koch-Ehrlich, the cover-glass preparation is removed and washed for a few moments with absolute alcohol. It is then placed for ten seconds in a 1 per cent. or 2 per cent. solution of permanganate of potash; a watery solution of pure sulphurous acid is then poured over it, after which it is washed in water. If the preparation still shows its color, it must be reimmersed for a few seconds in the potash solution and then in the sulphurous acid, and again washed with water.

7. Actinomyces.

8. The Bacillus of Glanders.

9. The Bacillus of Anthrax.

10. The Bacillus of Leprosy.

11. The Bacillus of Tetanus.

12. The Bacillus of Influenza. (See Sputum.)

13. The Micrococcus Lanceolatus. The Pneumococcus. The pneumococcus is often found in the pus of empyema and pericarditis, whether from the pleural cavity or after it has burrowed from this situation. It occurs in cerebro-spinal meningitis. It is easily detected by the usual staining-methods (for which see Sputum).

14. The Bacillus Coli Communis. The bacillus coli communis is found more commonly in infections within the abdominal cavity. (See Fæces.)

15. The Gonococcus. It is constantly present in virulent gonorrhœal pus, usually within the pus-cell or attached to the surface of epithelial cells. *Morphology.* Micrococci, usually joined in pairs or fours, flattened and separated, when stained, by an unstained intercellular space. Stains easily with anilines—not by Gram's method.

No other cocci are of the same shape, and at the same time within the cells, except one which, however, stains by Gram's method. (See Plate III., Fig. 3, *b*.)

Growth. Does not grow readily on ordinary media, but can be cultivated on blood-serum and other special media, such as urine, agar, etc.; 30° to 40° C. is best, and a moist atmosphere is needed. Growth is slow and often fails. Forms a thin, scarcely visible layer, with smooth, shining surface, grayish-yellow by reflected light—is aërobic. (See page 308.)

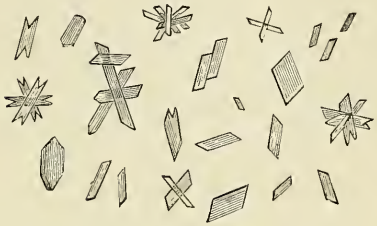
Protozoa in the Pus. Cercomonads have been observed in the pus of an empyema, probably from the lungs. Flexner has found the amœba dysenterica in the pus of an abscess of the jaw. It is found in abscess of the liver and secondary abscess of the lung. (See Sputum and Fæces.)

Vermes. Filaria have been found in abscess of the liver. In the suppuration of hydatids the pus contains membrane and hooklets.

Crystals. Crystals of *cholesterin* are found in the pus from cold abscesses, suppurating ovarian cysts, and fœtid discharges. They are similar to the crystals described under sputum.

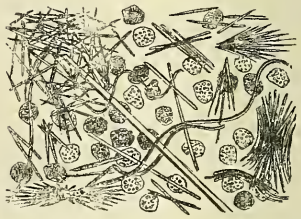
Hæmatoidin-crystals indicate a previous hemorrhage; they are most frequent in suppurating hydatid cysts. (See Fig. 96.) *Fatty needles* are found in old pus and gangrenous exudates. (See Fig 97.) *Triple phosphates* are frequently seen in pus, and are of the same appearance as the phosphates in the urine. The carbonates and phosphates are seen in fœtid pus.

Fig. 96.



Rhombic crystals of hæmin. (Charles.)

Fig. 97.



Pus from putrid empyema. (Eye-piece III., obj. 8, A. Reichert.) Shrunken leucocytes. Fat-crystals. (Von Jaksch.)

Chemical Examination of Pus. This does not yield any information of diagnostic value.

Serum-albumin, globulin, and peptone are detected by methods employed in the examination of urine. Fresh pus contains sugar. After being boiled with an equal weight of sulphate of soda and filtered the filtrate is examined by the reagents used in examination of urine for sugar. Pus also contains bile-pigments and biliary acids, cholesterin and salts of sodium and the fatty acids in jaundice. Von Jaksch has found acetone in pleural exudations.

Seropurulent Exudations. They resemble purulent discharges, chemically and morphologically. They point to antecedent inflammation.

Putrid Exudations. The exudations are brown or brownish-green in color. The odor is penetrating and offensive. They are usually alkaline in reaction. On *microscopical examination* old leucocytes and crystals of fat, cholesterin, and hæmatoidin are seen; fission-fungi of various forms are seen. (See Figs. 96 and 97.)

Hemorrhagic Exudations. Hemorrhagic exudations contain red blood-corpuscles and hæmoglobin in large amount. Fatty endothelial cells are found. Quincke states that when the glycogen-reaction is shown, if the fluid is from the pleura, carcinoma is probably present. A positive diagnosis depends upon the discovery of the epithelial cells (see page 364), which are seen in cases of cancer. Hemorrhagic exudations in the pleura are due most frequently to cancer, to tubercle, or to scurvy. To determine its exact nature (as to tubercle), inoculation and cultures are sometimes necessary.

Serous Exudations.

The fluid is clear and light yellow or straw-colored. On standing a white fibrinous clot is deposited. On *microscopical examination*, red blood-corpuscles, leucocytes, fatty globules, and endothelial cells are found. They may be bunched in groups or scattered about. The micro-organisms, if present, are detected with difficulty. If ulcerating tuberculosis of the pleura is present the bacillus may be found, but tuberculous pleurisy may exist without ulceration, and hence the fluid is clear of the bacillus. Cholesterin-crystals are found in old serum. On *chemical examination* the fluid contains more than 3 per cent. of serum-albumin and globulin; peptone is absent in pleural exudations; sugar in small amount and acetone are found.

The specific gravity of the fluid is above 1018.

Chylous Exudations. True chyle is found in fluids of low specific gravity. Such an effusion is rich in fat and is due to leakage of lymphatics into the peritoneal cavity. It is known as a chylous effusion. Chyliform effusion is a term applied to the second variety of effusions mentioned in this section. The fluid has the property of chyle. Sometimes in peritoneal exudation, particularly if the patient has been upon a milk-diet, the fluid contains fatty matter, which gives it a milky appearance. The same character of fluid is seen in obstruction of the thoracic duct.

Special Effusions. Effusions in the Pleura. It is of the greatest importance to distinguish the various forms of infection. Bacteriological examination is often necessary. In purulent exudation, if micro-organisms are absent (staphylococcus and streptococcus), it is probably tuberculous; serofibrinous exudations are usually free from fungi. When the *micrococcus lanceolatus* is found it is of favorable prognostic omen.

To distinguish the *effusion of inflammation* from that of *transudation* (obstruction) the specific gravity is of service. In the inflammatory effusions the specific gravity is high; they also contain a large amount of fibrin and more than 3 per cent. of albumin.

Transudations.

This class of fluids is serous, bloody, or chylous. The specific gravity is lower than in inflammatory effusion. The color is light and the reaction usually alkaline. On microscopical examination but little is found. In pleuritic effusions there may be considerable endothelium, which, if mixed with blood, may be due to carcinoma. Serum contains albumin and sugar, the former in great excess. Peptone is always absent. The fluid coagulates with difficulty on boiling.

Runeberg[1] lays stress upon the diagnostic importance of the amount

[1] Runeberg (J. W.): On the Diagnostic Importance of the Amount of Albumin in Pathological Transudations and Exudations. Berliner klin. Wochenschrift, 1897, No. 33.

of albumin in pathological transudations and exudations. His experience warrants the following statements :

1. Inflammatory processes, 4 to 6 per cent. of albumin.
2. Venous stasis, 1 to 3 per cent. of albumin.
3. Marked hydræmic conditions, as in amyloid degeneration or nephritis, 0.1 to 0.3 to 0.5 per cent.
4. Combination of two or three of the above causes, 0.2 to 6 per cent.

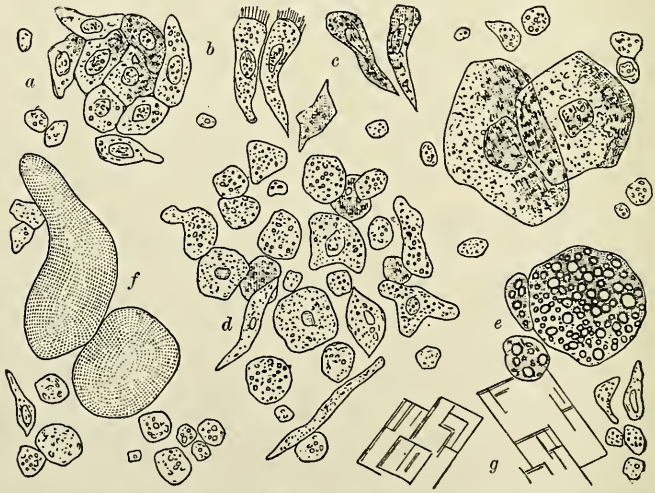
In group two, even without inflammatory complications, a high percentage may occur in old transudations.

Contents of Cysts.

In aspiration of the abdomen and of the pleura cysts are sometimes evacuated, the nature of which is often determined by an examination of the fluid. It is within the province of this work to discuss hydatid cysts, pancreatic cysts, and the cystic kidney. As tumors of the ovary so frequently resemble tumors in other situations, it is well also to discuss in this section the nature of the fluid withdrawn from them.

Hydatid Cysts. The fluid of hydatid cysts is clear, alkaline, and of a specific gravity of 1010. It contains chloride of sodium in excess, grape-sugar in small amount, and very little, if any, albumin.

Fig. 98.



Contents of an ovarian cyst. (Eye-piece III., obj. 8, A. Reichert.) *a*, squamous epithelial cells; *b*, ciliated epithelial cells; *c*, columnar epithelial cells; *d*, various forms of epithelial cells; *e*, fatty squamous epithelial cells; *f*, colloid bodies; *g*, cholesterin-crystals. (Von Jaksch.)

On microscopical examination hooklets are found, as in the sputum from hydatid cyst of the lung, as well as portions of membrane. The membrane is recognized by its peculiar transverse striation and the granular appearance of its inner surface. The heads or scolices are sometimes found. Two circles of hooklets and four disks on the ante-

rior aspect cross the head, which is separated from the hinder part by an annular constriction. (See Sputum and Fæces.) If suppuration has taken place the original nature of the cyst cannot be made out unless hooklets are found. After the fluid has been standing in a conical glass vessel the bodies may be found in the sediment.

Ovarian Cysts. The fluid from an ovarian cyst is of high specific gravity, 1026, of alkaline reaction, contains but a small amount of albumin, and does not coagulate. On microscopical examination various forms of epithelial cells are seen, colloid bodies, and cholesterin-crystals. If hemorrhage has taken place in the cyst the color of the fluid is correspondingly changed, and beside the squamous, columnar, and ciliated varieties, some epithelium in the stage of fatty degeneration and red and white blood-corpuscles are seen. In colloid cysts the usual concretions are found. (See Fig. 98.)

In *dermoid* cysts, in addition to the above, squamous epithelium, hairs, and fatty-, hæmatoidin-, and cholesterin-crystals are detected. *Ovarian fluid* contains albumin and methæmoglobin, or paralbumin. The latter is detected by mixing a portion of the fluid with three times its bulk of alcohol. It is then allowed to stand for twenty-four hours, when it is filtered. The precipitate is removed and suspended in water. After filtering the filtrate is seen to be opalescent, and is tested as follows :

1. On boiling no precipitate is formed, but the fluid becomes turbid.
2. There is no change with acetic acid alone.
3. The fluid becomes thick and of a yellowish tint when treated with acetic acid and ferrocyanide of potassium.
4. There is a change to a violet color when treated with concentrated sulphuric and acetic acids.

Some observers differ from the above statement in their description of the fluid of an ovarian cyst ; all agree as to the large number of cell-elements. At one time it was thought that the fluid contained a special cell, but this view has been abandoned. In rare cases the specific gravity may be lower than that of the fluid of ordinary ascites. A fluid of low specific gravity, with a small amount of albumin, is said to be characteristic of a cyst of the broad ligament.

Cystic Kidney. The fluid from a cystic kidney can be recognized by the properties it derives from the renal secretion. Urea and uric acid in large amounts point to its true source. Renal epithelium is of the greatest diagnostic value. (See Urine.) If epithelium from the urinary tubules can be detected after the fluid has settled the diagnosis is absolute. (See Hydronephrosis.) It must not be forgotten that both urea and uric acid may be found in other cysts, as in those of the ovary, if they communicate with the urinary tract.

Pancreatic Cysts. The fluid from cysts of the pancreas is of a specific gravity of 1012, but may be as high as 1028. It contains cholesterin-crystals in abundance, and blood or pigment. Serum-albumin is present, but metalbumin is not found. Three diastatic ferments are present :

(1) If on examination for sugar the latter is found to be a maltose, its presence is of diagnostic significance.

(2) The most pronounced property of the pancreatic fluid, and that by which we are enabled to distinguish it from other fluids, is the power of digesting albumin without the presence of an acid.

Boas (*Deutsche med. Wochenschr.*, 1890, Bd. xvi. p. 1095) developed the method of examination. The fluid is to be added to milk. After the casein is precipitated the biuret-test is applied, as follows: Heat the substance with caustic potash and add drop by drop a 10 per cent. solution of sulphate of copper. If digested albumin is present the fluid assumes a reddish-violet color. No other cystic fluid can dissolve albumin in alkaline solution.

It is not necessary that albumin or fibrin should be employed in performing this test, as it is sufficient to add milk to the secretion; when in such cases the casein of the milk is precipitated, and the biuret test is applied to the resulting filtrate, and the test compared with a control-milk from which the casein has been removed (this can be done by adding very dilute acetic acid with constant stirring), the digestive property of the liquid under examination may be with certainty determined. The peptone would not be precipitated with the albumin, and as all albumins give the same reaction as peptone with the biuret test, the albumin should be removed before applying the test. It is removed from the filtrate by a saturated solution of ammonium sulphate. Then test the resulting filtrate with the biuret test. Then compare with the control-test as above.

(3) The pancreatic fluid also emulsifies fats. In large cysts, however, particularly if of long standing, the physiological properties of the pancreatic juice are sometimes wanting.[1] In the case referred to by Boas and reported by Karewski, the old age of the cyst modified the character of the fluid, and hence rendered its nature doubtful. Moreover, in the exploratory puncture the stomach was penetrated. For two reasons the author advises against exploratory puncture. First, the age of the cyst is not known, hence an analysis would be misleading. Second, the danger of puncturing other organs is too great. Exploratory laparotomy is preferable.

[1] In a case operated on by Penrose the analysis of the fluid was as follows: Sp. gr. 1025; reaction slightly alkaline; serum-albumin; no metalbumin; diastatic ferment absent; maltose absent. By Boas' method, power to digest albumin appeared to be great; but when the albumin remaining in the filtrate was removed from the pancreatic fluid, it failed to show that peptone was formed. The method, therefore, appears to be fallacious in this class of cases. The cyst was old, and the fluid no doubt lost its physiological properties. Cholesterin was present in enormous amount; tyrosin-crystals were very scarce.

CHAPTER XXII.

THE BLOOD.

The blood is a tissue, the origin, growth, and decay of the elements of which has been the source of the greatest interest. It was the tissue held responsible in days gone by for many diseases, the origin of which was not known, so that skin eruptions, scrofula, and other affections were known as blood diseases. At present we hold such affections only blood diseases which show a demonstrable change in the physical or morphological characteristics of the blood. There is either diminution of the red cells, increase or diminution of the white cells, or diminution of the hæmoglobin. Strictly speaking, most of the blood diseases now so called are really diseases of the blood-making organs—the lymphatic glands or the spleen. It is interesting to note that as late as 1866, J. Hughes Bennett included under diseases of the blood leucocythæmia, chlorosis and anæmia, diabetes, the infectious diseases, rheumatism, gout, and scurvy. The most recent text-book divides the blood diseases into *anæmia*, with two subdivisions, and *leukæmia*. Of course, no one thinks of considering the infectious diseases blood diseases any more than we think of considering typhoid fever an ulceration of the intestine.

Although the blood diseases are thus limited, it is none the less true that the blood may be the only tissue by an examination of which we can determine the ailment from which the patient suffers. As has been previously related, many infections are recognized in this manner only.

The symptoms of blood affections are due to the physical change in the blood and the effect of this altered blood upon the function or the nutrition of the organs. Many functional symptoms thus arising may be the first indications of blood disease, as dyspnœa or palpitation, both very common symptoms. The symptoms may be subjective or objective, or both. The recognition of the former comes from the history of the disease and the complaints of the patient. The latter, or the objective symptoms, are determined by the physical examination of the patient and the examination of the blood.

We recognize scarcely any condition at the present day due to an increase of the bulk of the blood or of the red cells. *Plethora* is hardly a clinical identity. The symptoms of blood diseases, therefore, are the symptoms of *anæmia*. In like manner, all the data obtained by inquiry are those which belong to some form of anæmia.

THE DATA OBTAINED BY INQUIRY.

The Social History. Generally speaking, women, patients of early age, who have been subjected to want or had unusual care, or faulty nutrition, are those most liable to anæmia. No family predis-

position exists to a marked degree apparently, although it is well known that "pale people" are a family class. The previous history and the data to be elicited in investigating it are best appreciated by turning to the classification of the cause of anæmia in succeeding pages.

The HISTORY of the disease is usually that of gradual onset, although sudden fright or any cause producing profound shock is said to cause acute anæmia. But the reader must again be referred to the paragraphs just mentioned.

The SUBJECTIVE SYMPTOMS are general. Languor, debility, and fatigue are complained of. The patient with anæmia may have one group of symptoms preponderate. Thus headache, vertigo, restlessness, noises in the head, and neuralgias may be the most prominent symptoms. Again, dyspnœa and air-hunger may be the most distressing, or cardiac palpitation may be the earliest symptom, with or without cardialgia. Then gastro-intestinal symptoms are suggestive, although not pathognomonic. The peculiar appetite of chlorosis is well known. The causeless vomiting of many forms of anæmia has often been described. The bowels may be constipated or loose, varying more particularly because of the difference in the cause of the anæmia. Ringing in the ears has been referred to, and flashes of light, spots before the eyes, and other visual phenomena may be complained of, and show their origin in the state of the blood. Other alteration of the special senses are not marked in the course of any of the anæmias. These symptoms may occur singly or are combined in varying degrees.

THE DATA OBTAINED BY OBSERVATION.

While diseases of the blood, and especially forms of anæmia, are recognized by an examination of the blood, much information can be secured by general physical examination. It is true no disease would be pronounced a blood affection unless that tissue is examined by the modern means of research.

An examination of a case of anæmia includes a study of the appearance of the patient, the color or hue of the surface, and the occurrence of œdema. Both these subjects are carefully considered in the chapters devoted to them respectively. Examination of the eye-grounds should always be made, when the findings discussed in the Chapter on the Eye may be present, if the case is one advanced in its course. No consideration of anæmia can be made, however, without an examination of the organs thought to be engaged in the blood formation, hence the state of the glands and the size of the spleen are inquired into.

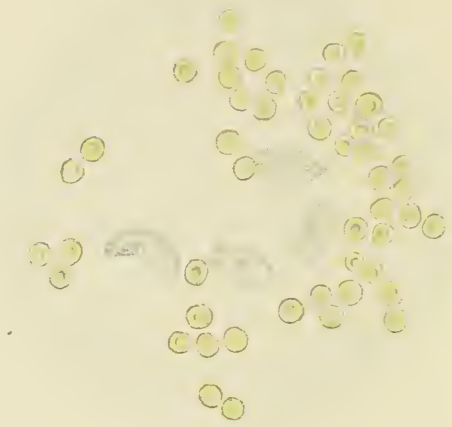
Finally, as evidence of the presence of anæmia, we observe frequently cardio-vascular phenomena. The murmurs that are heard in the heart and bloodvessels in this disease are fully discussed in the Chapter on Diseases of the Heart, to which the reader is referred.

Examination of the Blood.

NORMAL BLOOD. Before a consideration of the examination of the blood, it may be well to review the elements of which the blood is composed.

PLATE IX.

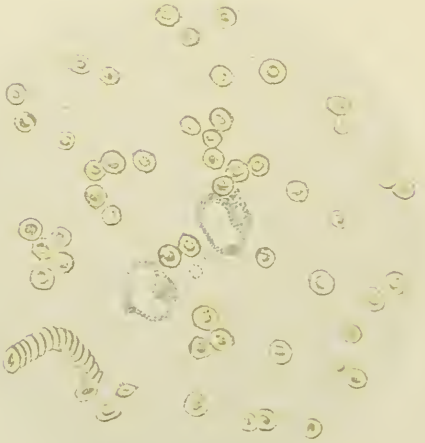
Fig. 1



Blood from Case of Pneumonia, showing Leucocytes

(Oc. 4 of Zeiss Immersion.) Drawn by J. D. Z. Chase

Fig. 2



Normal Blood, showing Rouleaux and Leucocytes.

(Oc. 4 of Zeiss Immersion.) Drawn by J. D. Z. Chase

The blood consists of corpuscles and serum. The corpuscles are four: (1) Red blood-cells or erythrocytes; (2) nucleated red blood-cells; (3) blood-plaques; (4) leucocytes.

The ordinary red blood-cells measure $\frac{1}{3200}$ inch; the leucocytes, $\frac{1}{2500}$ inch. In an adult man the red cells number from 5,000,000 to 5,500,000 to the cubic millimetre; in an adult woman the number is usually less, being from 4,500,000 to 5,000,000. There are 8000 to 10,000 leucocytes in a cubic millimetre of blood, or 1 to 350–600 red blood-cells.

Varieties of Leucocytes. In the normal blood there are found the following varieties of leucocytes: 1. *Small mononuclear* forms, which are cells about the size of a red blood-corpuscle, and have a round, large, deeply staining nucleus, surrounded by a narrow rim of non-granular protoplasm. These are known as *lymphocytes.* 2. *Large mononuclear* leucocytes several times as large as the foregoing. They have a round or oval nucleus, with a relatively larger amount of non-granulated protoplasm. 3. *Transitional* forms, which resemble the last named, except that the nuclei are indented or S-shaped. 4. *Polynuclear* leucocytes. These are usually about the size of the foregoing variety, but they may be somewhat smaller. The nuclei are long and irregular and stain deeply. The protoplasm contains granules that stain by a combination of both basic and acid dyes, but by neither alone. The cells are therefore called "*neutrophiles.*" 5. Leucocytes similar to the last form, except that their protoplasm contains highly refractive granules that are stained by acid dyes alone. For this reason they are usually called "*eosinophiles.*" The proportion of each variety in the normal blood is fairly constant; lymphocytes, 15 to 25 per cent.; polynuclear, 65 to 80 per cent.; mononuclear and transitional forms, 6 per cent.; and eosinophiles, 2 per cent. or less. (See Plate IX.)

Physical Appearance. For the purpose of examination of the blood a drop or two is quite sufficient. In olden times much stress was laid upon the physical character of the blood drawn in bulk. The significance of the "buffy coat" was dwelt upon by all clinicians, not alone because of its value from a therapeutic stand-point, but also because it was held to indicate the type of the disease that was present. At present, however, we rely very little upon the results of the naked-eye examination. By this examination we may be able to distinguish bright-red arterial blood from darker venous blood, and also when arterial blood has become deficient in oxygen from any of the causes of venous engorgement and cyanosis. In chlorosis and hydræmias the blood is pale, as though mixed with water, while in severe leukæmias it has a slight milky tinge. On the other hand, in carbonic-oxide poisoning the blood becomes of a brighter red, while in poisoning with chlorate of potash and aniline, and in grave cases of poisoning with nitrobenzol and hydrocyanic acid, it is brownish-red or chocolate-colored.

For accuracy in diagnosis reliance must be placed upon instruments of precision. These are the microscope, the hæmoglobinometer, the hæmocytometer. By this examination we determine (1) the size and

shape of the red cells; (2) the morphological characteristics of the white cells; (3) the number of the red cells; (4) the number of the white cells; (5) the presence of new elements as nucleated red cells and myelocytes; (6) the presence of parasites; (7) and the amount of hæmoglobin.

Method. A drop of blood for this examination may be taken from the lobe of the ear or the finger-tip. The surface should be thoroughly cleansed with alcohol, and dried carefully. If the finger is used, it should not be unduly constricted. The puncture should be made forcibly and quickly, in order that the drop of blood may ooze freely. If it is difficult to secure the blood, it is well to allow the first or second drop to escape before any is collected. When the flow is started and the finger cleansed the succeeding drops are gathered on cover-slips. If the lobe of the ear is selected, it should be steadied with the fingers of the left hand, which at the same time stretches the skin. It may be necessary to puncture to the depth of one-eighth of an inch, or even more if the skin is bloodless. The puncture should be made on the lower surface or edge of the lobe. A surgical needle, a small lancet, or the bayonet-pointed instrument devised for the purpose, should be used. The nib of a new steel pen, one-half of which has been broken off, answers fully as well.

It is well to remember the precaution insisted upon by all who examine the blood frequently, to beware of "bleeders." It sometimes becomes a very serious matter when hemorrhage is started in a patient who is the subject of hæmophilia.

Mode of Examination. As soon as the blood flows freely, without pressure, the apex of a drop may be touched by the cover-glass, which has been previously prepared. The cover-glass should not touch the skin, and as soon as it is covered by the blood it should be placed face downward upon the slide, or if cover-slip preparations are to be made, upon a corresponding cover-glass. The precaution must be taken to have the slide and cover thoroughly cleansed. It is well to keep them in alcohol or in a weak acid solution after they have been previously cleansed with soap and water, and when removed from the alcohol solution they should be thoroughly polished with a clean handkerchief. The blood will then spread evenly over the surface with the slightest pressure upon the cover-glass. If the slide and cover are warmed slightly before using, it will not be necessary to use the pressure just referred to.

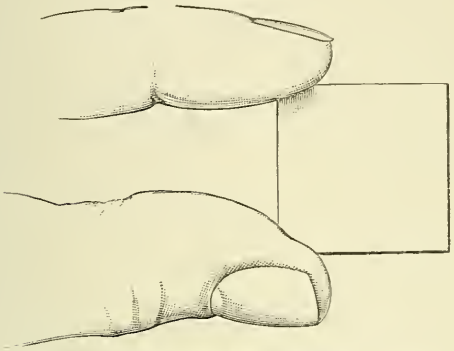
Blood collected in this way may be examined fresh or be put aside for staining and future examination.

Examination of Fresh Blood. By the examination of fresh blood we learn of the presence of parasites and of the occurrence of rouleaux formation. In a general way we can learn the number of red and white cells respectively, the degree of coloring of the red cells, the shape and size of the red cells, and the presence of blood-plates. An unusual increase in leucocytes may be detected, and the diagnosis of leukæmia made without further investigation.

Cover-slip preparations. For the purpose of future study, and particularly in order to determine the differential count of the white

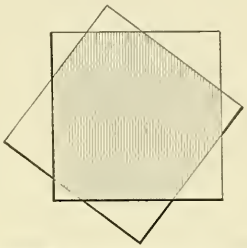
corpuscles, cover-slip preparations are made. The covers are cleansed and the blood secured in the manner previously described. The cover-glass, which has been touched to the summit of the drop, is let fall upon another somewhat diagonally. (See Fig. 100.) The drop

FIG. 99.



Proper method of holding a cover-glass. (CABOT.)

FIG. 100.



Illustrating the position of cover-glass during the spreading of blood films (CABOT.)

spreads over the adjoining surfaces of the cover-glass. As soon as the spreading ceases, slide the glasses off, but do not lift them apart. Dr. Manson introduced the use of tissue paper drawn over a slide, with the object of getting a more uniform thickness of film. Pakes uses this method applied to cover-glasses, which should be not less than $1\frac{1}{2}$ inch by $\frac{3}{4}$ inch. The cover-glasses are held in a clip and smeared by means of cigarette paper cut into strips across the direction of the rib. The cover-slip should be dried in a gas or alcohol flame at once, by means of which the preparation is fixed.

"Fixation" may also be done by alcohol and ether, or by corrosive sublimate solution. The cover-glass should be immersed for one-half hour in equal parts of alcohol and ether. After such fixation malarial organisms and nucleated red corpuscles are more readily found.

Fixation with formol is quickly secured. Dilute one part of formol with nine times its volume of water; dilute one part of this mixture with nine times its value of alcohol. The resulting fluid will fix immersed specimens in one minute.

Fixation of heat is best when the white cells are to be studied. By this method it is best to put the cover-slips in a dry-heat sterilizer at a temperature of 110° or 115°. If this cannot be done, place the cover-slips on the end of a copper plate at least a foot long, the other end of which is heated by a Bunsen burner or a gas flame. The cover-slips should be placed on the plate at that point on which water boils when dropped upon the surface of the copper. They should be placed face downward and kept there from fifteen to twenty minutes. When they cool they are ready for staining.

STAINING. The greatest care should be taken to have a perfectly clean, dry cover-glass, which should be handled with forceps, to avoid moisture and soiling. (1) The prepared cover-glass, arranged as above, should then be immersed for a few minutes in a solution of eosin:

Eosin	0.5
Alcohol (70 per cent.)	100.0

This solution should be diluted one-half before using. (2) The cover-glass should then be dried and stained for three or four minutes in a saturated aqueous solution of methylene blue, also diluted one-half before using (Chunzinsky-Plehn's mixture). Or, instead of the latter, stain for half an hour to several hours in Delafield's hæmatoxylin. This hæmatoxylin-stain is made in the following manner: To 400 c.c. of a saturated solution of ammonia alum add 4 grammes of hæmatoxylin-crystals dissolved in 25 c.c. of strong alcohol. Leave this exposed to the light and air in an unstoppered bottle for three or four days. Filter and add 100 c.c. of glycerin and 100 c.c. of methylic alcohol. Allow the solution to stand until the color is sufficiently dark. Then filter and keep in a tightly stoppered bottle. The stain should ripen for at least two months before using. For blood-work the solution is used in its full strength. By this double stain, a modification of *Ehrlich's hæmatoxylin-eosin mixture,* the red corpuscles are stained red, the nuclei blue, the bodies of the leucocytes light lilac and their nuclei darker, the eosinophile granules a brilliant red.

Ehrlich's Tri-acid Stain. The Ehrlich tri-staining mixture is the best that can be selected for staining. Thayer says the following is a satisfactory modification of Ehrlich's formula:

Saturated aqueous solution of acid fuchsin	2
Water	3
Saturated aqueous solution of orange-G.	6.25
Saturated aqueous solution of methyl-green	6

To be added, drop by drop, while shaking the solution:

Water	15
Alcohol	10
Glycerin	5

The stain is spread over the cover-glass specimen with a glass rod, and in from one to five minutes washed off with water. If the cover glass has not been heated very long it will not be necessary to keep the stain long in contact with the blood, although specimens which are heated an hour require at least five minutes for the stain to take. After the specimen is stained and washed in water it should be dried between layers of filter paper and mounted in balsam. It can then be examined at leisure with the twelfth oil-immersion with diaphragm open.

Specimens heated for one or two hours stain better than those which have been treated only a short time. The red cells appear orange or buff, the nuclei of the colorless corpuscles green or greenish-blue, the neutrophilic granules a violet or lilac color, the eosinophilic granules a deep red. The nuclei of nucleated red corpuscles, when present, are stained an intense deep green, almost black.[1]

Another method much used and urged by Hewes is as follows: The blood, after fixation, is subjected for four minutes to the modified Ehrlich stain, which is made as follows:

[1] Thayer, loc. cit.

Ehrlich-Biondi-Heidenhain three-color mixture	1.7 grammes.
Acid fuchsin	0.05 "
Absolute alcohol	2 c.c.
Distilled water	18 c.c.

After immersion wash the specimen in water and then subject it from one-half to ten seconds to Löffler's solution of methylene-blue. Again wash the specimen, dry, and mount in balsam.

Löffler's solution is saturated alcoholic solution of methylene-blue, 30 c.c. ; potassic hydrate (1 : 10,000 solution), 100 c.c.

The Red Corpuscles or Erythrocytes. In thickly spread blood the cells are arranged in the form of rouleaux. If such rouleaux are absent in a preparation thus poorly spread it is an indication of great reduction in the red cells.

In thinly spread films the red cells are recognized by their color and shape. They vary from 6 to 9μ in diameter. The lighter colored centre, due to the biconcavity of the corpuscle, sometimes causes confusion. It must be remembered, too, that the corpuscles readily become crenated, an appearance which may be confounded with pigmentation or other abnormal change. In them, too, a slight molecular movement is sometimes seen, which must not be confounded with the amœboid movements in dying cells or with the rapid motion of malarial pigment.

Poikilocytosis. The variations in size and shape are indications of disease. In forms of anæmia the red cells may be larger than normal ; they may be irregular in shape, or they may be smaller than normal. Large cells are known as *macrocytes*, small cells as *microcytes*. Cells that are irregular in shape are known as *poikilocytes*. They may be oval, pointed, angular, or reniform.

Achromia. When the red cells are stained the hæmoglobin takes the orange-G. of the tri-colored mixture of Thayer, causing the red cells to be brilliant yellow or pale orange in tint. An idea of the amount of hæmoglobin can thus be obtained. When the hæmoglobin is diminished the centre is pallid, although in extreme poverty of hæmoglobin the colored rim may be a faint outline only (achromic forms).

Nucleated Red Corpuscles or Blasts. They contain one or more nuclei. The stroma takes the golden acid stain and the nucleus the pure basic stain. They are divided in accordance with their size, and the depth of the color of the nuclei, into three varieties :

(1) The *normoblast*. It is the size of a normal red blood-corpuscle. The stroma is golden in color ; the one or more nuclei are deeply bluish-black, homogeneous. The nucleus occupies one-fourth to three-fourths of the whole corpuscle. It is deeper in color than the nuclei of the white blood-corpuscle. It is the parent cell of the red blood-corpuscle.

(2) The *megaloblast*. They are larger than a red blood-corpuscle. The color of the stroma is less intense than that of the normoblast, and the nucleus is blue rather than black, and not compact and homogeneous. The nucleus is more compact and more clearly defined than the nucleus of a white blood-corpuscle. It is found on the marrow of the embryo.

(3) The *microblast*. They are smaller than the normal. There is but little stroma, and the nucleus is deep black

Blasts are found in anæmia. An excess of normoblasts indicates very active regeneration of blood.

Polychromatophiles. These are red blood-corpuscles in which the stroma takes not only the normal acid staining elements but also the blue basic or purple neutral stain. They are degenerate forms of red blood-corpuscles.

Degenerate Forms. The coloring matter is irregularly distributed and the stroma appears disintegrated.

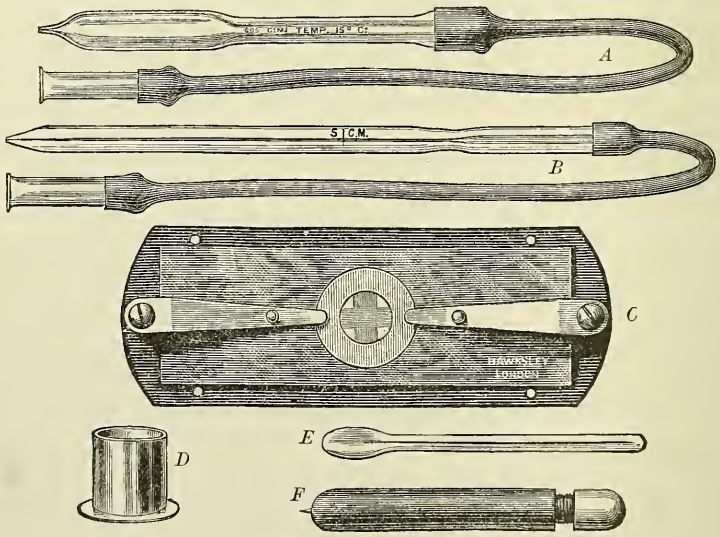
When thus stained we can readily find nucleated red cells, but the fibrin or blood-plates, as a rule, are destroyed.

Counting the Corpuscles. It is of the greatest clinical importance to be able to estimate the number of red cells in a given quantity of blood, in order that approximately at least we may know of its globular richness. For this purpose hæmocytometers are used.

The hæmocytometers, or blood-counters, most frequently used in this country are those of Gowers and Thoma-Zeiss.

Gowers' instrument (Fig. 101) consists (1) of a small pipette, A, which, when filled, holds exactly 995 cubic millimetres ; it is for meas-

Fig. 101.



Hæmocytometer of Gowers.

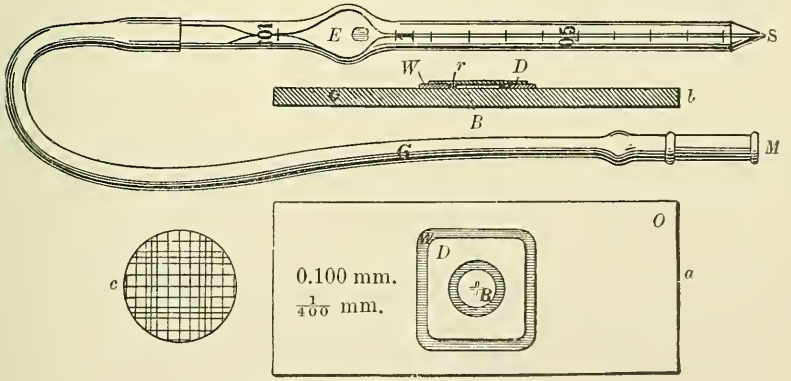
uring the diluting fluid ; (2) a capillary tube, B, graduated for 5 cubic millimetres ; (3) a small glass jar, D, in which the dilution is made ; (4) a small glass stirrer, E, for mixing the blood and diluting fluid in the jar ; (5) a small lancet, F ; (6) a brass stage-plate, C, carrying a glass slip on which is a cell one-fifth of a millimetre deep. The bottom of the cell is divided into one-tenth millimetre squares. On the top of the cell rests the cover-glass, which is kept in place by the pressure of

two springs proceeding from the ends of the stage-plate. 995 cubic millimetres of the diluting fluid are measured and blown into the mixing-jar ; then 5 cubic millimetres of blood are added and the two thoroughly mixed. A small drop of the mixture is then placed upon the cell, the cover-glass gently adjusted and held in place by the two springs. From five to ten minutes should be allowed to elapse, so that the corpuscles will have time to settle to the bottom of the cell. The stage-plate is then placed under a microscope, and the number of red blood-cells in ten squares counted. This number multiplied by 10,000 gives the number in a cubic centimetre of pure blood. It is better to count a large number of squares, take the average, and multiply by 100,000. This number is the product of the dilution (200) by the square surface of the cells, 100 (10×10), and again by 5, the depth of the cell : $200 \times 100 \times 5 = 100{,}000$. To facilitate seeing the fine lines marking the squares, a soft black lead-pencil should be gently rubbed over them before the drop of diluted blood is placed on the cell. Counting of the white cells is made much easier if the diluting fluid is colored a pale violet with a very small quantity of gentian-violet. The white cells then appear a distinct blue, while the red cells are unaltered. As diluting fluids, a 1 per cent. solution of common salt, or a $2\frac{1}{2}$ per cent. solution of bichromate of potash, as recommended by Daland, may be employed ; or Toison's fluid can be used.

Toison's Fluid. It is made up as follows : Distilled water, 160 c.c. ; glycerin, 30 c.c. ; sulphate of soda, 8 c.c. ; chloride of soda, 1 gramme ; methyl-violet, .025 gramme.

Another hæmocytometer is the Thoma-Zeiss (Fig. 102). It is preferred by most clinicians. It consists of a heavy glass slip (*a*), in the

FIG. 102.



Thoma-Zeiss blood-counting apparatus.

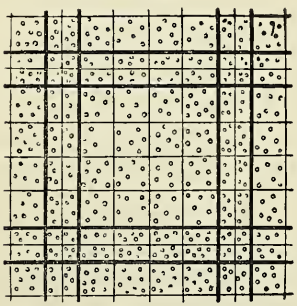
middle of which is a cell (*B*) exactly $\frac{1}{10}$ millimetre in depth. The cell is limited at the periphery by a circular gutter to prevent fluid placed upon the cell from flowing beyond it between the slip and cover-glass. The floor of the cell is ruled into squares whose sides are $\frac{1}{20}$ mm. Double lines mark out large squares, each containing sixteen small

squares. Thick, carefully ground cover-glasses (*D*) are provided in the case. The ordinary Potain *melangeur* (*S*) is used to measure and mix the blood. It consists of a capillary tube, the upper portion of which is blown into a chamber (*E*) holding 100 c.mm. The stem of the tube is graduated at 0.5 and at 1 c.mm.

To use the instrument, a drop of blood is obtained from the finger or lobe of the ear, the point of the capillary tube is inserted into the drop, and blood sucked up to the mark 1 c.mm. The point of the tube is then quickly wiped free from excess of blood and inserted into the diluting fluid, which is drawn up to the level of the mark 101. The proportion of blood and diluting fluid is then 1 to 100 c.mm. The blood and diluting fluid are now thoroughly mixed. The diluting fluid in the stem of the *melangeur* is now blown out and a drop of the blood-mixture placed on the cell. The cover-glass is adjusted carefully to avoid bubbles and to prevent the escape of the fluid between it and the slip. The cover-glass is now pressed firmly down until Newton's color-rings appear, and then the slip is allowed to stand for five or ten minutes, until the corpuscles have settled to the bottom of the cell.

The cell is ruled into 400 small squares, groups of sixteen squares being separated by double lines. The surface of a square is $\frac{1}{400}$ square millimetre, and the depth of the cell being $\frac{1}{10}$ millimetre, the space overlying each square is $\frac{1}{4000}$ of a cubic millimetre. In estimating the number of corpuscles in a cubic millimetre of blood, multiply the number of corpuscles counted in all the squares by 4000 and the product by the dilution, which is 1 to 100 or 1 to 200, according as 1 or 0.5 c.mm. of blood has been used. The last product is now to be divided by the number of squares which have been included in the count, the quotient being the number of corpuscles in a cubic millimetre of blood. The results are accurate in proportion to the care exercised in the measurement of the blood and diluting fluid, and especially in proportion to the number of squares counted.

FIG. 103.



Appearance of blood in the Thoma-Zeiss cells.

In the estimation of *white blood-cells* the pipette made by Zeiss is employed. In this instrument the blood is diluted ten times by a solution of one part of a $\frac{1}{3}$ per cent. acetic acid solution to ten parts of distilled water. By means of this solution red cells are dissolved and the nuclei of the white cells are rendered distinct and easy of recognition. Toison's fluid, mentioned above, may also be used. The ordinary Thoma-Zeiss slide is employed, and the average number of white cells in each small square is multiplied by 40,000. To obtain accurate results four entire fields should be counted.

The *hæmatokrit* is an instrument devised for the estimation of the percentage-volume of red corpuscles by means of centrifugal force. In Daland's article will be found a full description of the instrument, and from the same article the following method of using it is abstracted :

" The finger or ear and apparatus are prepared as above. An incision is made deep enough to produce a good-sized drop of blood. This is drawn into a hæmatokrit tube by means of suction through an attached rubber tube, one finger being placed over the free end when the rubber tube is removed, to prevent the loss of blood. The filled tube is then placed in the frame of the hæmatokrit and a second prepared exactly as the first. The larger wheel is then rapidly rotated for two minutes at seventy-seven turns of the handle-crank per minute (giving altogether 20,000 rotations of the frame), and the result read from the scale multiplied by 2 gives the percentage-volume. It has been found by experimenting that each division upon the scale of the hæmatokrit tube represents 100,000 corpuscles." This procedure is not available for the determination of the volume of leucocytes unless the number exceeds 20,000, at and above which number an approximate estimate may be readily determined. A distinct white band appearing between the red cells and the clear fluid, having the width of one line, may be considered as representing from 15,000 to 20,000 leucocytes.

Number. The normal number of red cells—as stated previously—is approximately 5,000,000 per cubic millimetre. They may be reduced to 500,000. A reduction below 3,000,000 indicates grave anæmia. When the reduction is less than 1,500,000 the anæmia is said to be pernicious or malignant. It must be remembered that temporarily the red cells are reduced during menstruation and lactation. At puberty there is also a reduction. On the other hand, when the blood is concentrated by profuse sweating or exhaustive diarrhœa, the number of red cells is increased, while they are lowered when the blood is diluted by large draughts of fluid or by subcutaneous injections of fluid. A cold bath may temporarily concentrate the peripheral blood, and thereby increase the number of cells. Red cells are always lessened in the aged, and are reduced in number after great exertion. They are increased in number after fasting, and diminished after a meal, particularly if much fluid is taken.

Oligocythæmia. Oligocythæmia is the name applied to a diminution in the number of red blood-cells, from whatever cause. It is usually associated with *oligochromæmia* (deficiency of hæmoglobin), which, however, in idiopathic anæmia is absolute, not relative. Marked oligocythæmia can be detected with the microscope alone, and can be estimated accurately with the hæmocytometer or hæmatokrit. (See Fig. 102.)

The White Corpuscles. The white or colorless corpuscles are recognized by their absence of color, by their irregular shape and their size, which is larger than that of the red, and by the amœboid movements which they undergo, particularly if placed on a warm stage. They number from 8000 to 10,000 per cubic millimetre. They are readily recognized by the peculiar affinity which they have for various aniline dyes. They appear as granular nucleated cells in stained specimens. The method of staining has been described, and the varieties of leucocytes found in normal blood indicated on page 371. In addition to determining the number by counting, as described in the paragraph which gives the method of counting the red cells, a so-called

differential count is made. This count enables us to determine the proportion of the many varieties of leucocytes.

In counting the white blood-corpuscles, Phear advises the use of the camera lucida. The most convenient form is the Zeiss-Abbe drawing camera, used with the stage of the microscope in a horizontal position. The image of the field is projected on a piece of paper or cardboard lying horizontally on the table immediately to the right of the microscope stand. The ruled squares on the floor of the hæmocytometer cell are accurately marked out on the cardboard. The image of the corpuscles which lie on the unruled part of the cell floor is thrown by means of the camera on the cardboard, and the corpuscles which appear to lie over each square are enumerated and included in the count. It is convenient to use a mechanical stage. It is essential that the eye-piece, objective, and tube-length used during the count should be the same as on the occasion of marking out the squares on the cardboard. For the dilution of the blood, that recommended by Sherrington,[1] consisting of distilled water, 300 cubic centimetres; sodium chloride, 1.2 grammes; neutral potassium oxalate, 1.2 grammes, and methylene-blue, 0.1 gramme, is excellent. The blood-corpuscles are not stained, but their shape and color are preserved. The nuclei of the white corpuscles are in every instance stained, facilitating the distinction of the white from the red corpuscles. For the differential count of the white corpuscles it is desirable to work with an immersion lens.

Differential Counting. After the specimen is carefully stained with the triple solution it is ready for differential counting of the white cells, as well as determining the presence of nucleated red cells. To make the differential count a large number of leucocytes should be studied. The best plan to pursue is to begin at the upper left-hand corner of the blood film and count across the film to the right-hand corner. Then move the slide so that an adjacent field comes into view, when the process is to be repeated. In this manner the entire field is covered. In ordinary leucocytosis a thousand leucocytes can be seen in a seven-eighth inch cover-glass specimen. We may find an abnormal variety of leucocytes; an abnormal proportion of some one of the normal leucocytes; an abnormal number of all the leucocytes.

Fluid Preparations. Dr. A. G. Phear lays stress on the advantages of fluid preparations over the cover-slip method. In the cover-slip method leucocytes are inevitably flattened and distorted in the process of making and fixing the film; some are washed away during the staining; others obscured by the red corpuscles. In the fluid preparation the white cells are fixed and preserved as approximately spherical bodies; camera lucida drawings and measurements of them could be relied on as accurate. A solution of methylene-blue (0.2 per cent.) in 40 per cent. alcohol is used for diluting the blood. The red corpuscles are laked so that the white cells alone remain conspicuous. "A small quantity of the diluting solution is added to a drop of blood on a glass slide and the two are thoroughly mixed by directing a cur-

[1] Proceedings of the Royal Society, vol. lv.

rent of air through a pipette on to the surface of the fluid. The fluid is allowed to spread as a thin film under a cover-glass and the edges then sealed with vaseline." The contour of the normal polymorphonuclear cells is rounded. Their diameter vary from 9 to 10μ. The complex nucleus can be made out by changing the focus, the nucleus being, in fact, "an undivided elongated body, in places deeply constricted, elsewhere bulged into rounded lobes." The lymphocytes and the large hyaline cells represent the extremes of cells, differing in the amount of protoplasm around the nucleus; all grades are readily found. The nuclear diameter is fairly constant in these cells, varying only between 4.5 and 5.5μ. Large oval cells, as much as 14μ in length, with the nucleus large and irregular, usually reniform, are seen. The protoplasm becomes rapidly and uniformly stained an opaque blue color with methylene-blue. The coarsely granular or eosinophile cells (diameter from 9.5 to 10.5μ) are at once recognized in the film prepared with methylene-blue solution, notwithstanding the absence of an acid dye; the large refractile granules are tinged with a greenish color. The cells containing basophile granules (diameter about 8μ) have a characteristic appearance. The protoplasm contains granules of medium size, many of which are aggregated in one or more deeply stained clumps near the surface of the cell. The non-granular part of the protoplasm is stained a peculiar mauve or purple color. The nucleus is usually massed at the centre of the cell, and stains a slate or grayish-blue color.

Separate counts over different areas of one preparation gave uniform results, showing that the blood was evenly mingled with the diluting fluid. Not less than 500 cells should be enumerated at a time; the more the better. It was desirable to use a mechanical stage and to work with an immersion lens. The blood should always be procured, if possible, before the first meal of the day is taken, since this is the time at which the influence of meals is least likely to be evident.

Leucocytosis. Leucocytosis is a temporary increase in the number of white blood-cells of the same morphological varieties as in health, with an excess of the polynuclear forms (neutrophile leucocytosis). Such increase may be physiological or pathological, as indicated in the following:

Physiological Leucocytosis. (1) Pregnancy (14,000 and upward); (2) during digestion (from 1000 to 7000 above normal; more in children); (3) new-born (12,000).

Pathological Leucocytosis. An excess of *leucocytes* occurs in the following diseases: (1) Leukæmia; (2) pernicious anæmia; (3) chlorosis; (4) diseases of lymphatic glands; (5) disease accompanied by exudations, as *pleurisy*, *pericarditis*, *meningitis*, *polyarthritis*, and especially *croupous pneumonia;* (6) inflammatory condition associated with exudation, as appendicitis, pyonephrosis, perinephritic abscess, tonsillar and retropharyngeal abscess, acute pancreatitis, cholangitis; (7) many acute infectious diseases, as *varicella*, *variola*, *vaccinia*, *epidemic cerebro-spinal meningitis;* cholera, typhus fever, trichinosis, glanders, diphtheria, scarlet fever, erysipelas, pyæmia and septicæmia, rheumatism, abscesses, and gangrenous inflammation; (8) after hemor-

rhage and (9) just before death, leucocytosis of agony. On the other hand, *leucocytosis* is not found in uncomplicated cases of (1) *influenza* (*Boston Medical and Surgical Journal*, March 22, 1894); (2) uncomplicated cases of *typhoid* fever; (3) *tuberculosis* when not associated with cavity-formation or hyperplasia of lymphatic glands (Stein and Erbman, *Deutsch. Archiv. f. klin. Med.*, Bd. 56); (4) many forms of carcinoma and sarcoma, gastric ulcer and benign pyloric stenosis (Schreuger, *Zeitschr. f. klin. Med.*, 1895, 27, 475), although it may be present in gastric carcinoma.

Leucopenia. Diminution of the number of leucocytes is seen (1) in starvation, as in cancer of the œsophagus; (2) the latter weeks of typhoid fever; (3) leukæmia complicated by infection.

Diagnostic Value. The value in diagnosis of determining the presence of leucocytosis is great. Its absence excludes the first series of cases; its presence the last. If leucocytosis is present in the course of, or convalescence from, typhoid fever, it points to a complication, as thrombosis. A post-febrile rise, due to a complication, may be distinguished from a true relapse by an increase of the white cells.

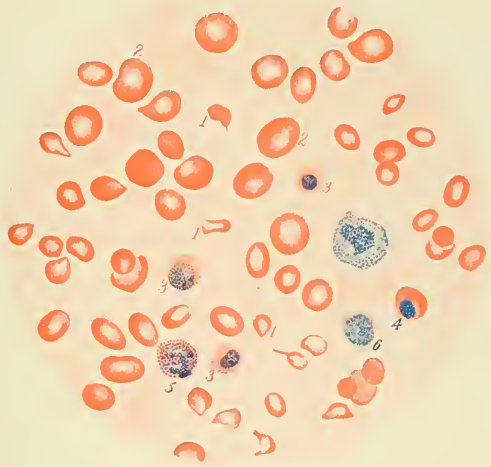
It is best determined with a hæmocytometer. Dry preparations, according to Ehrlich's method, are necessary for a study of the various forms of leucocytes. (See under Leucocythæmia, page 396, and Plate X.)

Increase of Special Leucocytes. Lymphocytosis. A relative increase in the lymphocytes, with or without a total increase of leucocytes, is seen in infants, and is a common accompaniment to rickets and hereditary syphilis. In some forms of scurvy it is also found. In adults lymphocytosis occurs in chlorosis and pernicious anæmia and in secondary anæmia of syphilis and typhoid fever. It occurs in hæmophilia, in adenitis, and splenic tumors. Cabot states that it is also found at the end of scarlet fever and measles, in pneumonia with delayed resolution, and in some forms of phthisis. The larger forms of leucocytes are seen. Absolute lymphocytosis occurs in lymphatic leukæmia.

Eosinophilia. An increase in the percentage of eosinophiles, with or without leucocytosis, is seen in many affections of the bones, in affections of the skin, and in diseases of the genital apparatus in females. It is also seen in certain disturbances of the sympathetic nervous system, as in cyanosis and vasomotor troubles associated with menstruation and pregnancy. The bone diseases in which the eosinophiles are increased are osteomalacia, sarcoma, carcinoma, and in those affections of the bone and marrow with which pernicious anæmia and splenic myelogenous leukæmia are seen. The skin diseases are urticaria, pellagra, herpetiform, dermatitis, and pemphigus, in herpes, eczema and prurigo, psoriasis, lupus, and myxœdema. In the eruption of scarlet fever and syphilis they are increased, but not in measles or smallpox. In various affections of the uterus and ovary, in functional disorders connected with the same, the eosinophiles are increased. They are also increased in gonorrhœa and prostatitis. They are increased in those infections in which Neusser's granules are found. Thayer, in Osler's clinic, has found marked increase in the eosinophiles in trichi-

PLATE X.

FIG. 1.

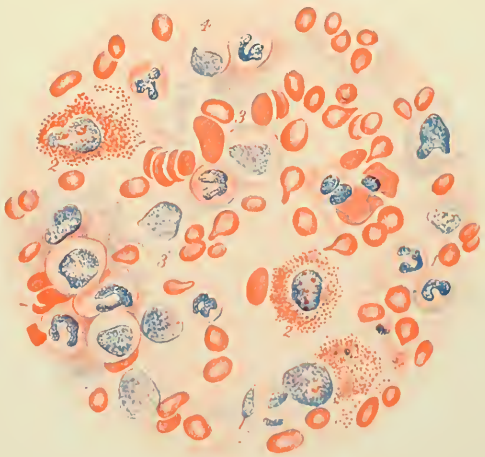


Blood from Case of Secondary Anæmia.

1. Poikilocytes.
2. Macrocytes.
3 and 6. Lymphocytes.
4. Nucleated red blood-corpuscle.
5. Polynuclear leucocytes.

(Oc. 4, ob. $\frac{1}{12}$ immersion.) Drawn by J. D. Z. Chase.

FIG. 2.



Leukæmic Blood.

1. Polynuclear leucocytes.
2. Eosinophile cell (mononuclear).
3. Large mononuclear leucocyte.
4. Small lymphocyte.

(Oc. 4, ob. $\frac{1}{12}$ immersion.) Drawn by J. D. Z. Chase.

nosis—in fact, recognizing this condition by the differential count. Diminution in the eosinophiles takes place during digestion, and in most of the infectious disorders accompanied by leucocytosis, and in typhoid fever and diphtheria. Malignant disease with hemorrhage which causes leucocytosis is, however, associated with diminution of the eosinophiles. Neusser has indicated the following diagnostic points of value in eosinophilia. They are given by Cabot as follows:

1. In the diagnosis between puerperal mania and puerperal sepsis, eosinophilia points to the former.

2. Between a tumor connected with the genital system and one not so connected, eosinophilia points to the former.

3. In determining whether a given case of hysteria, neurosis, or psychosis is likely to be benefited by castration, the presence of eosinophilia favors the operation.

4. In malignant disease an eosinophilia points to a metastasis in the osseous system (tumors of the spleen are not included in this rule).

5. In cases of doubtful syphilis, eosinophilia combined with lymphocytosis (see above) speaks in favor of syphilis.

6. The diagnosis of any obscure form of "uric-acid diathesis" is helped by finding an increase of eosinophiles.

7. In distinguishing malignant liver disease from other liver disease, eosinophilia points to the latter.

Pathologic Leucocytes. Myelocytes. The occurrence of myelocytes in the blood is pathological. Their well-known occurrence in myelogenous leukæmia and pernicious anæmia need not be referred to. They have been found, however, in a number of infections, but usually only when there is present a grave form of anæmia. Their occurrence is not of great diagnostic value. They are *non-amœboid*. They are large mononuclear neutrophiles or eosinophiles, with large, well-defined, lateral, spherical nuclei. Occasionally they are small when they are recognized by the granules and the very pale large nucleus. The "*mast-zellen*" are mononuclear, coarsely granular basophiles. The nucleus is fragmented or three-lobed.

Neusser's Granules. When making a differential count we also study certain granules in the leucocytes. Neusser has described perinuclear basophilic granulations in the leucocytes, which are demonstrated by staining the blood with the following modification of Ehrlich's triple stain:

Saturated aqueous solution of acid fuchsin	50 c.c.
Saturated aqueous solution of orange-G.	70 "
Saturated aqueous solution of methyl green	80 "
Aquæ dest.	150 "
Abs. alcohol	80 "
Glycerin	20 "

The granules in question occur as separate bodies or as groups, lying in the protoplasm immediately around the nucleus. They are met with in the mononuclear forms in particular, and, according to Neusser, are composed of some derivative of the nucleo-albumin and indicative of increased uric-acid formation. The granules occur in gout, and also in certain cases of myelogenous leukæmia, tuberculosis, diabetes, and

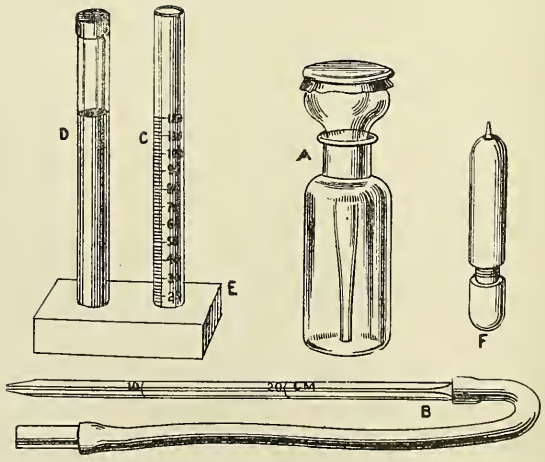
other diseases. They are significant of a uric acid diathesis "in the clinical sense." In discussing Neusser's paper, Lönit called attention to the fact that similar granules occur in the leucocytes of the bone-marrow of rabbits.

Other observers have found these granules in a variety of conditions, and incline to regard them of less significance than Neusser is disposed to admit.

The Hæmoglobin. An estimation of hæmoglobin is made, in order to determine the richness of red cells in this substance. For this purpose a hæmoglobinometer is used.

Hæmoglobinometers. Gowers' hæmoglobinometer (Fig. 104) consists of (1) a closed tube, D, containing coloring-matter representing the color human blood should have normally if diluted one hundred times; (2) a corresponding empty tube, C, graduated in an ascending scale from 10 to 120; (3) a capillary glass tube, B, marked at 20 cubic millimetres; a small guarded lancet, F, and a small bottle with a pipette stopper, A, for distilled water. A few drops of distilled water are first placed in the empty tube, C, to prevent the coagulation of the blood, which would occur if the blood were first put in the tube. The finger or lobe of the ear, previously cleansed with water and ether, is then deeply stabbed with the lancet, so that the blood will flow freely, care being taken to avoid squeezing the punctured part; 20 cubic millimetres of blood are then quickly drawn up in the capillary tube and

Fig. 104.



Gowers' hæmoglobinometer.

at once blown into the graduated tube, which is shaken, to allow the blood to become diffused in the water. The tubes containing the standard coloring-matter and the diluted blood are now held up, side by side, against a sheet of paper, and more distilled water added, drop by drop, with repeated shakings, until the colors in the two tubes match. The height to which the column of diluted blood and water has risen

in the graduated tube represents the percentage of hæmoglobin contained in the blood tested.

Fleischl's hæmometer consists of a small metal table with an aperture in the middle, under which is a reflector made of plaster-of-Paris. The opening is occupied by a small well having a glass bottom and divided into two equal compartments. The standard color of the blood at different dilutions is represented by a wedge of glass, colored with Cassius purple, which is, of course, pale in color at the extreme edge and deepens in intensity with its thickness. This wedge of glass is moved under the table by a rack and pinion, and is accompanied by a graduated scale. One-half of the well receives simply the light from the plaster-of-Paris reflector, while the other rests upon the ruby glass and obtains light through it. The light from a candle, gas-jet, or oil-lamp must be used. A small pipette and several capillary tubes about $\frac{3}{8}$ inch in length, and mounted on slender metal handles, are employed to obtain the necessary amount of blood; each one of them will hold enough normal blood to produce, when properly diluted, a color corresponding to that of the ruby glass at the 100 mark. For use, one end of a capillary tube is carefully lowered upon a drop of blood, which immediately fills it; the tube is then at once washed in one of the compartments of the well, which contains some water. The compartments are now equally filled with water, and the well so placed that the side containing blood receives yellow light, as from a candle, while the other receives light through the wedge of glass. The glass is now moved by the rack and pinion until the intensity of the color in the two compartments is the same, and the percentage is then read off through the small opening behind the well.

Both Gowers' and Fleischl's instruments are about equally accurate, and both are graduated for a higher percentage of hæmoglobin than is the average with Americans, which may be as low as 96 per cent.

Color-index. The hæmoglobin usually increases or diminishes with increase and diminution of the red cells. If there is any variation from this percentage the determination of this variation is known as the color-index. In a healthy individual with 5,000,000 red cells per cm. the normal percentage of hæmoglobin should be 100. We then say the color-index = 1. If the hæmoglobin is diminished, the color-index is less than 1. The color-index is estimated, first, by reducing the count of the cells to a percentage; second, by dividing this percentage into the hæmoglobin percentage. Thus if the normal percentage of red cells is present—that is 100—and the hæmoglobin is reduced to 50 per cent., the color-index is $\frac{50}{100}$, or 0.5; a reduction of the red cells to 2,500,000 cells = 50 per cent. of the normal. Now, if the hæmoglobin is 40 per cent., the color-index will be $\frac{40}{50}$, or 0.8.

Diminution in the amount of hæmoglobin is seen in anæmia, and usually the reduction is lower than the reduction of the red cells. In chlorosis the reduction in hæmoglobin is very great, and in consequence the color-index is lower than in secondary anæmias. The average hæmoglobin per cent. in a large number of chlorotic cases studied by Cabot and by Thayer was about 42 per cent. At the same time in most of these cases the number of red corpuscles was over 4,000,000.

Melanæmia. Melanæmia is a rare condition, in which black, brown, or yellow granules are seen floating, either free among the blood-cells, or, more commonly, enclosed in cells resembling leucocytes. They are present in malarial fevers, particularly the chronic forms, and in relapsing fever.

Lipæmia is the presence in the blood of fat, usually in the form of small droplets, easily detected by the microscope. The diagnosis can be confirmed by treating the fresh preparation with a 1 per cent. solution of osmic acid, followed by a weak aqueous solution of eosin. The fat-drops will appear black among the faintly stained acid corpuscles. A saturated solution of Soudan three in 96 per cent. alcohol will stain fat drops bright red or orange. Lipæmia occurs in chronic alcoholism, chronic nephritis, and diabetes, and after-injuries to the bone-marrow.

The Acidity of Blood. The total acidity of the blood is best determined by Landois' titration-method, as follows: Prepare a decinormal solution of tartaric acid by dissolving 7.5 grammes of the chemically pure salt in 1 litre of distilled water. By diluting centinormal and millinormal solutions are obtained. Prepare a series of solutions as follows:

I. contains 0.9 c.c. centinormal solution tartaric acid + 0.1 c.c. saturated potassium sulphate solution.

II. contains 0.8 c.c. centinormal solution tartaric acid + 0.2 c.c. sulphate solution.

IX. contains 0.1 centinormal acid + 0.9 c.c. sulphate solution.

X. contains 0.9 c.c. millinormal acid + 0.1 c.c. sulphate solution.

XVIII. contains 0.1 c.c. millinormal acid + 0.9 c.c. sulphate solution.

In each of a series of watch-glasses mix 1 c.c. fluid (each watch-glass containing a different strength, as in the series above given) with 0.1 c.c. of blood. This can be done by a graduated pipette. The pipette of a Thoma-Zeiss hæmocytometer answers very well.

Test the contents of each watch-glass with a strip of delicate litmus-paper, and note in which solution the reaction is neutral. This operation must be done quickly, the whole process not taking more than one and a half minutes (V. Jaksch).

Suppose 0.4 c.c. tartaric acid neutralizes 1 c.c. of blood; now, 0.4 c.c. tartaric acid neutralizes 0.0016 gramme caustic soda. Therefore 0.1 c.c. blood = 0.0016 sodic hydrate and 1 c.c. = 0.16. The normal alkalinity is 1 part NaOH to 26 to 30 parts of blood, or 1 c.c. blood = 0.33 to 0.38 gramme NaOH.

The alkalinity of the blood is diminished in:

1. Fevers and cachexias.

2. Toxic conditions, as uræmia, diabetes, and jaundice. Or certain poisons, as CO_2 and phosphorus.

3. Pernicious anæmia, simple anæmia, and leukæmia.

4. Chronic articular rheumatism and gout (*not* in acute articular rheumatism). This may, perhaps, be due to the accompanying anæmia.

It is increased, perhaps, in chlorosis, though this is doubted by some authorities.

Uric Acid. *Garrod's test.* By this test we can determine the presence or absence of *large* amounts of uric acid in the blood. A few c.c.

of blood-serum or of serous fluid are placed in a watch-crystal; add to this 6 to 10 drops of a 30 per cent. solution of acetic acid. Immerse a thread of linen in the fluid, and keep it at a low temperature for from twelve to twenty-four hours. If uric acid is present in large amounts at the end of twenty-four hours, crystals collect upon the thread. Their true nature is determined by the microscope (see Urine) and the murexide test. The serum may be secured by a blister.

The Specific Gravity. The specific gravity of the blood is best determined by the following method:

Prepare a series of solutions of water and glycerin in such proportions that they form a series gradually ascending in specific gravity from 1040 to 1080. Place from 80 to 100 c.c. of each solution in a series of small glass jars and bring a drop of blood exactly in the middle of each, as follows: A hypodermic syringe is connected by a small rubber tube with a right-angled glass capillary tube. A drop of blood is obtained from the finger in the usual manner, and is drawn by means of the syringe into the capillary tube. By a gentle motion of the syringe a small drop is expelled into the fluid from the point of the tube. The drop will remain stationary if the specific gravity of the fluid equals that of the blood; it will sink if the fluid be of less specific gravity than that of the blood, or will rise if the fluid be of greater specific gravity than the blood. By repeated examination the specific gravity of any specimen can be easily determined. The glycerin mixture can be preserved by the addition of a small amount of thymol, and may be used a second time; but in this case it is necessary to redetermine its specific gravity before each usage. By the specific gravity one can estimate the amount of hæmoglobin because the former runs parallel to the percentage of the latter. Two methods are employed—the water and glycerin method and the method of Hammerschlag.

Hammerschlag's method is as follows: Mix in a urinometer glass such quantities of chloroform and benzol that the specific gravity is about 1059. Take a drop of blood from the punctured ear by a medicine dropper or a capillary tube, and blow it into the chloroform-benzol mixture. The blood does not mix but floats like a red bead. Add chloroform, drop by drop, if the blood sinks to the bottom. Add benzol if it rises to the top. After each addition stir the mixture with a glass rod. When the drop remains stationary in the body of the fluid its specific gravity is the same as that of the fluid as a whole. Take the specific gravity and you have the specific gravity of the blood. Air should not be blown into the fluid with the blood drop. The following table gives the relations of the specific gravity to the hæmoglobin, from which an estimate of the hæmoglobin can be made:

Specific gravity.				*Hæmoglobin.*			
1033	to	1035	. . .	25	to	30	per cent.
1035	"	1038	. . .	30	"	35	"
1038	"	1040	. . .	35	"	40	"
1040	"	1045	. . .	40	"	45	"
1045	"	1048	. . .	45	"	55	"
1048	"	1050	. . .	55	"	65	"
1050	"	1053	. . .	65	"	70	"
1053	"	1055	. . .	70	"	75	"
1055	"	1057	. . .	75	"	85	"
1057	"	1060	. . .	85	"	95	"

The specific gravity of the blood is normally less in women, and is diminished in severe symptomatic anæmias, pernicious anæmia, chlorosis, leukæmia, and, according to Monti (*Archiv. f. Kinderheilk.*, Bd. xviii. S. 161), in nephritis. It is increased in infancy and acute febrile diseases, as pneumonia, pleurisy, etc. (Monti, *ibid.*), and also in diphtheria (Fibrenthal and Bernhard, *ibid.*, Bd. xvii. H. 5 u. 6).

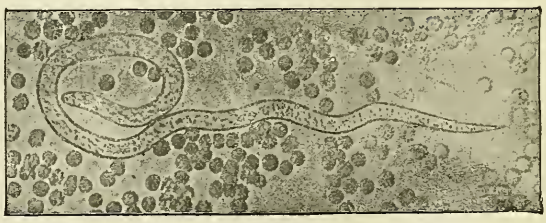
Coagulation Time. An estimate of the time required for the blood to clot is valuable, particularly in prognosis. In case of jaundice, for instance, in which blood destruction is going on rapidly, it is well to know the clotting power of the blood, as surgical interference should be resorted to in obstructive forms whenever the coagulation time is very rapid. The method devised by Wright is the best at our command.

Parasites in the Blood.

The principal vegetable parasites are those associated with the infections and described in Chapters XIX and XX. They are (1) spirilla of relapsing fever; (2) tubercle-bacilli; (3) anthrax-bacilli; (4) bacilli of glanders; (5) typhoid bacilli; (6) streptococci and staphylococci; (7) the bacilli of yellow fever.

The animal parasites are: (1) Filaria sanguinis hominis; (2) distoma hæmatobium; (3) plasmodium of malaria.

Fig. 105.



Filaria alive in the blood. Instantaneous photomicrograph. Four hundred diameters magnification. Four millimetres Zeiss apochromatic. (F. P. Henry.)

The Filaria Sanguinis Hominis. Filariæ are found in the blood and lymph of persons who live in the tropics, and in a few instances have been found in native Americans (John Guitéras). They have a blunt, rounded head with a tongue-like process and a long, pointed tail.

They produce lymphatic swellings (particularly of the scrotum), chyluria, and hæmaturia.

Patrick Manson[1] says the following are the commonest mistakes in the search for filariæ: (1) The use of too high a magnifying-power; (2) employing too strong illumination; (3) searching unmethodically and in too small a quantity of blood; (4) looking for filariæ in blood drawn from the body at a time when the particular species sought for is normally absent from the circulation. He describes three forms: *Filaria sanguinis hominis nocturna* (the ordinary form); *filaria sanguinis hominis diurna;* and *perstans.* The last appears to be the one

[1] Trans. Seventh International Congress of Hygiene and Dermography, vol. i. p. 93.

associated with the production of the disease known on the west coast of Africa as "*sleeping sickness.*" He prefers dry preparations of the blood, stained with a $\frac{1}{2}$ per cent. eosin solution or a weak solution of fuchsin (one drop of the saturated alcoholic solution to an ounce of water). If a thin film of blood, before it has fully dried, be held over acetic acid so as to imbibe the fumes, and be then stained in a $\frac{1}{2}$ per cent. solution of eosin, the blood is stained, but any filariæ remain pearly white.

The filariæ may have been discovered accidentally, or are sought for because of *hæmato-chyluria*, or *lymph-scrotum*, *elephantiasis*, or varicose groin *glands* ("Demerara groin"). In the former the chyluria is intermittent. Microscopically, the urine contains molecular fat-globules or granules and a few red corpuscles.

ANÆMIA.

Anæmia is a condition characterized by a reduction in the number of red blood-cells, or of their hæmoglobin, or of the albumin, or of all combined.

The most casual observation may be sufficient for the recognition of anæmia. The color of the surface, the appearance of the mucous membranes, and the evident breathlessness of the patient are indications of diminution in the amount of blood, or of some of its constituents, as the red cells, or of the coloring matter of these cells. On inquiry it would be found that the patient is easily prostrated, that there is breathlessness on exertion (aggravated on ascending any height), that there is palpitation and perhaps cardiac oppression. The patient will complain of neuralgias in various parts of the body, and especially of the neuralgia so often seen in the inframammary region of the left side. (See Pain.) Headache will be a more or less constant symptom, and of this peculiarity, that it is increased when the patient goes up stairs, and is often throbbing or pulsating. The anæmic subject has usually a poor appetite and suffers from gastralgia, although it must be remembered that the gastric symptoms of anæmia are as often primary as secondary. Many of the train of symptoms which attend neurasthenia occur in the course of anæmia.

On physical examination of the patient the appearances as above indicated are found, although grave anæmias may be present, and yet the lips are bright red, the color under the nails fair, and the cheeks flushed, especially if the examination is made in the evening. Reference must be made to the chapter on the Color or Hue of the Surface for a description of the appearances of anæmia.

A study of the heart and bloodvessels usually yields the physical signs that attend anæmia. The vascular phenomena are described in the section on Diseases of the Heart. Here, again, it must be remembered that considerable anæmia may be present without any murmurs in the bloodvessels.

The Blood. The final diagnosis rests upon an examination of the blood. Sometimes the most apparently anæmic subjects yield normal results in blood examination, while the most plethoric in appearance

may be very anæmic. The various forms of anæmia give rise to blood changes in a measure peculiar to the respective variety. The primary anæmias, or hæmolytic varieties, to which pernicious anæmia and chlorosis belong, have characteristics which will be described in the special sections.

In anæmia from hemorrhage the red corpuscles may be reduced to 1,500,000. The hæmoglobin is reduced to a degree greater than that of the red cells. The leucocytes are increased in number, the polynuclear forms being relatively much less than the other varieties.

The red corpuscles are paler than normal; their white centres are increased in size. This is known as *achromia*. There is some poikilocytosis. An excess of nucleated red corpuscles, or *blasts*, are seen in grave anæmias. If the *normoblasts* are in excess, active regeneration is in progress; if the megaloblasts, there is reversion to embryonal regeneration, a serious import in an anæmia. A megaloblast anæmia is associated with general increase in size of red cells and an increase of the macrocytes. In fatal anæmia, as in purpura, the red cells are like those in the form just described, although nucleated red corpuscles are absent. The white cells are also reduced, although the mononuclear forms are numerous.

In the oligocythæmic forms of anæmia, other than the hemorrhagic, the occurrence of poikilocytosis is constant and marked. Nucleated red corpuscles are not common, but large nucleated cells in which karyokinetic figures occur. These corpuscles have pale staining nuclei. Achromic forms, polychromatophiles, and degenerate forms are seen. There is usually moderate leucocytosis.

For clinical purposes it is necessary to make a number of divisions of anæmia, though on etiological and pathological grounds many of them will no doubt soon be grouped together.

The following classification of anæmias is helpful in the study of anæmia. In it both pernicious anæmia and chlorosis are regarded as hæmolytic in origin, the destructive agent probably being absorbed from the intestine.

- ANÆMIA,
  - Non-cytogenic,
    - Hæmolytic,
      - Pernicious anæmia.
      - Other toxic anæmias.
      - Chlorosis.
      - Parasitic anæmia (some forms).
    - Oligocythæmic,
      - Parasitic anæmia (some forms).
      - Post-hemorrhagic anæmia.
      - Anæmia from loss of albumin.
      - Anæmia of malnutrition.
  - Cytogenic,
    - Leucocytic,
      - Leucocythæmia,
        - Spleno-myelogenic.
        - Lymphatic.
        - Medullary or myelogenic.
    - Non-leucocytic,
      - Hodgkin's disease (?).

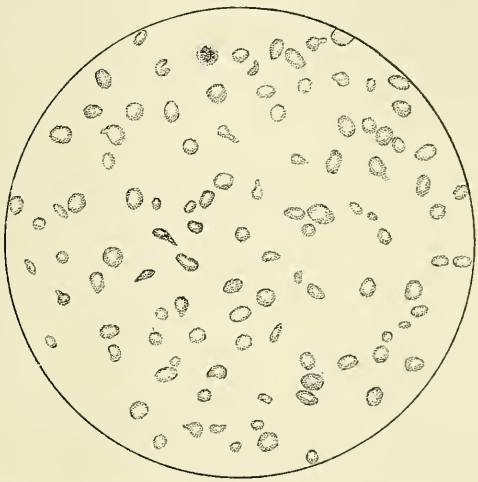
I. Toxic Anæmias. The poison may be developed in the body or introduced from without. Toxæmia is, sometimes at least, a factor in the anæmias which develop in the course of acute infectious diseases or during convalescence from them. According to Hunter, pernicious anæmia should be classed under this head. The metallic poisons, par-

ticularly lead, mercury, arsenic, phosphorus, the potassium salts, especially the chlorate; certain of the antipyretics, notably pyrodin; and the aniline-preparations are capable of producing anæmia.

II. Parasitic Anæmias. Anæmia may be *parasitic*. 1. To this class belongs the anæmia of malaria, which is believed to be due to the *plasmodium malariæ* described by Laveran.

2. Certain intestinal worms are found associated with marked anæmias. (*a*) The *bothriocephalus latus* sometimes produces a disease closely resembling pernicious anæmia, but whether by direct destruction of the blood, or by the development of toxic products, themselves destructive, is uncertain; it may be present in large numbers without giving rise to anæmia.

FIG. 106.



Severe anæmia (Reproduced from colored plate.) Dry preparation. × 300. Great poikilocytosis. Many macrocytes and microcytes. To the left above, a mononuclear leucocyte.

(*b*) The *ankylostomum duodenale* is believed to be the cause of the anæmia known variously as Egyptian or African chlorosis, tropical anæmia, brick-burner's anæmia, etc.

(*c*) The *anguillula intestinalis* is the cause of "Cochin-China diarrhœa" and its associated anæmia.

3. The *filaria sanguinis hominis* may produce anæmia by blocking up the lymph-channels.

4. The *Bilharzia hæmatobia* may produce anæmia by inducing hæmaturia.

III. Anæmia from Hemorrhage. Anæmia may be due to *hemorrhage*. In addition to accidental and *post-partum* causes, purpura, hæmophilia, menorrhagia, and metrorrhagia are frequent causes.

IV. Anæmia from Constitutional and Local Diseases. Anæmia is often a marked symptom of *constitutional* and *local diseases*, such as tuberculosis, syphilis, cancer, rheumatism, scrofula, scurvy, rickets, Bright's disease, chronic catarrhal gastritis, and others. The anæmia here may be due to the malnutrition and interference with digestion

brought about by the disease, or, as in the case of Bright's disease, in part to the direct loss of albumin, and in dyspeptic conditions to inability to take and assimilate food.

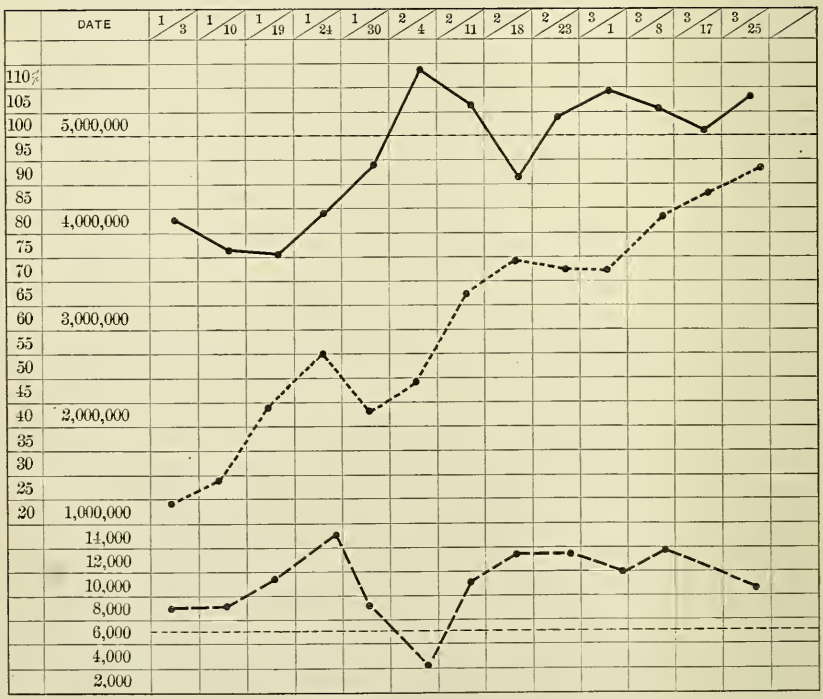
In many cases of simple symptomatic anæmia the spleen may become progressively enlarged, probably secondarily. In other cases there is an enlargement of the spleen in Hodgkin's disease. In no case is there a primary splenic anæmia.

V. Anæmia of Malnutrition. Anæmia may also be the result of malnutrition from deficient or improper food, or from the poisonous influences of unsanitary surroundings.

Chlorosis.

Chlorosis, or chloro-anæmia, is a form of anæmia occurring especially in young girls about the period of puberty, and characterized by great pallor of the skin and mucous membranes, with a greenish tint of the skin, a pearly eye, languor, weariness, suppression or irregularity of

FIG. 107.

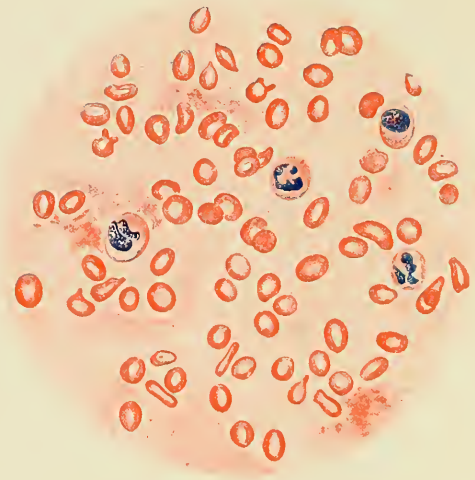


Chlorosis. Straight lines, number of red cells; small dots, per cent. of hæmoglobin; large dots, number of white cells.

menstruation, venous hum in the vessels, dyspnœa, palpitation, dizziness, neuralgias, and an unstable condition of the nervous system. In spite of the extreme pallor there is usually but little loss of flesh. The skin

PLATE XI.

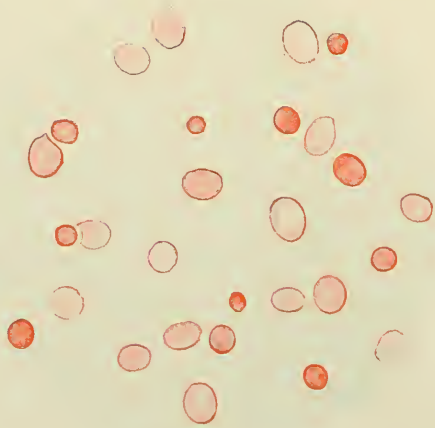
FIG. 1.



Blood from Case of Chlorosis, showing slight Staining of the Red Blood-corpuscles, and presence of Mononuclear Leucocytes.

(Oc. 4, ob. $\frac{1}{12}$ immersion.) Drawn by J. D. Z. Chase.

FIG. 2.



Blood in Pernicious Anæmia, showing Macrocytes and Microcytes.

(Eosin stain, oc. 4, ob. $\frac{1}{12}$ oil immersion.) Drawn by J. D. Z. Chase.

may be pigmented, especially around joints. The bowels are usually constipated; the urine abundant, pale, and of low specific gravity. The digestion is disturbed, the appetite capricious, and the patients sometimes crave unwholesome things, such as earth, slate-pencils, vinegar, and the like. Hyperacidity of gastric juice is commonly present. A systolic murmur over the base of the heart is common. Gastralgia is more common than in other forms of anæmia.

The changes in the blood are very important. There is always a marked reduction in the hæmoglobin, the percentage falling sometimes to 30 or 25 per cent. of the normal. The red blood-cells are usually also reduced, but not in the same proportion as the hæmoglobin. For example, there may be 4,000,000 red cells, but only 30 per cent. of hæmoglobin. Sometimes there is no diminution in the number of red cells; the latter, however, appear pale (achromia), vary considerably in size, microcytes and occasionally poikilocytes are present, and, in severe cases, nucleated red corpuscles are found; occasionally macrocytes occur, but in general the size of the red cells is below that which is usually found. The number of leucocytes varies but little from the normal, but there may be a slight increase. Occasionally there is a rise of temperature, but it is probably due to some complication. (See Plate XI., Fig. 1.)

The cause of chlorosis has not been determined satisfactorily. Virchow has established the existence of congenital narrowing of the blood-vessels. Sir Andrew Clark thinks it is due to the absorption of poisonous matter from the intestine; the great benefit that follows saline purgatives in many cases indicates that fæcal toxæmia is a factor in these cases. Forchheimer[1] also looks upon it as intestinal in origin.

Sex and puberty are predisposing causes; but chlorosis may occur in boys, and appear in girls before puberty, and in young women considerably after that period. The prognosis is favorable; it may, however, be complicated with gastric ulcer, chorea, tuberculosis, and endocarditis. Recovery is often slow and interrupted by relapses.

Pernicious Anæmia.

Pernicious or idiopathic anæmia is a form in which the diminution of red blood-cells reaches an extreme degree. It occurs without adequate known cause, and runs a progressive course with remissions; it usually terminates in death.

The disease usually develops slowly and insidiously, the patient presenting the ordinary symptoms of anæmia—pallor, weakness, shortness of breath, palpitation, venous murmurs, loss of appetite, and impaired digestion. As the disease progresses the skin becomes of a pale lemon hue, weakness and dyspnœa increase, the patient has attacks of dizziness, faintness, and ringing in the ears; there may be slight œdema, and hemorrhages from the nose, the bowels, and into the retina occur. The hemorrhages are small and distinct in the skin and mucous membranes. The urine is of low specific gravity, and usually contains an increased amount of uric acid. According to Hunter, the urine should

[1] Trans. Assoc. Amer. Phys., 1893.

be dark and contain a pathological amount of urobilin, some renal epithelium, a few casts containing blood-pigment, and an increased amount of iron. The bowels may be disturbed by diarrhœa.

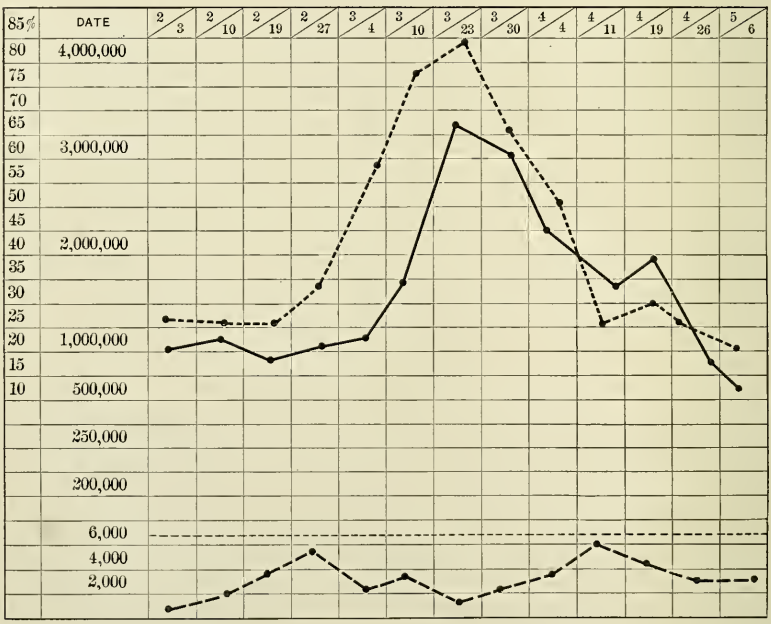
A peculiarity of the disease is the occurrence of fever of an irregular type. The temperature rarely rises higher than 102° or 103° in the evenings, and is followed by a morning remission. It is not usually present in the early stages of disease, may be absent for weeks at a time when the disease is fully developed, and may cease entirely in the later stages.[1]

In spite of extreme exhaustion, anæmia, and wide-spread functional disturbance, there is no emaciation ; the patient appears well nourished.

The blood appears pale and watery to the naked eye ; there is difficulty in obtaining by puncture a sufficiently large drop for examination. The specific gravity is lowered, often being 1028 instead of 1055. It has been found deficient in fibrin, iron, and nitrogen.

The blood-changes in idiopathic anæmia are characteristic, and are essential to the diagnosis of the disease. In brief they are : (1) Very great reduction in the number of red blood-cells ; (2) an absolute dimi-

Fig. 108.



Pernicious anæmia. Straight lines, number of red cells; small dots, per cent. of hæmoglobin; large dots, number of white cells.

nution in the amount of hæmoglobin, but as compared with the number of red cells there may be a proportionate increase ; (3) considerable

[1] See "Idiopathic Anæmia: A Report of Three Cases." Musser, Phila. Co. Med. Soc. Trans., 1885.

variation in the size of the cells, the average size of the cells probably being larger; (4) poikilocytosis; (5) nucleated red blood-cells; (6) degenerative cells. (See Plate XI., Fig. 2.)

Reduction in the number of red blood-cells (oligocythæmia) reaches a more extreme degree in pernicious anæmia than in any other disease; the number often falls below 1,000,000, and in one case reported by Quincke[1] the number was only 143,000 per cubic millimetre. The shape of many of the cells is altered; they are oval, elongated, bent, or have projections of their substance (poikilocytosis). The size of the cells varies; there are microcytes and megaloblasts; but the occurrence of a distinct proportion of large nucleated red blood-cells (megaloblasts) is regarded by Ehrlich as almost diagnostic. The average size of the red cell seems to be increased, and so is the proportionate amount of hæmoglobin in each cell. The latter is a very characteristic symptom (the only one, according to Hunter). There are also red corpuscles which are stained by methylene-blue; these are regarded as degenerative by Ehrlich. The leucocytes are "usually diminished in number, showing a relative increase in the small mononuclear elements (lymphocytes, small transparent forms), while the multinuclear elements are relatively diminished, sometimes being under 50 per cent." [2]

The blood condition is not constant, but is subject to wide variations. Von Noorden has recently found that in a very short time a change in the form of the blood, a "formal" crisis, may occur. A "formal" overflow of the blood with polynuclear leucocytes and nucleated red blood-cells takes place before a period of improvement. Whereas, before a period in which the blood becomes worse and before the final stage, the blood becomes poor in leucocytes and nucleated red blood-cells.[3]

Secondary selerotic changes in the spinal cord cause late symptoms of *locomotor ataxia*.

The etiology of the disease has not been determined satisfactorily. It is more common in Germany and Switzerland than in other parts of Europe or in America. It occurs most frequently after the twentieth year, and between that and the age of fifty. Excluding the influence of pregnancy and parturition, sex makes no difference. Previous exhausting disease, chronic gastric and intestinal catarrh, great physical over-exertion, exposure, great shock or fright, precede in certain cases the development of the disease. It is probably due to faulty hæmatogenesis and hæmolysis.

Petrone and Halst regard the disease as infectious and its germ identical with that found by Frankenhauser. Von Jaksch supposes that it is brought about by a living contagium. Hunter traces the cause to a poison produced by bacteria in the gastro-intestinal canal.

Diagnosis. The most important diagnostic features of the disease are extreme oligocythæmia, relatively high percentage of hæmoglobin (color-index high), great poikilocytosis, which may, however, occur in

[1] Deut. Arch. für klin. Med., Bd. xx.
[2] W. S. Thayer: Boston Med. and Surg. Journ., February 16 and 23, 1893.
[3] Quoted by Weiss, Diagnostisches Lexikon.

any severe anæmia, a noticeable number of large nucleated red blood-cells (gigantoblasts), an average increase in the size of the cells, and all this without emaciation or discoverable local disease which can bear a causative relation to the anæmia. In addition, retinal, subcutaneous, and submucous hemorrhages, a urine with high specific gravity, high color, with urobilin in excess, alternating with urine of low specific gravity, in the absence of organic disease, point to *pernicious* or *idiopathic* anæmia.

Leucocythæmia.

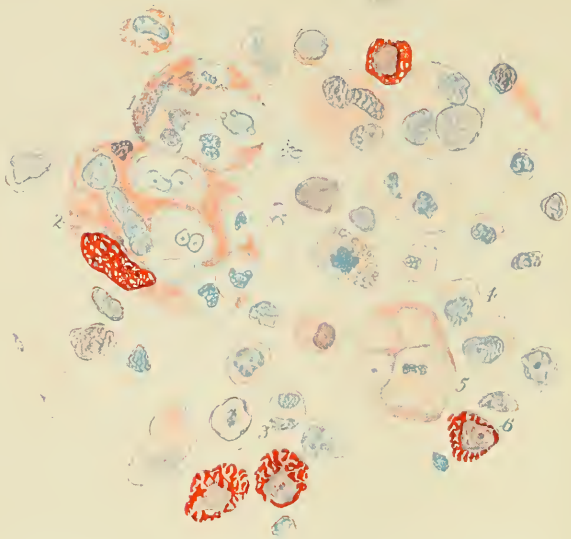
Leucocythæmia, or leukæmia, is a disease of the blood-making organs, characterized by great and persistent increase in the white blood-corpuscles; by a diminished number of red blood-cells, which are altered in shape and size, and display nucleated and degenerate forms; by a lessened amount of hæmoglobin, and by changes in the spleen, lymphatic glands, or medulla of bone. It is a persistent and progressive cellular proliferation. It resembles a tumor of solid tissue in its cellular overgrowth. The disease occurs twice as frequently in men as in women, and two-thirds of the cases appear between the twentieth and fiftieth years. In women, pregnancy, parturition, and the cessation of menstruation are causative factors, while in both sexes depressing influences upon body or mind and antecedent disease, particularly malarial fever, have a distinct influence.

The first symptom noted is generally enlargement of the abdomen; subsequently the patient complains of pains in the splenic region, weakness, dyspnœa, hemorrhage, œdema, and digestive derangements. Occasionally profuse hemorrhage from trifling cause, as the drawing of a tooth, has been the earliest symptom noted. The increase of white cells and diminution of red cells is progressive, and soon makes itself evident in the pallor of the skin and mucous membranes, and in increasing weakness and dyspnœa. Pallor is not a constant symptom of leukæmia. A high grade of color is consistent with advanced leukæmia.

In the so-called *spleno-medullary form* of the disease the spleen steadily enlarges, but may attain considerable size before the patient becomes aware of it. The enlargement is not usually painful, but gives rise to a feeling of distention, weight, and dragging. There may be tenderness on palpation and pressure, and sometimes the patient complains of sharp, stabbing pains, due either to attacks of local peritonitis or to sudden enlargement of the spleen and consequent stretching of the capsule. The splenic enlargement is uniform, so that its shape and characteristic notch are unchanged. Moreover, the spleen remains in contact with the abdominal walls, lying in front of the splenic flexure of the colon, pushing aside the descending colon and small intestine, moving with respiration, and presenting the usual physical signs of a solid organ. Not infrequently the enlargement is so great as to fill the left hypochondriac and iliac regions, and reach beyond the middle line toward the right groin. Sometimes a venous hum can be heard over it. Pallor, however, is not a constant symptom; more frequently the cheeks are flushed and the lips red.

PLATE XII.

FIG. 1.



Lymph-gland, Retroperitoneal Region.

Hardened in Alcohol; Rosin's Stain. × 1500.

1. Globiferous cell.
2. Globiferous cell containing polynuclear and eosinophile cells.
3. Polynuclear cell.
4. Mononuclear cell.
5. Globiferous cell.
6. Eosinophile cell.

FIG. 2.

Blood—Leukæmia.

Hæmatoxylin and Eosin.

1. Lymphocyte.
2. Eosinophile cell (mononuclear).
3. Polynuclear leucocyte.
4. Large mononuclear leucocyte (myelocyte?).

As the result of this enlargement the diaphragm is pushed upward, increasing the dyspnœa already caused by anæmia, and sometimes inducing palpitation. The gastric functions are disturbed from pressure; vomiting and other symptoms of dyspepsia are common.

A rise in temperature is a very common symptom. The fever is of irregular type, usually with nocturnal exacerbations, the temperature not often rising above 102°. The febrile type may be intermittent or remittent, and sometimes there are periods of apyrexia.

The pyrexia is said to be most marked toward the close of the disease. Gowers states that the cases in which there is most fever are usually those of rapid course, considerable dropsy, and extensive hemorrhage.

As the disease progresses weakness increases; anæmia becomes more intense; œdema, ascites, or hydrothorax occurs; hemorrhages from the nose, gums, bowels, stomach, lungs, or uterus further exhaust the patient; digestion is poor and diarrhœa is common.

Headache and tinnitus are frequent symptoms, occasionally delirium and coma may occur, and deafness is not uncommon toward the close of the disease. The eyes may be the seat of leukæmic retinitis.

The liver is enlarged, often to a considerable degree, but without special symptoms. The same is true of the lymphatic glands and other adenoid tissue. (See Plate XII., Fig. 1.) The marrow of the bones becomes the seat of disease in some cases, but it does not usually give rise to symptoms during life; certain bones, however, may be tender.[1]

The Blood. The most characteristic and important changes from a diagnostic point of view occur in the blood. The blood when drawn from the finger is strikingly pale and whitish, an appearance supposed at one time by Bennett to be due to admixture of pus. It coagulates slowly, is of lower specific gravity than normal, and its alkalinity is diminished. When placed under the microscope it is at once seen that the number of white cells is greatly increased. If a drop of blood is mixed with some distilled water containing a small quantity of gentian-violet, the white cells are stained a decided blue and can be picked out with the greatest ease. Instead of there being one white cell to 300 or 500 red, the ratio falls as low as 1 : 5 or 1 : 3, or even lower. Authorities differ as to the degree of increase necessary to distinguish leucocythæmia from leucocytosis, some including all in which the ration is 1 : 50 or lower, and others excluding those in which the ratio is greater than 1 : 20 or 1 : 12. In leucocytosis the increase takes place solely in the polynuclear neutrophilic leucocytes.

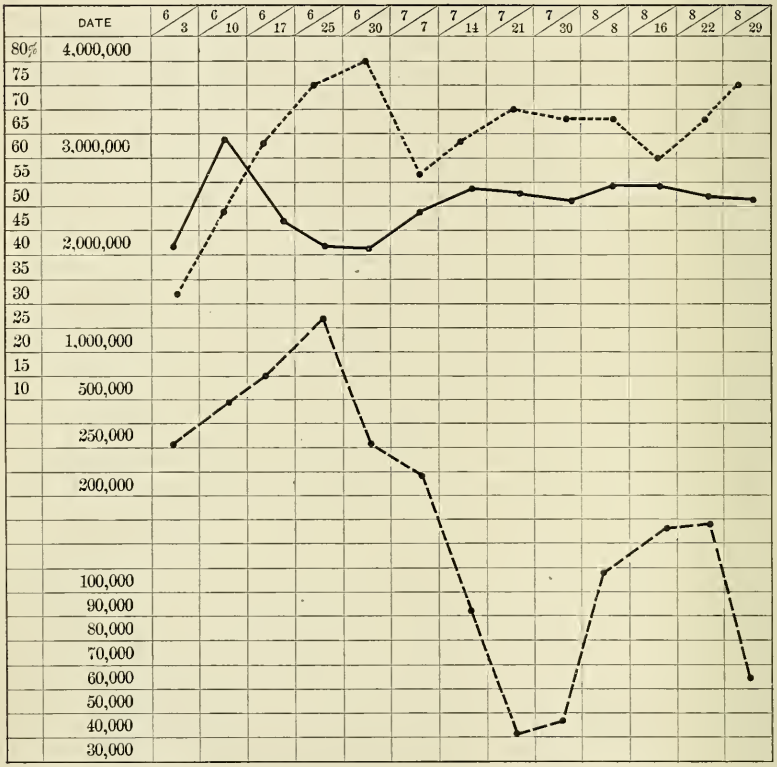
Not only the white cells greatly increase in number, but they vary considerably in size and react differently to staining-fluids.

Ehrlich has described five varieties of leucocytes. The pathological changes in the normal leucocytes in this disease are: (1) The small mononuclear elements are relatively diminished; (2) the great difference in size of the multinuclear elements; (3) the presence of myelocytic elements, in which the protoplasm is filled with fine neutrophilic

[1] See "A Case of Leucocythæmia." Musser and Sailer, Amer. Journ. of the Med. Sciences, 1896.

granules ; (4) the presence of a normal proportion of eosinophiles in so extensive an increase of leucocytes.[1] (Plate XII., Fig. 2.) (5) Large mononuclear elements with karyokinetic figures (Müller). (6) Mast-cells. Satisfactory study of these can be obtained only by cover-glass preparations.

Fig. 109.



Leukæmia. Straight line, red cells; small dots, hæmoglobin ; large dots, white cells.

The essential points in the diagnosis of leucocythæmia are : 1. Such an excess of leucocytes in the blood that the ratio of white to red falls below 1 : 50 or 1 : 20 ; if the ratio is higher, the white cells should show a progressive increase. The individual leucocytes vary in size and characteristics, as already described. 2. Enlargement of the spleen or lymphatic glands. 3. The occurrence of hemorrhages and dropsies unexplainable by disease of the heart, kidneys, or other organs. 4. The symptoms of anæmia of a high grade, as dyspnœa. 5. Leukæmic retinitis. 6. Anæmic fever. 7. The presence of the myelocyte of Ehrlich, "mast-cells," and nucleated red blood-cells. 8. Specific gravity below 1040. 9. Excess of uric acid in the urine.

[1] W. S. Thayer, loc. cit.

The *lymphatic form* of the disease is rare. It is characterized by enlargement of the lymphatic glands and by the great increase in the proportion of the lymphocytes. The total increase in the colorless elements is not so excessive. Eosinophiles and nucleated red cells are rare. The myelocyte of Ehrlich is not present. A case of a purely myelogenous form has never been authenticated. Combination-forms may also occur. It must be remembered that the number of myelocytes is no indication of the involvement of the bone-marrow.

In *secondary* or so-called *splenic anæmia* we find the same enlargement and the general symptoms, though hemorrhage is not so common. Leucocythæmia is distinguished from it by the great excess of leucocytes and by their special characteristics.

In *lymphadenoma*, or *Hodgkin's disease*, there is extreme anæmia, though the excess of leucocytes found in leucocythæmia is seldom reached, and the cells are smaller. The glandular enlargement of lymphadenoma is an early and constant symptom, the spleen not being much enlarged. The cervical glands are the ones usually first involved.

The *duration* of leucocythæmia is usually two or three years; but some cases terminate in six months or less, and some last six or seven years. The size of the spleen and the degree of oligocythæmia appear to have no influence. Gowers states that the cases in which enlargement of the lymphatic glands is an early symptom run a course apparently much more acute than others, but he admits that the number of such cases is comparatively small.

Death results most frequently from gradual loss of strength. Hemorrhage from various organs and surfaces is the immediate cause in many cases. It occurs in about three-fourths of the cases, and, when not directly fatal, increases the pre-existing asthenia. Diarrhœa and pulmonary complications are not infrequent causes of death.

Acute Leukæmia. Cases have been described, especially in children, in which there is a diminution of red cells of hæmoglobin. Nucleated red cells are present as well as an excess of white blood-corpuscles, which consist almost entirely of large mononuclear elements, without granulation. There is usually fever, and the disease runs a course much resembling an infectious one. The lesions are leucocytic infiltration of the various organs.[1]

[1] See "Acute Leukæmia." Fussell, Jopson, and Taylor, Assoc. Am. Phys., vol. x. 1898; and Musser, Trans. Phil. Co. Med. Soc., 1887.

CHAPTER XXIII.

THE MORBID PROCESSES AND THEIR SYMPTOMATOLOGY.

Knowledge of symptoms of morbid processes essential; they control conclusions drawn from data.—Morbid processes are few. I. Alterations in blood and circulation: Anæmia and plethora—Hyperæmia, active and passive—Œdema and dropsy—Thrombosis and embolism—Hemorrhage—Blood-pressure. II. Disturbances of nutrition: Inflammation—Gangrene and necrosis—Fever—Atrophy and hypertrophy. Degenerations: Albuminous—Fatty—Colloid—Mucous—Pigmentary—Calcareous—Amyloid—Fibroid. III. Anomalies of growth: Tumors—Cysts—Cancer.

Although we may have secured all the data obtainable by inquiry and by observation, and, if possible, made a diagnosis based upon them, it frequently happens that the conclusion arrived at is not final and perhaps cannot be, from the nature of the case. We are prompted, therefore, to view the case from a different stand-point, to utilize our knowledge of the phenomena of morbid processes, and, for the purpose of comparison, to review the features of such as apparently resemble the process under consideration. Thus, for instance, in an obscure case of fever, the objective and subjective phenomena have been fully inquired into—we are unable to decide whether the disease under consideration is a septic process with obscure lesion, a form of miliary tuberculosis, or of malignant endocarditis. The known symptoms of each are considered (our knowledge of such symptoms depending upon our knowledge of the phenomena of the respective morbid process) and compared with the symptoms presented by the case in question. In this manner a diagnosis by *exclusion* is made. Moreover, after a diagnosis is made, a review of the symptomatology of morbid processes serves as a check upon the conclusions that have been reached. We should also, after making a diagnosis, compare the symptoms of the process as exhibited in the patient with the symptoms which we know to be common in the suspected disease.

It is necessary, therefore, that the student should fully know the symptoms of morbid processes. Each process is characterized by special phenomena by which it can be recognized. The symptoms are modified by the function and anatomical structure of the organ in which the process takes place. Thus the pathological products of inflammation of the mucous membranes of the bronchial tubes and of the stomach are the same, but the symptoms differ, because of the difference in their functions, and hence we have cough in the former case, in the latter, vomiting. Very frequently the symptoms differ because of the physical alterations. Thus inflammation of the pericardium is similar to inflammation of the pleura, but the pressure-symp-

toms of pericarditis are entirely different, because of the anatomical relations, from the pressure-symptoms of pleuritis.

The morbid processes are not many. They include : I. Alterations in the blood and circulation ; II. Disturbances of nutrition ; III. Anomalies of growth.

I. Alterations in the Blood and Circulation. The composition and distribution of the blood affect all the tissues for weal or woe. The quantity of the blood alone will be referred to ; changes in quality will be considered under diseases of the blood. Practically the symptoms, when the quality is affected, are those of *anæmia* plus the symptoms (physical and functional) of the primarily diseased organ—as the spleen in leucocythæmia. The quantity may be increased or diminished.

1. Increased Quantity of Blood, or Plethora. Formerly this was considered an entity, and the symptoms of flushed face, hot and full head, throbbing pain, throbbing temporals, a full, strong pulse, sluggish intellect, were thought to indicate an excess of the general bulk of the blood. True plethora is rarely permanent. If transitory, the veins and not the arteries are overfilled. The symptoms are not due to general plethora but to excess of blood-pressure or to special fluxions of blood to superficial vessels, determined by a nervous mechanism. Increase in one of the cellular elements of the blood, the leucocytes, is not a plethoric condition.

2. Diminished Quantity of Blood, or Anæmia. Anæmia embraces the diminution of the bulk of the blood as well as of the red blood-cells and their hæmoglobin.

The term might be used for loss of the water of the blood, as in cholera Asiatica (see Infectious Diseases), or in serous purging. The symptoms are those of *collapse*.

Oligæmia or spanæmia are terms that may be used to define the general thinness or poverty—atrophy of the blood. Clinically, anæmia is divided into simple anæmia, general poverty of the blood ; pernicious or idiopathic anæmia, reduction in the number of red cells ; chlorosis, reduction in the quantity of hæmoglobin ; leucocythæmia, relative loss of red and increase of white corpuscles. (See Diseases of the Blood.)

3. Local Disturbance of the Circulation. A. Hyperæmia or Congestion. The process may be acute or chronic. It is usually local, although it may be general. When the latter, many organs may be simultaneously involved from a common cause.

Acute Hyperæmia. The acute or active form of hyperæmia is always local and arterial. There is an excess of blood in the part. If the skin is the seat, there are redness and increased heat, and throbbing or pulsation may be seen. The parts are swollen. The excitability of the nerves is increased, with local symptoms of warmth, fulness, or itching.

The morbid blushing, or flushing, that occurs at the menopause or reflexly from internal disorder is a hyperæmia, and in erythema of the skin hyperæmia is also very marked.

Causes. Arterial hyperæmia is caused by (1) neuroparalysis of the inhibitory or vasoconstrictor fibres, of the cervical sympathetic, splanchnic, and other sympathetic and some mixed nerves, as the sciatic; (2) neurotonic stimulation of the actively dilating or vasomotor dilator nerves, as the chorda tympani. There is relaxation of the arterial walls. This may also occur directly through the vasomotor system, being induced by heat, electricity, or chemical irritants, or from paralysis of muscular fibres, after spasmodic contraction due to cold, as in frost-bite.

(1) *Neuroparalytic Hyperæmia.* Destruction of the cervical sympathetic nerve by abscess, wounds, or a tumor pressing upon it, produces hyperæmia of the side of the face, rise of temperature, and contraction of the pupil. Later on the vascular conditions are reversed. Lesion of the fifth nerve, or one of its branches, causes hyperæmia of the iris, the conjunctiva, the cheek, the gums, and other structures supplied by it, with associate loss of sensation followed, by atrophy. The sensory symptoms have nothing to do with the vascular paralysis.

(2) *Neurotonic Hyperæmia.* After wounds of the brachial plexus hyperæmia of the fingers is seen. (See Fingers.) The local temperature rises and there is neuralgic pain. Local hyperæmia with hyperæsthesia, known as erythromelalgia, belongs to the same class, being due to affections of the nerve-trunks, or the peripheral nerve-endings. It must be remembered that a reflex hyperæmia is possible.

Chronic or Venous Hyperæmia (passive congestion). The blood accumulates in the veins, and, by backward pressure, in the capillaries. The venous capillaries are over-distended and, as compared with the arterial, much enlarged. They contain venous blood.

Any congested part, as the exterior, is bluish or purple in tint, often swollen (clubbed fingers), cooler than normal, with lessened sensation, and without pulsation. (See Cyanosis.) The dependent parts are first affected, as the legs, or the lungs. In fevers a weak heart and recumbent posture predisposes to congestion of the lungs.

Causes. Obstructive heart and lung diseases cause *general* venous congestion. Local venous congestion is caused by tumors, the pregnant uterus, or collections of fæces pressing upon the veins. It is also caused by inflammation of the veins, and thrombosis.

B. Local Anæmia. This may be due to arterial thrombosis or embolism, arterial obstruction through endarteritis, or to arterial spasm. Raynaud's disease is a form of arterial spasm. The grave effects of arterial obstruction are seen in cerebral anæmia from endarteritis, or myocarditis from obstruction of the coronary arteries.

C. Œdema and Dropsy. The changes of the circulation which produce these conditions have been referred to in previous chapters of this book. The symptoms and signs of the condition are also noted in the same section.

D. Thrombosis and Embolism. The student should be familiar with the symptoms of these conditions, and, what is fully as important, with the causes that give rise to them. Thrombi may form in the heart, the arteries, or the veins. Emboli may be formed in either heart or vessels, but lodge in the vessels only.

Thrombosis. The symptoms of thrombosis are: 1. *Mechanical.* The channel is obstructed; hyperæmia, engorgement, œdema, and cyanosis arise. Its most typical form is seen in femoral thrombosis, with cyanosis, and œdema of the leg. When an artery is obstructed the symptoms are like those of occlusion under other circumstances (see Embolism); when a vein, the mechanical symptoms vary according to the particular vein affected. Thus, in thrombosis of the coronary vein, the heart's action is interfered with. In thrombosis of the portal vein, jaundice (not because of the obstruction), œdema (ascites), and congestion of mucous membranes (gastric and intestinal) occur, as from obstruction in any vein. In thrombosis of the cerebral veins, disturbance of the function of the brain is seen; of the pulmonary veins, dyspnœa. 2. *Inflammatory or septic.* If it should happen that the thrombosis developed secondarily to an inflammation of septic origin, as in the extension of an inflammation into the radicles of the portal vein from an abscess about the rectum or vermiform appendix, the liver would be infected with micro-organisms. An infectious inflammation with chills, fever, sweats, and other phenomena of a septic character would result (pylephlebitis). 3. *Embolic.* From the thrombus emboli are sometimes swept off; hence, embolic symptoms arise in the course of thrombosis.

While thrombosis is, as a rule, easily recognized, it is necessary to call attention to the very great importance of going a step farther to look for the cause. A thorough knowledge of the causes of thrombosis often leads to the diagnosis of a thrombus when without such knowledge its presence would never have been suspected. The causes are not many. 1. Stagnation or stoppage of blood. It is seen chiefly in the veins and the heart. External pressure upon the veins: as upon the pelvic veins in pregnancy or abdominal tumor, upon the hemorrhoidal veins, upon the portal veins by tumor, upon the pulmonary veins by mediastinal tumor. It must be remembered that some change takes place in the internal coat of the vein also, but that the pressure is primary. Then we have weakness of the heart as a cause of stagnation. Feeble contractions lead to the formation of cardiac thrombi. 2. Thrombosis from changes in the vessel's walls. The change is usually inflammatory and often proceeds from wounds. If the wound was septic, the inflammation will be septic. In the heart, endocarditis; in the aorta, atheroma leads to the development of thrombi. 3. Thrombosis from the entrance of a foreign substance into the vessels. A carcinoma or other new growth may extend into the veins. Micro-organisms penetrate the vein and cause inflammation and thrombosis, or infect a previously existing thrombus. The clot is then broken and distributed throughout the system, causing pyæmia. 4. Thrombi are produced by extension. A clot enlarges by coagulating the blood next to it. A large venous distribution may become blocked, as, first the uterine veins, then the internal iliac, then the external iliac, and after that the femoral—causing the affection which frequently occurs in the puerperal form, phlegmasia alba dolens.

Embolism. An embolus is a substance which is swept into and plugs a vessel. It may be a fragment of a blood-clot (thrombus), vege-

tations from valves of the heart, parasites, new growths which had entered the veins, fat, or air. If obstruction of the vessel alone is produced, the embolism is said to be *simple;* if a new process, as inflammation, accompanies the obstruction, it is *specific*. Fragments from a thrombus in the systemic veins may become an embolus and block the pulmonary artery; a clot or portion of valve-leaflet from the left heart may block a systemic artery, as a cerebral artery or the femoral artery or its branches; a clot in the portal vein may obstruct branches in the liver.

The symptoms occur suddenly and depend upon the artery obstructed. The cutting off of the blood-supply causes cessation of function beyond the point of obstruction. In pulmonary *venous* embolism dyspnœa is pronounced, the heart's action rapid and irregular, and many cases are said to be "heart-failure." In the middle cerebral artery the embolus causes aphasia and monoplegia or hemiplegia. In embolism of the pulmonary artery cough and hemorrhage with dyspnœa occur suddenly. The patient in whom this occurs usually has had antecedent mitral regurgitation and dilated right heart.

The blocking of an artery may lead to various symptoms. If, for instance, the main artery of the leg is blocked, anastomosis may be set up; if it does not, gangrene ensues. If an artery supplying any internal organ is blocked, anastomosis may occur, if the artery is not terminal. If the artery is terminal, there results rapid necrosis or softening, as in the brain; gradual wasting, as of the kidney, or engorgement of the arterial area and diffuse hemorrhage. The latter is known as a *hemorrhagic infarct*. This may occur in the lungs (pulmonary artery), spleen, kidneys, retina, and, rarely, the intestinal canal. The symptoms of hemorrhagic infarct are swelling and hemorrhage. In the lungs, there are physical signs of consolidation, with hæmoptysis, cough, and dyspnœa; in the kidneys, pain and hæmaturia; in the spleen, pain and at times enlargement; in the retina, blindness with ophthalmoscopic changes; in the intestine, pain and hemorrhage with sloughing of mucous membrane. *Infective emboli* cause abscesses. *Capillary embolism* is seen in the skin and mucous membranes in many infective diseases, notably ulcerative endocarditis. *Fat-embolism* occurs in the pulmonary capillaries, and is due to fat-globules which sometimes enter the circulation in pregnant women, or in patients with bone disease, as osteomyelitis, or fractures. The symptoms are those of intense dyspnœa. It may cause sudden death. *Air-embolism*. Air may enter wounds of the veins of the neck. It accumulates in the heart, and as the ventricle cannot contract on it the blood is not propelled. Death takes place with the symptoms of heart-clot, the heart being in asystole.

Hemorrhage. Hemorrhage may be arterial, venous, or capillary. It may occur because the blood soaks through the walls, by diapedesis; or it may occur from rupture, or rhexis. Hemorrhage by diapedesis takes place in venous engorgement, stasis, or inflammation. It is the small passive hemorrhage of congestion, as in pulmonary congestion from heart disease; it is venous or capillary; the blood is dark. Hemorrhage by rupture is arterial, venous, or capillary. If the artery

ruptures, it has been torn by violence, destroyed by ulceration or suppuration, or it is the seat of endarterial change. Veins are also diseased, or their walls destroyed, before rupture takes place. Rupture of capillaries occurs from violence or great internal pressure. In death from suffocation the capillaries are the seat of hemorrhage because of the increased venous pressure. Such capillary hemorrhage occurs in typhus, hemorrhagic smallpox, and scarlatina. The state of the blood is sometimes the cause of hemorrhage, as in scurvy, purpura, and other conditions. Hæmophilia is a peculiar hereditary affection possibly due to the state of the blood, more likely, however, due to the condition of the bloodvessels.

The special forms of hemorrhage and their symptoms, etiology, and diagnosis will be considered in the sections to which the names in the following list point:

Bleeding from the nose—*epistaxis.*
Vomiting of blood—*hæmatemesis.*
Bleeding from the lungs—*hæmoptysis.*
Blood passed with the urine—*hæmaturia.*
Blood passed from the uterus—*menorrhagia* or *metrorrhagia.*

There is also intestinal hemorrhage—*melæna.*

Hemorrhages underneath the skin are known as *petechiæ* if small, and *ecchymoses* or *suffusions* if large.

Hemorrhage into internal organs receives its name from the organ affected, and is known as a *parenchymatous hemorrhage. Apoplexy* is applied to hemorrhage into the substances of organs, particularly if it occurs suddenly and is localized—as pulmonary apoplexy, cerebral apoplexy, spinal apoplexy. Long usage has associated the term with hemorrhage into the brain, so that it is applied to that form alone by most writers. *Hæmatoma,* or blood-tumor, is a collection of blood that has coagulated in a cavity, organ, or tissue. (See Ear.)

The *symptoms* of hemorrhage vary in degree, depending upon the amount of blood which escapes from the vessel, and whether the hemorrhage is external or internal. By external hemorrhage we mean one which is accompanied by a discharge of blood visible to the bystander. An internal or concealed hemorrhage is not apparent by any outward sign of blood.

The symptoms by which *external* hemorrhage is recognized need not be detailed. The show of blood in situations or at times other than normal is sufficient. It must be remembered that arterial blood is bright red, venous blood dark. It must also be remembered that the character of the blood coming from internal organs is modified by the secretion of the affected organ. Thus the blood from the stomach is coagulated and black, like coffee-grounds; blood from the intestine, tarry. The general symptoms of the various degrees of external hemorrhage are similar to the symptoms of internal hemorrhage, which will be described later. Both vary with the rapidity of the flow of blood. If the bleeding is slow, large quantities may be lost and more or less profound anæmia result. It is often more difficult to determine the source of hemorrhage. The mode of recognition of the anatomical varieties of hemorrhage will be discussed under the respective systems

which are the seat of the bleeding. Hemorrhage may take place in a cavity, as the stomach, bowels, or bladder, and after the blood has undergone changes it may cause symptoms of, and be discharged as, a foreign body.

Although *internal* hemorrhage presents vivid phenomena, they may not be characteristic, and its recognition is often impossible without some knowledge of the history of the case. The symptoms are complex. First, we have pain, a symptom due to rupture of a vessel or to the filling of a tissue with blood. In the beginning the pain is sharp, severe, and of itself may cause shock. In the second place, the symptoms due to loss of blood arise. After pain, sudden prostration ensues; pallor spreads rapidly; the extremities become pallid and cold; a cold sweat breaks out on the forehead; the features become pinched and shrunken; the pulse becomes weak and rapid, and later thready, or disappears altogether at the wrist; the carotids pulsate; the heart throbs violently and a diffuse impulse is seen, at first vigorous, soon like a slap against the chest-wall, and then it fades away completely. On examination of the heart and vessels so-called anæmic murmurs are heard. The patient is restless, and sighs and yawns frequently. The respiration becomes slow and shallow. Nausea and sometimes vomiting may occur. He may faint but once or repeatedly, to be restored again and again, or the syncope may terminate in death. In the intervals between the syncopal attacks the mind is clear. If, however, profound shock is associated with the hemorrhage, there is dulness or stupor; the intellect is dazed; otherwise delirium and agitation may be present. When the hemorrhage is profuse convulsions may take place. The temperature of the body falls. If the patient has fever at the time, the temperature suddenly falls to or below normal. We have, therefore, the following conditions in hemorrhage: syncope, shock, and collapse. They may all be present in the same subject, or one or two may be absent. The same symptoms may, however, occur from other causes, which must be excluded. Sometimes the shock may be due to the same cause as the hemorrhage. The causes of shock are so evident that they serve to distinguish it from the collapse of hemorrhage. They are injury, anæsthesia, railway accidents, surgical operations, perforative peritonitis, strangulated hernia, intestinal obstruction, profound mental impression, and pain.

Shock from hemorrhage must be distinguished from concussion. In the latter the intellectual disturbance occurs at once, and is more marked than the circulatory symptoms. The absence of the usual phenomena of hemorrhage serves to distinguish syncope due to concussion from that due to the many well-known causes of fainting.

There are many forms of internal hemorrhage sufficiently grave to have a probably fatal result, or at least to create alarming symptoms. In the chest, diseases of the lungs or the aorta cause hemorrhage. In concealed pulmonary hemorrhage the blood accumulates in a large phthisical cavity. When the aorta or an aneurism ruptures the blood may enter the mediastinum or the pleura. Under these circumstances a knowledge of the previous history is essential. Careful examination of the lungs or of the heart or bloodvessels must be made in a case

which presents the above-mentioned symptoms of internal hemorrhage. Internal concealed hemorrhage into organs or cavities of the abdomen occurs in gastric, duodenal, or intestinal ulceration ; in aneurism or in ulceration of large vessels, from septic inflammation around them. It must not be forgotten that alarming or fatal internal concealed hemorrhage may be due to hæmophilia or purpura.

II. Disturbances of Nutrition.

Hypertrophy and Atrophy. (See the Size, Chapter VI., and Muscles.)

Inflammation. Inflammation, a process largely attended with vascular alteration, but also with disturbance of nutrition. It may be acute or chronic. It is due to injury, mechanical, physical, chemical, or vital. The invasion of micro-organisms or the irritation of their products is the most frequent cause in cases that come within the province of the physician. The symptoms are modified by the structure affected and by the cause of the inflammation. The intensity and the character also modify them. The classical symptoms —*pain, heat, redness*, and *swelling*—are indicative of the tissue-process. In addition we have *exudation* and *alteration of function*. *Pain* varies in degree with the sensibility of the part. It is increased by pressure or movement, and by the functional activity of the affected organ. *Heat* is detected by the hand or surface-thermometer. It may be described by the patient, in abscess within the peritoneum, or pyosalpinx, as a ball of fire. The surface-temperature over an inflamed lung or pleura is higher than over the healthy side. *Redness* can only be observed in parts open to inspection, as the nasal, oral, faucial, and other cavities. *Swelling* is observed with the redness ; it is shown by enlargement of the affected organ, if the latter can be measured by palpation or percussion. *Exudation* takes place from mucous surfaces, into serous cavities, into the connective or any affected tissue, or into tubes or channels (heart and bloodvessels, lymphatics, etc.). The symptoms are : characteristic discharges from mucous surfaces ; pressure and physical signs from accumulation in cavities ; symptoms of the obstruction of channels. Grave pressure-symptoms arise when the exudation presses upon the nerves, nerve-centres, or nerve-tracts (brain cord, peripheral nerves). The pressure-symptoms are often more pronounced than the inflammatory in simple or tuberculous meningitis. *Alteration of function :* The symptoms cannot be detailed here ; each organ and structure must be referred to. The function may be stimulated at first, but is soon perverted, or suppressed.

General Symptoms. Fever is the general expression of the local process. It may be primary from reflex irritation of afferent nerves which influence the heat-centre and disturb the thermotaxic mechanism. It may be secondary, the products of inflammation (pus, toxins, etc.) irritating the centres. The degree depends upon the cause. Active inflammation may not be attended by fever.[1]

Suppuration. The character of the fever indicates the variety of

[1] Musser : "Abscess of Liver," Univ. Med. Magazine, 1892.

the inflammatory process. In most inflammations the fever is continuous. When there is suppuration, however, it becomes intermittent or remittent. The presence of suppuration is also made known by *hectic*, in which the fever is attended by chills and sweats. The appetite is lost or impaired. There is also leucocytosis. The urine contains a large amount of indican. In obscure inflammations about the peritoneum the indicanuria points to a suppuration. While fever-symptoms in inflammation are similar, save in degree and in the peculiar type of the temperature-range—intermittent, remittent, or continuous—septic inflammations are attended early by cerebral symptoms, prostration, and the typhoid state. (See Fever, pages 218 and 224.)

As a corollary, when fever is present, local inflammation must be sought for. *Chronic inflammations* may only give rise to altered function and cause exudation (swelling, effusion, etc.).

Inflammation of Various Structures. The symptoms vary according to the anatomical and physiological peculiarities of the structure.

Inflammation of *mucous membranes*. *Pain* is not excessive; *heat* is complained of (rectum); *redness* is marked and varies with the intensity from bright to dark red; *swelling* is always present. In narrow channels, as the nose, or the gall-ducts, it causes occlusion. The *exudation* is at first mucous, then mucopurulent, and then purulent. Before exudation there is a stage of dryness. The microscopical appearance of the exudate varies with the anatomical character of the membrane affected. Its peculiar epithelium is always present, also micrococci, pus, red cells; from the lungs or liver, special crystals. The functions are impaired. Fever is usually not very high and is continuous. The causes are direct local irritants or congestions from external impressions (cold ?).

Inflammation of *serous membranes*. *Pain* is extreme and may cause collapse. *Heat*, *swelling*, and *redness* cannot be estimated. The surface-temperature rises. *Exudation* occurs after a brief dry stage. The cavities—pleura, pericardium, peritoneum, joints, cerebro-spinal canal—are filled, causing mechanical symptoms and physical signs. Fever is excessive in some forms. Function is impaired or abolished. General symptoms are more pronounced. Shock or collapse is common in peritonitis. The affections are always secondary to a general process (rheumatism), to infection, to disease of neighboring structures, or to Bright's disease, diabetes, cancer, scurvy, or other diathetic condition.

Inflammation of *muscles* (rare), of *connective tissue*, and of *glands* is characterized by symptoms common to the morbid process, with alteration of function.

Inflammation of *bone* and *periosteum* presents the same group of symptoms. The pain may be intense or of a dull, aching, or boring character.

Inflammation of the *heart* and *vessels* is also attended by the cardinal symptoms. When the central organ is the seat of the disease pain is not common, but in the arteries or veins it is of frequent occurrence. The striking symptom, however, is the *obstruction* to the channels. It is characteristically seen in phlebitis, as of the femoral vein. Œdema of the leg, and cyanosis, reveal the obstruction. In the heart

the acute process or the results of the process give rise to all the symptoms of obstructive heart disease.

Inflammations of the *nerves*, the *spinal cord*, and the *brain* are followed more strikingly by pressure-symptoms and by the symptoms of degenerations secondary to the inflammatory process. Hence, while pain and tenderness are present in the exposed nerves, increased irritability, then abeyance, perversion, or abolition of function are the principal signs of inflammation of these regions.

Inflammation of *internal organs*, *lung*, *liver*, *kidneys*, and *pancreas*, is made known by pain (minimum amount) and swelling (enlargement of liver), and by change in the function, indicated by modifications of the respective secretions as well as by functional and physiological symptoms.

Local Death, Necrosis, and Gangrene. If nutrition is not complete, the life of the cell is endangered. This process is known as necrosis or gangrene. The nutrition is annulled: 1. By stoppage of the circulation. 2. By the direct action of an irritant which destroys the cells. 3. By abnormal temperature. A combination of the three causes quickly produces gangrene. Stoppage of the circulation may be due to an embolus or thrombus, or to stagnation by pressure, or to capillary stasis alone. Sloughing and "bed-sores" ensue in the latter instance; gangrenous eschars in the former. The cells are destroyed by corrosives and caustics, by heat and cold, by bacteria. Where decomposition takes place, as in retained and infiltrating urine, cell-destruction and sloughing ensue. All pathogenic bacteria cause necrosis to a greater or less degree. Frost-bite and burn illustrate the destructive power of abnormal temperature.

Nerve-lesions, trophic disorders, produce necrosis. We have, allied to bed-sores and known as decubitus, a form of necrosis in spinal-cord diseases. The sloughing is extensive and rapid. Trophic disorders cause paralytic hyperæmia, and hence necrosis.

It must not be forgotten that debility, cachexia, and feeble circulation play a great part in assisting the local changes.

Gangrene of internal structures concerns us. This form is nearly always due to stoppage of the circulation. It is seen in constriction of the intestine, from hernia, or obstruction. It occurs in phthisis from thrombi. Clinically, we see it frequently in diabetes. The lung, the brain, the intestines, are most frequently affected.

The symptoms of necrosis or gangrene are modified by the tissue involved, the function interfered with. If external, the decomposing structures emit a foul odor, there is rapid prostration and development of the typhoid state. Fever ensues from intoxication by decomposing substances—sapræmia. Often the symptoms are latent. A man aged sixty, in my ward, was about all the time. He died suddenly of pulmonary hemorrhage, the result of gangrenous ulceration of a large vessel; at the autopsy gangrene of the lung was found. The only symptom was the characteristic odor. In the course of inflammatory processes the onset of gangrene is frequently attended by the cessation of pain, the peculiar odor when it communicates with the exterior, and the development of exhaustion and the typhoid state. The character

of the discharge points to gangrene. When the lungs are affected the expectoration is like prune-juice; when the bowels, the discharge is dark and putrid.

Fever is a morbid process, with the cause and symptomatology of which the student must be familiar. It has been fully treated in previous chapters. (See Fever.)

The Degenerations. The symptomatology varies with the form of degeneration and the organs affected. The prostration of the general economy is due to the same cause as the degenerations themselves.

Albuminous degeneration occurs in fever, and causes the weak heart and defective gland action. The weak heart of the convalescent period in diphtheria and other infective diseases is well known.

Fatty Degeneration and Infiltration. In *fatty degeneration* there is cell-destruction. The brain, the heart, the kidneys in Bright's disease, the liver, all undergo degeneration. It may be due to phosphorus-poisoning or to snake-bite. It is seen in acute yellow atrophy of the liver. It is caused by other toxic agents. *Fatty infiltration or lipomatosis* is seen in the "fat" heart of brewers, the enlarged liver, the excess of fat in the abdomen, etc. The affected organs are enlarged, but they are functionally weak. Fatty infiltration of organs is recognized by its etiological associations. In alcoholic subjects of sedentary habits, in subjects who eat an excess of fatty foods, in overfed and pampered children, and in tuberculosis it is commonly seen. In fatty infiltration the cells are not destroyed. If with the above conditions the liver is enlarged or the heart weak, or both, we may expect to find fatty infiltration. There is enlargement of the affected organ, which is painless, smooth, not usually soft on palpation. The condition occurs at any age, but usually in later life. Emaciation may not be present. Lithæmia is common in fatty infiltration.

Amyloid Degeneration. This is rarely confined to one organ of the body. The causes are syphilis, malaria, tuberculosis, and prolonged suppuration. The *liver* and *spleen* are *enlarged*, hard, smooth, and painless. There are great *pallor*, and œdema of the feet and face. There is *anæmia*, but no fever. The kidneys are affected, hence *polyuria* and low specific gravity of the urine; a few casts are found. The bowels are likely to be loose because the process has involved the intestine. It occurs at any age. The diagnosis rests on the presence of a cause, the painless enlargement of organs, the pallor, and the polyuria.

Fibroid Degeneration. This is not so much a degeneration as an overgrowth of connective tissue with coincident primary or secondary atrophy of the parenchyma. The function of the organ is impaired or abolished. Increase of connective tissue in the nerve-structures is known as sclerosis, in the liver or kidney as cirrhosis. In the artery it leads to the changes known as endarteritis. Whatever the pathology may be, whether atrophy of cell-elements of the affected structure be primary or secondary, the condition is productive of serious, even grave consequences. It is part of the senile process. It leads to the manifold symptoms of endarteritis; it is the cause of many nervous affections which will be discussed in their proper sections.

The varied phases of so-called interstitial nephritis are due to the fibroid changes primarily in the kidneys, and secondarily in the arterial system. In the lungs it attends emphysema, or may even be productive of that condition. The fibriod heart is another manifestation of the same process. The tubes and channels are closed by the same process as in fibrous stricture of the duodenum. Wherever situated its development means gradual abolition of function.

Mucous Degeneration. This form of degeneration is seen in myxœdema. The albuminous intercellular substance is replaced in the connective tissue by mucin.

Pigmentary, calcareous, and colloid degenerations are local morbid processes without other symptoms than those of the primary affection.

III. Anomalies of Growth.

Tumors. Tumors, other than cancer or sarcoma, produce only mechanical symptoms, and must be considered in their special section. The mechanical symptoms are due: 1. To the tumor (foreign body). 2. To obstruction of any channel in near relation.

New Growths. They cause local symptoms. This is most striking in structures which must necessarily be destroyed as the growth increases in size, as in the brain or spinal cord, or where tubes or channels are closed, as in cancer of the stomach or œsophagus. Local symptoms may precede the general symptoms; on the other hand, general symptoms may arise for which no local cause can be assigned. The local symptoms of cancer are variable and depend upon the anatomical nature and physiological offices of the organ affected, and upon its anatomical relation to surrounding organs. This class of symptoms will be referred to in the section on special diagnosis. Suffice it to say they cause gradual abolition of the function of the organ, or closure of the channels in connection with it, as the intestinal canal, the pharynx, or the hepatic ducts. Cancer and sarcoma are accountable for a group of symptoms to which the term *cachexia* has been applied. In addidition, a few symptoms belong to the cancerous process wherever situated. They may or may not all be present; in the large majority of cases one or more are wanting; they should always be sought for in order to confirm a diagnosis of cancer. These symptoms are:

1. *Pain*, recognized by peculiar characteristics in most cases: (*a*) It is sharp and lancinating; (*b*) it is paroxysmal; (*c*) it is increased by irritation, as food when the stomach is affected; (*d*) it is increased by functional activity, as speaking or swallowing in carcinoma of the larynx or pharynx; (*e*) at the outlet of canals, as the bladder or rectum, it gives rise to tenesmus.

2. *Hemorrhage.* If the malignant mass is in communication with the exterior, the blood may be discharged *per vias naturales.* In malignant disease of the upper air-passages or the lungs hemorrhage is likely to occur. It is common in gastric carcinoma as well as in uterine cancer. If the organs do not communicate with the exterior, and the lesion gives rise to exudations or transudations, the latter are frequently bloody, as in carcinoma of the pleura or peritoneum.

3. *Abnormal Discharge.* This occurs especially in cancer of the

hollow viscera and of the canal-structures. The discharge is the result of inflammation, suppuration, and necrosis, and particularly microbic inflammation. It is recognized by its more or less *bloody character* and by its *odor*, which is peculiar. It is most offensive and penetrating, and, particularly in uterine cancer, is almost pathognomonic. Even the utmost cleanliness will not obviate it.

4. *Tumor.* It may be readily detected or elude all search. Some swelling is certainly present. It is discovered by external examination, by the objective physical signs of enlargement or change of contour of the affected organ.

5. *Foreign Body.* The growth gives rise to symptoms similar to those present when a foreign body is fixed in any portion of the hollow viscera, as the respiratory tract, the gastro-intestinal, including the hepatic and the genito-urinary tract. *a.* Through reflex influence an attempt is made to remove it, hence cough, vomiting, diarrhœa with tenesmus, repeated and painful micturition with tenesmus, etc., the particular symptoms varying with the organ affected. *b.* Obstruction of the channels, with all the accompanying symptoms, depending upon the location of the growth.

6. *Temperature.* A morbid process is often recognized by its negative symptoms, if the term may be used. Thus, fever is absent or the temperature is even subnormal in carcinoma.

7. *The Cancerous Cachexia.* Wherever situated the disease is sooner or later attended by extreme general symptoms which are, in a measure, striking. It is to be admitted that cases of carcinoma often occur without marked cachexia. *a.* One symptom may always be looked for; it is *emaciation.* It may be rapid or gradual and extend over one or two years; toward the end it is always rapid. Ultimately, if the patient does not succumb to other conditions, it presents an extreme picture. The eyes are sunken, all normal accumulations of fat disappear. The fat in the rectal fossæ disappears, causing deep depression of the rectum. The abdomen is retracted. The appearances are most striking in cancer of the œsophagus. *b. Pallor* (see Color); this may be present. *c. Anæmia,* with breathlessness, palpitation, vertigo. *d. Exhaustion.* This with accompanying emaciation is progressive, and may be the first symptom. Progressive weakness is often seen without fever or local disorder to account for it. Toward the end it becomes so extreme as to forbid exertion. *e. Malnutrition.* Evidences of malnutrition appear; the skin is hard and dry; its elasticity is impaired and it becomes the field for parasitic invasion. Tinea and other parasites may flourish. Bacteria invade the susceptible areas, and boils make their appearance. The secretions are perverted. In the mouth ulcers develop; the fungi of this situation (the throat, etc.) become more active; the gums are inflamed. In the later stages the "typhoid state" (see Fever) may ensue. If the gastro-intestinal tract is invaded, symptoms of acute intoxication may arise.

8. *Metastasis.* We are often aided by the occurrence of this event, particularly by involvement of the glands. In gastric carcinoma secondary hepatic disease or enlarged glands above the left clavicle are found; in rectal carcinoma, secondary hepatic cancer. In many

instances the presence of cancer is revealed by the metastasis, even when the primary growth cannot be recognized.

The *diagnosis* rests upon the above conditions. In obscure cases the age, the sex, the associate pathological conditions, the duration of the disease become important factors in the diagnosis. Cancer usually occurs after forty, or, some authorities say, after fifty years of age. The female sex is most frequently affected. It may be associated with a history of previous lesion or irritation, as ulcer in vaginal, gastric, or rectal cancer; the irritation of teeth or a pipe in labial and lingual cancer; of gallstone in cancer of the bile-ducts; of renal or visceral calculus in disease in that situation. A disease of grave and malignant character, the duration of which is over eighteen months or two years, is not, in all probability, cancer.

Morbid Processes in Tubes or Channels. *The effects produced by obstructions.*

When tubes or channels are the seat of disease symptoms arise apart from the special morbid process, which are due to obstruction and are common to all tubes or channels. The symptoms of obstruction of the bloodvessels and lymph-channels—cyanosis, œdema, gangrene (thrombosis and embolism)—have been described. But in addition we have hypertrophy, a secondary condition, not referred to above, which, nevertheless, follows obstruction of any channel. In the cases of vascular obstruction the hypertrophy is seen in the heart and the arteries. (See Diseases of the Heart.)

In obstruction, therefore, of tubes or channels we have to a greater or less extent (1) hypertrophy behind obstruction; (2) diminution of the normal flow of fluid and consequent accumulation of material which normally passes through the channels; (3) atrophy and cessation of functional activity beyond the point of obstruction; (4) dilatation of the primary hypertrophy; (5) degeneration, ulceration, low-grade inflammation (bacterial), secondary rupture of the affected viscera. The morbid anatomist can readily point out the examples of the morbid changes sequential to obstruction. Thus in cancer of the œsophagus there are hypertrophy of the muscular coats, regurgitation of food, atrophy of the stomach, dilatation with accumulation of food, secretions from the glands of the œsophageal mucous membrane, secondary ulceration, rupture into the lungs, with gangrene or pneumonia. In obstruction at the pylorus there are (1) hypertrophy; (2) accumulation; (3) intestinal atrophy; (4) dilatation of the stomach, with its train of symptoms. In obstruction of the biliary channels, or the bladder, or ureters, the same secondary conditions arise *plus* obstruction to the flow of bile or urine. Secondary symptoms arise from accumulation of the non-escaping fluids. Subjective symptoms, it may be said, are not marked; there are pain and difficulty in the performance of the usual functions. It need scarcely be said that the obstruction sometimes gives rise to symptoms which are due to the abnormal obstructing material which acts as a foreign body. The symptoms are reflex and depend entirely upon the seat of the foreign body.

The *causes* of obstruction in whatsoever channel situated are, first, pressure from disease outside (growths, hernia); second, disease of the walls, with contraction; third, occlusion by a foreign body, as gall-stone, renal calculus, worms, or other material according to the channel obstructed. The symptoms are most marked when the obstruction is due to disease outside the walls or to obstruction by occlusion within the walls.

In all cases of obstruction, nasal, faucial, laryngeal, bronchial, œsophageal, gastro-intestinal, biliary, renal, or pancreatic, look for the symptoms of the secondary morbid change. Each form of obstruction will be specially considered elsewhere. (See Special Diagnosis.)

The Bloodvessels. *Blood-pressure.* It must not be forgotten that the bloodvessels are in a measure distinct from other tubes, although subject to the same laws, physiological and pathological. They contain fluids, and have a continuous function by which the fluids are propelled. They are subject to the laws that govern the flow of fluids under all circumstances in nature. Any derangement or disease will effect changes which are explainable by hydrostatic or hydrodynamic laws. Fluids within vessels exert pressure. Pressure produced by weight of the fluid is known as the hydrostatic pressure; that produced by the flow is known as the hydrodynamic pressure. Pressure can be gauged by proper instruments. In the case of fluid in the bloodvessels it is called the blood-pressure. The blood-pressure is estimated at the pulse by the educated finger and by the sphygmograph. A certain definite pressure is always present in health. It is subject to slight fluctuations, but tracings with a sphygmograph follow a definite course. In the description of the pulse, modifications of blood-pressure will be given in detail; it is sufficient here to say a few words regarding hydrostatic and hydrodynamic pressure.

Hydrostatic pressure is modified by the weight of the fluid. It is of pathological importance in the veins only, and especially in those of the lower limbs. When the pressure is increased the increased weight of the blood-column causes increased bulk and over-distention, as in varicose veins, unless the support to the blood-column is increased. Inflammations of the lower limbs are attended by venous accumulation and followed by ulceration. For this reason dropsies arise more readily in these portions. The common occurrence of gout in the feet may be due to slow circulation.

Hydrodynamic pressure is variable. Its changes indicate increase or diminution of blood-pressure. The bloodvessels are resisting elastic tubes; the resistance is always equal to the pressure within, hence blood-pressure and arterial tension are equivalent terms. We speak of increased or diminished pressure, or correspondingly of high or low tension. Now, the hydrodynamic or blood-pressure depends upon: (1) Variations in the volume of blood; (2) variations in the capacity of the vascular system; (3) facility of the capillary circulation; (4) the force of the heart. The tension of the artery depends upon the same conditions.

1. Variations in the volume of the blood. *a.* Volume increased. Causes: absorption of fluid after meals or drinking to excess. Result: increased blood-pressure and increased tension. Controlled in health by action of the vasomotors relaxing the vessels, and by enlargement of the veins. *b.* Volume diminished. Cause: hemorrhage, serous purging. Result: diminished blood-pressure, lowered tension. Controlled in health by contraction of arteries through vasomotor nerves. In hemorrhage the loss of blood produces anæmia. The latter is a stimulant to the vasomotor centre in the medulla, and produces contraction of peripheral arteries and high tension.

2. Variations in the capacity of the vessels. *a.* Diminution of the capacity of the blood-channels (volume of blood not lessened). Cause: cutting off of a vascular area by ligation or obstruction, by narrowing the calibre of the wall, as in arterial spasm or endarteritis, by disease of the kidneys, contracting the lessening channels in the aortic circuit, or disease of the aorta, causing obstruction to the outflow of blood. Result: increased pressure, high tension. Controlled by normal regulating vasomotor apparatus, or by diminution of the volume of blood. *b.* Increase of capacity of blood-channels. Cause: relaxation of muscular coats of vessels. Result: diminished blood-pressure, lowered arterial tension. Controlled by contraction of vessels or increase in amount of blood. In shock, the vasomotor sympathetic system of the splanchnic arteries is so disturbed that the arteries are dilated and all the blood is sent into the abdominal vessels (fall of pressure).

Mode of action of the vasomotor apparatus. Centres in the medulla, in the spinal cord, and locally in the sympathetic ganglia of different parts, control the vasomotor nerves, which influence hydrodynamic pressure. 1. If the centres are stimulated, tonic contraction of the vessels is produced. This may be general or local. Increased pressure or heightened tension is the result. It may be reflex from the periphery, or due to some state of the blood. 2. If the centres are paralyzed, or inhibited, or cut off from the arteries, the latter become relaxed (dilated). The pressure is lowered, the tension is less. *Shock*, pain, certain drugs, reflexes (probably) produce inhibition.

3. Facility of capillary circulation. Obstruction to outflow of blood from capillaries into the veins increases blood-pressure. Cause: the same as when arteries contract. Result: increased blood-pressure, high tension. Regulated in the same manner as arteries. Relaxed capillaries produce opposite conditions.

4. The force of the heart. *a.* Heart's action (left ventricle) increased. Cause: hypertrophy, palpitation. Hence the greater force of blood-impact, greater resistance by arteries. The tonic resistance narrows the calibre of the vessels. Result: increased pressure, higher tension. *b.* Heart's action weakened. Hence, less force of blood, less resistance. Result: lessened pressure, low tension.

The *recognition* of variations in tension. (See Pulse.)

1. High arterial pressure or tension. By (*a*) incompressibility and tension of the arteries; (*b*) accentuation of the aortic second sound; (*c*) prolongation of the left ventricle first sound; (*d*) increased flow of urine, pale and watery; (*e*) characteristic pulse-tracing by sphygmo-

graph. If the high tension is permanent, (*f*) hypertrophy of the heart; (*g*) atheroma, more or less.

2. Low arterial pressure or tension. By (*a*) soft, compressible, often dicrotic pulse; (*b*) enfeebled sounds, aortic second and left ventricle; (*c*) scanty, high-colored urine; (*d*) special pulse-tracing. If permanent, stases, congestions, cyanosis, with general weakness and impaired nutrition.

PART II.

SPECIAL DIAGNOSIS.

CHAPTER I.

THE NOSE AND LARYNX.

The Nose.

THE symptoms of disease of the nose result from disturbance of the function or alteration of the structure of the organ and the morbid process. *Physiological symptoms:* Impairment of the sense of smell, anæmia, and symptoms of obstruction may occur. *Obstruction* causes retention of secretions. These secretions are exposed to infection. Putrefaction and fermentation set in and give rise to offensive *odors*. More serious is the effect of the obstruction on the rest of the respiratory tract. The patient becomes a *mouth-breather*. The appearance of the face is altered; the voice changes, snoring is common, mastication is interfered with, and there is a diminution in the amount of air passing to the lungs. As a result a vacuum is created which is compensated for by external pressure. In children the result is marked deformity of the chest, leading to the development of the "pigeon" or "chicken breast." (See the Lungs, Chapter II., Part II.) The general symptoms attending mouth-breathing will be referred to again.

Symptoms due to the Anatomical Structure. The nose is an open space or a series of air-spaces lined with *mucous membrane*. The mucous membrane is the frequent seat of infectious inflammation, as in hay fever, influenza, and measles. Most of the nasal symptoms are due to disease of the mucous membrane. The membrane is subject to affections that are common to all mucous membranes, and the subjective and objective symptoms are similar to those that arise in other organs, modified by the function and anatomical arrangement.

The abundance of bloodvessels and glands is the cause of one of the symptoms—namely, the *discharge*. Moreover, the difficulty of removing the discharge from the various cavities in the nose in which they are pent up leads to putrefaction and *odor*. Because the air is constantly passing over the parts, discharges are very liable to become dry, and hence *crusts* and *scabs* form. Again, the *vascularity* of the structures of the nose is the cause of development of symptoms. The bloodvessels are richly supplied with nerves, which cause them to contract or dilate, on comparatively slight provocation, by reflex action. Chilliness of the body, or of local areas of the body, chilling of the extremi-

ties, and other peripheral impressions, are followed by congestion of the nasal mucous membrane, which may go on to inflammation. The vascularity predisposes to hemorrhage.

The nose is richly supplied with *nerves* (in addition to the olfactory nerve), which are susceptible to various irritations or impressions—impressions made by the air laden with unusual material, as fumes of a chemical nature, emanations from animals, or plants, and certain substances not yet isolated, which are decidedly irritating. There is often local irritation from polyps and adenoid growths, and foreign bodies, or enlarged bone. The nerves are connected by a mechanism directly with the centres in the medulla, with particularly the pneumogastric centre. The effect of peripheral nasal irritation may be felt reflexly in the area of distribution of that nerve ; hence an unpleasant odor may bring on sudden nausea or vomiting. But of more striking and frequent pathological significance is the occurrence of asthma, or sudden dyspnœa, from reflex excitation of the pulmonary division of the pneumogastric nerve.

Morbid processes in the nose are symptomatic of some general affections. The occurrence of asthma, or of deformity of the chest and general ill-development, has been spoken of. Acute inflammations are significant of the exanthematous diseases, particularly measles. An acute inflammation (as pointed out by Meigs), with great obstruction of the nares and an abundant, puriform discharge, is a complication or symptom of Bright's disease that may portend the onset of uræmia. Chronic inflammations may be due to syphilis or other chronic infection.

The Data Obtained by Inquiry.

Of the data obtained by inquiry, that belonging to the social history, the family history, and the history of previous diseases yield but little information of diagnostic value. It is true the acute inflammations secondary to measles and other exanthemata occur at an early age, while the chronic attacks occur late in life, as do also tumors, except adenoid. Foreign bodies are more likely to be found in children and the feeble-minded. Those occupations which are in-doors, in overheated apartments, and among noxious vapors predispose to catarrhs. In the family history we must look for gout, rheumatism, syphilis, and affections which lead to osseous changes. More marked than all is the influence of syphilis. A chief predisposing factor in the production of nasal disease is the morphological arrangement of the parts, which may be congenital, or the result of early infantile disease. Thus, when congenital, the high palatal arch, etc., is looked upon as the stigmata of degeneration.

On inquiry of the history of previous diseases, we look for syphilis, the exanthemata of early life, the occurrence of gout or rheumatism, and of those gastrohepatic and nutritional disorders which lead to catarrhs.

The Subjective Symptoms. General. They are often accompanied by extreme distress, but do not lead to a fatal termination. The general subjective symptoms are like those of inflammation of other mucous membranes.

1. Lassitude occurs when there is fever. It is a frequent precursor of rhinitis, and is pronounced in croupous and diphtheritic rhinitis; extreme prostration may attend the latter.

2. Chilliness following the lassitude, or *rigor*, may occur in the same class of cases. If distinct rigors occur, an abscess in one of the cavities may be suspected, if the subjective and objective symptoms point to it; or glanders may be present.

3. Fever. This occurs in the inflammations; it is never marked, and is not of diagnostic significance. It is most severe in *glanders*. It is then attended by general symptoms of rigor, with pain in the trunk and limbs. In the first twenty-four hours there may be nausea and vomiting. Locally, a small pimple is seen which is quite painful. A yellowish sanious discharge oozes from the nostrils. Hard pustules appear about the nose and other parts of the body. (See Infectious Diseases.) It is of low type in *diphtheria*, and of *hectic* character when there is *abscess*. High fever associated with inflammations of the nose points to influenza or one of the exanthemata as the primary cause of the rhinitis. Foreign bodies in the nose may cause fever. Emaciation occurs with malignant growths.

Local. Pain, varying in degree, occurs in all acute affections of the nose. Its seat and character are of some diagnostic significance. Smarting or burning pain at the root of the nose accompanies acute *rhinitis* and attends *post-nasal catarrh*. The pain is diffuse and indefinite in *dry catarrh* and in *diphtheria*. The most severe pain occurs when *foreign bodies* are present in the nose and in cases of *glanders* and *primary syphilis*. *Foreign bodies* of a vegetable nature by swelling and germinating induce pain, which increases gradually in intensity.

In tropical regions parasites may be found in the nostrils. They are the larvæ of the *lucilia hominivora*. It is said that the pain is so severe at the root of the nose, extending backward, as to cause maniacal delirium. Sleeplessness is marked, and there may be extensive destruction of the bones and skin. There is a fetid, sanious discharge.

Pain Over the Frontal Sinus. The pain of an inflamed frontal sinus is more severe than the pain of inflamed nostrils. It is sometimes intense and agonizing. Pain may be located in the cheek from inflammation or tumors of the antrum. In disease of the nose, if the pain radiates to the *ear*, the Eustachian tubes are probably involved.

Headache is frequently caused by nasal disease of all forms. (See Chapter IV., Part I.)

Disturbance of the Sense of Smell. (See the Nerves.) Anosmia and Parosmia. *Loss of smell*, or *anosmia*, occurs to a moderate degree in all the inflammatory and obstructive diseases of the nose. The intensity depends upon the degree of change in the mucous membrane. It may also be due to disease of the nerves or the olfactory centre in the brain. *Parosmia* is the perception of abnormal odors, and may be a neurosis or psychical difficulty entirely, and hence purely subjective, or there may be inability to distinguish an odor when presented to the nostril. All odors may appear the same, or agreeable odors may seem to the patient very disagreeable. In addition, the patient

may complain of the perception of an odor in connection with the nasal disease with which he is affected. Parosmia is due to an involvement of the olfactory nerves.

A sense of *dryness* is a symptom of which the patient frequently complains, particularly in the early stages of acute rhinitis and throughout the entire course of dry catarrh, or atrophic rhinitis.

Obstruction or Stenosis. This sometimes causes the greatest discomfort to the patient. There may be simply a sense of stuffiness and fulness in the nasal and frontal region, or complete obstruction, causing difficulty in breathing. In infants it prevents nursing, and should always suggest inherited syphilis. It occurs in all the obstructive diseases of the nose and nasopharynx, as acute rhinitis, chronic inflammation (except the atrophic form), hyperæmia, the hypertrophies, polyps, tumors, deviations of the septum, foreign bodies, and adenoid vegetations.

Deafness is present when the Eustachian tubes are invaded or obstructed from inflammation or stenosis. When associated with anosmia it may be of central origin. *Tinnitus aurium* frequently accompanies the deafness.

Cough. The discharge may pass into the pharynx and the larynx and cause cough. (See Chapter on Cough.) It occurs, therefore, in the catarrhs and obstructive diseases, and is not diagnostic of any nasal condition. When the nostrils are too wide, as in atrophic rhinitis, cough may occur because irritating particles are admitted through the widened aperture. A so-called reflex cough occurs in hypertrophic and post-nasal disease.

Reflex Neuroses.

Hay Fever. Hay fever is an acute affection ushered in by paroxysmal sneezing, itching, and smarting of the inner canthus of each eye, or of the throat or nose. After hours or days of sneezing coryza develops. The disease continues for a varying length of time, is more pronounced at certain seasons of the year, particularly the late fall. Coughing may be an additional symptom, and paroxysms of asthma may develop which are hard to distinguish from true bronchial asthma. The attack may be excited by vegetable emanations, particularly the pollen of plants, but other emanations may also induce it. Certain conditions of the nasal mucous membrane predispose to the attack. Local inflammation of the nose or obstructive diseases from hypertrophies are primarily present. To the exciting cause and the local predisposing cause may also be added a neurotic factor. The disease affects families of nervous constitution, and may occur through several generations. It is more common in this country than in other countries, and dwellers in cities are more subject to it than residents in the country. *Asthma* may be due to disease of the nose, but the only proof that it is of nasal origin is that it disappears after the nose has been treated for the various ailments that are supposed to cause it.

Idiopathic Rhinorrhœa. Characterized by a sudden profuse discharge of yellowish water. It ceases as suddenly as it develops, and is thought to be due to some functional derangement of the fifth nerve.

The Data Obtained by Observation.

The Objective Symptoms. Of the general objective symptoms, fever has been noted. In certain affections of the nose defective development of the general system is observed. This is particularly the case in adenoid vegetations of the nasopharynx in children. (See Diseases of the Pharynx.)

Local Examination. The Exterior. The external appearance of the nose is of diagnostic significance when marked deformity takes place. Its true shape is changed in myxœdema (*q. v.*). It is changed in disease of the bone due to syphilis. The bridge of the nose is sunken or depressed. It must not be confounded with the depression that occurs in fracture. The nose may be broadened in cases of tumors of an expanding nature in the nasal cavities. The local change soon extends to the cheek. The nose is also the seat of eruptions, as acne and hyperæmia, but they are usually of local origin. They may be suggestive of a gouty diathesis.

Internal Examination. The examination of the cavities of the nose consists of two procedures, both of which are necessary to determine with accuracy the condition of the organ. These are :

1. *Anterior Rhinoscopy.* For this are needed a good light, a nose speculum of some form, probes, a 10 per cent. solution of cocaine, and a head-mirror with central opening.

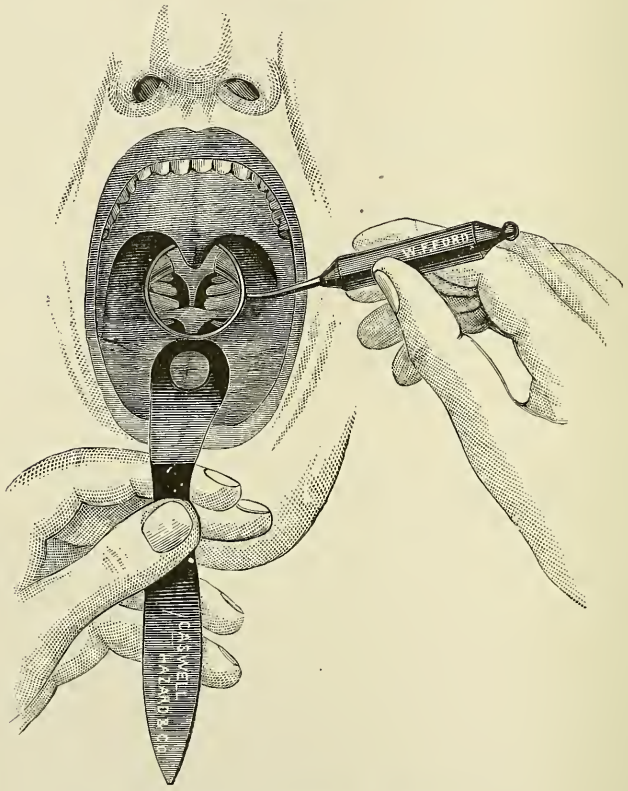
The examiner proceeds as follows : The patient is seated facing the surgeon, with the light behind and at one side of the head, as nearly as possible on a level with the eye of the operator. He must sit with shoulders and head a little forward. The operator adjusts his head-mirror so that the central aperture is in front of his own eye, and the reflected light falls on the nose of the patient. It is very important for nose-examination that the operator look through the aperture and not *under* the mirror. The speculum is then taken in one hand and the nostril dilated, so that the view of the interior is unobstructed. Do not try to dilate the bony part of the nose, but only the nostril. Proceed from before backward with the examination, carefully focusing the light on each part in succession, and gradually tilting the head of the patient backward. Thus the floor of the nose, the septum, inferior turbinated bones, middle turbinated bones, and sometimes the superior turbinated bones, are brought into view successively. In a broad nose one may at times see the posterior wall of the pharynx, which is distinguished by its peculiar wave-like movement when the patient swallows. The use of the probe is important, and without it no positive diagnosis can be made. With the probe the operator tries the condition of the mucous membrane, tests the consistency of tumors or hypertrophies, and so judges of the character of the condition. After this the enlarged parts should be touched with cocaine and the result observed. Contraction of a swelling under its influence proves its vascular origin.

2. *Posterior Rhinoscopy.* This is the most difficult part of the examination and requires much practice on the part of the operator. The instruments needed are a tongue depressor, head-reflector, two

sizes of throat-mirrors, a palate-hook or flat strings for holding forward the soft palate, and a curved applicator for cocaine, or a spray bottle with tip turned upward.

The patient is seated as before, the tongue held down by the tongue-depressor, and the patient is told to breathe freely through both mouth and nose. The light is directed into the pharynx and a mirror of the largest possible size inserted carefully behind the soft palate. The proper angle and the movement necessary to bring all parts into view can only be learned by practice. As a rule, it is best to hold the

FIG. 110.



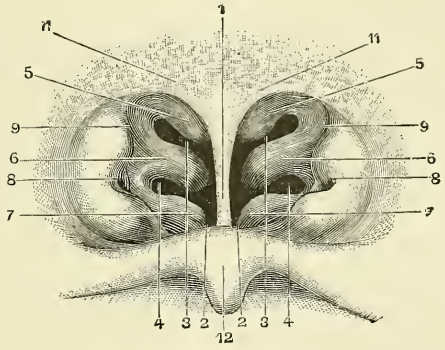
Rhinoscopic mirror in position. (BOSWORTH.)

handle well up at first, and note the condition of the vault of the pharynx, then gradually depress it, examining the choanæ from above downward. Do not keep the mirror too long in the throat. It is better to insert it several times than to weary the patient by attempting to see everything the first time. After the choanæ have been examined a turn of the mirror to either side will bring into view the orifices of the Eustachian tubes, and the examination is complete. If, after repeated attempts, it is found to be impossible to see the posterior nares, one must first seek to accustom the patient to the presence of

the instruments; if this fails, it may be necessary to resort to the palate hook or the cords to hold the uvula forward. The best hook is White's. It is necessary to apply cocaine to the soft palate before inserting the hook. Another plan, which is preferred by some, is to take the flat cords used for corset-laces, soak them in mucilage and dry them. These are then stiff enough to pass through the nostril, yet flexible enough to pull down and out through the mouth with forceps. Then by drawing forward both ends the soft palate is pulled out of the way. This is almost always necessary when applications are to be made to any spot in the pharynx.

Sometimes a view of the posterior nares may be obtained by making the patient breathe in short, quick gasps, by which the uvula is released. In ordinary breathing it is often tightly pressed against the posterior wall of the pharynx.

FIG. 111.



Rhinoscopic image.

1. Vomer or nasal septum. 2. Floor of nose. 3. Superior meatus. 4. Middle meatus. 5. Superior turbinated bone. 6. Middle turbinated bone. 7. Inferior turbinated bone. 8. Pharyngeal orifice of Eustachian tube. 9. Upper portion of Rosenmüller's groove. 11. Granular tissue at anterior portion of vault of pharynx. 12. Posterior surface of velum. (SEILER.)

By the above methods we are to determine the appearance and nutrition of the mucous membrane, relative size of the cavities, the nature of the discharge, and the presence of ulceration or perforation of the nares. Deviations of septum, enlargement or contraction of turbinated bones, the size of the cavities, and the presence of foreign bodies or abnormal growths are also detected.

Inspection. APPEARANCE OF THE MUCOUS MEMBRANE. The observer may find it unusually pale. This is seen in tuberculosis and in atrophic rhinitis. If a protuberant mass is observed to be transparent and shining, as well as pale, it is due to a *polypus*. If the mucous membrane is bright red, it may be due to *acute inflammation*, to *glanders*, or to *syphilis*. It is dull red in *chronic catarrhs* and *caseous rhinitis*. The coatings of the mucous membrane are of significance. If a dry mucus covers the part, it is due to *dry catarrh;* on the other hand, a dirty-gray membrane is indicative of diphtheritic rhinitis.

It is swollen and bathed with a serous, seropurulent, or purulent discharge, the character depending on the stage of inflammation. The

contractile tissue over the turbinated bones is congested and swollen. When probed it is elastic, and when cocaine is applied it shrinks.

In *chronic hypertrophic rhinitis* the uvula is thickened and elongated, on account of the hawking. The outer surface or the edges of the turbinated bones are enlarged throughout or in localities. The mucous membrane covering these spots is thickened, hard, and rough. If cocaine is applied, the mucous membrane does not contract, as in the swelling due to hyperæmia. The posterior ends of the inferior or middle turbinated bones are enormously enlarged, forming round tumors which obstruct more or less the posterior nares and project into the pharynx; polyps and deviation of the septum complicate these cases.

The same appearances are seen in chronic post-nasal catarrh, and in addition a mammillated and thickened appearance of the pharyngeal mucous membrane and that of the posterior third of the septum. In dry catarrh the mucous membrane is coated with mucus or covered with crusts. The membrane is thin, pale, hard to the touch, and covered with a layer of dried secretions and crusts in atrophic rhinitis. The nasal passages are abnormally wide and one or both turbinated bones are atrophied.

Abnormal Growths. A grayish yellow or *greenish shiny* mass, with a broad base, soft and yielding on probing, is a nasal polypus. It cannot usually be circumscribed. The passages are enlarged in atrophic rhinitis. One may be occluded by an enlarged turbinated bone or by deviation of the septum.

Ulceration. Ulceration of the mucous membrane is usually a manifestation of lupus, tuberculosis, or tertiary syphilis. In *lupus* the ulceration has extended from the exterior. If ozæna is present in a patient with lupus it is probable that there is also lupus of the nasal passages. The ulcers may be followed by necrosis and caries of the bones. If the ozæna is not removable by antiseptic sprays the bones are probably affected. A discharge of sequestra makes the diagnosis positive. Rhinoscopy and careful palpation may reveal the ulcer and a carious bone. *Tuberculous* ulcers are usually found in the septum. They are rarely primary. They present a whitish-gray surface, with elevations of infiltrated tissue. They bleed on the slightest provocation. The mucous membrane surrounding them is torn. Tubercle bacilli can be found in the scrapings from the ulcer. In *syphilis* the ulcers are situated anywhere in the nares. A history of infection, or of secondary and tertiary manifestations, can be obtained. The stench of the breath is sickening, and the patient complains of stenosis and loss of smell. There is some localized tenderness, and sleeplessness, debility, and emaciation may ensue. They may be mere superficial excoriations, or deep serpiginous ulcers surrounded by an inflammatory zone. Caries can be detected with a probe. The ulcerated surfaces are covered with a dry, greenish crust. *Foreign bodies* usually cause ulceration if impacted.

Neuro-paralytic ulcers are painless and spread rapidly over considerable surface; they follow paralysis of the fifth nerve. They are dry and sluggish and do not extend to the skin. Post-febrile ulcers follow

measles, scarlatina, typhoid, and variola, and are due to rupture of small abscesses, with the subsequent formation of ulcer. They are usually anterior on the septum or inside the alæ, and scabs form over the surface. They are very irritable. Ulcers may perforate the septum or the floor of the nose. They are usually due to syphilis. Simple perforating ulcer of neuro-paralytic origin may also occur.

Nasal Secretion. The *odor* of the discharge is suggestive of diphtheria and also of the presence of *foreign bodies*. The discharge in the latter instance is sanious or purulent. Animal parasites, as well as pease and beans, cause pain, symptoms of obstruction, and ulceration. In syphilis with caries the odor is marked, usually gangrenous.

Atrophic Rhinitis, or Ozæna. The *odor* is characteristic, and is diagnostic if syphilis is excluded. A sense of dryness is complained of. Occasional obstruction arises from accumulation of crusts, otherwise the passage is unduly open. There are constant hawking and spitting of brownish-green crusts, which are often blood-tinged. Frontal headaches may occur in paroxysms. The patient is often depressed in spirits. The bridge of the nose may fall in slightly.

Physical Character. The character of the secretions is of diagnostic significance. They may be liquid, semi-solid, or solid. The liquid secretions may be serous, mucous, or purulent. *Serous* secretions occur in acute rhinitis, hay fever, and idiopathic rhinorrhœa, and follow bursting of cysts. The secretion of *mucus* occurs in the later stages of inflammation of the mucous membrane and in chronic forms. A *mucopurulent* secretion is seen in chronic rhinitis, and pure *pus* in abscesses of the septum or cavity. In hereditary syphilis it is at first mucopurulent, then purulent, and then sanious. A sanious acrid discharge, with false membrane discharged or evident on inspection, is due to *diphtheria*. A fetid, sanious, or ichorous discharge, with frequent attacks of epistaxis, attends malignant nasal growths. A discharge of blood is known as epistaxis. (See page 426.) The *semi-solid* secretions may be due to mucus alone, or to blood-clots mingled with serum or with pus. The latter occur in atrophic and hypertrophic catarrhs.

Caseous Rhinitis. A semi-solid secretion is diagnostic. On examination the cavities in this affection are found to be filled with cheesy matters, easily broken up with the probe. The mucous membrane is dull red. The material is discharged in masses at intervals through the mouth or nostrils, relieving the previous extreme stenosis. If neglected for a long time, deformity of the face and disease of the bones and cartilages ensue from pressure.

The *solid* secretions may be mucous crusts, as in acute and chronic catarrhs, blood-crusts after epistaxis and traumatism, membrane in diphtheritic rhinitis, slough from ulcers, and rhinoliths. The latter are gray or greenish-brown in color, hard and rough, either fixed or movable.

Microscopical Character. The normal secretion from the nose contains squamous and ciliated epithelium, isolated leucocytes, and various fungi. The fluid is thick, alkaline in reaction, and has a slight odor. It contains mucin. In disease of the nasal cavities the fluid

changes. In acute nasal catarrh it is more copious and thinner. It remains alkaline, and contains epithelium and fungi. When the stage of suppuration is reached, the secretion may consist entirely of pus. Cerebro-spinal fluid may also be discharged through the nose in certain brain-tumors. In such fluid albumin is absent. Detection of this fluid is of diagnostic value, as it points to the central lesion.

The Charcot-Leyden crystals are found in the nasal secretion in asthmatic patients, and sometimes in acute coryza.

Bacteriological Character. In diphtheria the characteristic micro-organism is seen. Recognition of glanders may be based upon finding the bacillus in the nasal secretion. (See page 336.) Cultivations may be made. The nature of ulcers may be determined by microscopical examination. The tubercle bacillus can sometimes be detected. A pneumococcus or bodies that resemble it have been found in the secretion in ozæna. Thrush-fungi have also been found, as well as some mould fungi.

Mouth-breathing. Much valuable information is obtained by noting the breathing and the condition of the voice. Mouth-breathing may be present if the face is drawn and vacant and there are cracks and fissures in the mouth. The voice is usually nasal. The resonating quality is lost entirely. Snoring accompanies these conditions. (See Obstructive Symptoms.)

Palpation. The probe is used to determine the character of enlargements or tumors, and the patulency of foramina ; also to examine the mucous membrane as to induration and the presence of caries or necrosis. By the finger the nasal pharynx is palpated to confirm the results of rhinoscopy. In this manner adenoid vegetations and hypertrophy of the inferior turbinated bones are detected. The finger should be protected by the use of a mouth-gag or by a jointed thimble.

Epistaxis. The blood may flow in drops, or a continuous stream may pour out from the anterior nares. Sometimes it falls into the pharynx and is hawked up, or is swallowed and then vomited.

It may be due to local causes, or to constitutional conditions. Traumatisms (scratching the nose), new growths, and foreign bodies are causative agents ; it may be due to fractured skull. Local causes : On inspection, the cause may be found in enlarged veins at the anterior inferior portion of the septum, a bleeding ulcer, a new growth, or the ulceration of a foreign body. The general conditions which are causal are : (1) Plethora ; (2) engorgement due to the ascent of an elevation ; (3) all forms of anæmia ; (4) hæmophilia ; (5) cerebral congestion and severe headache ; (6) the commencement of fevers, particularly typhoid fever ; (7) early stages of leprosy. In children exposed to the sun, and after exertion, it is of frequent occurrence, and is seen often at puberty in delicate children.

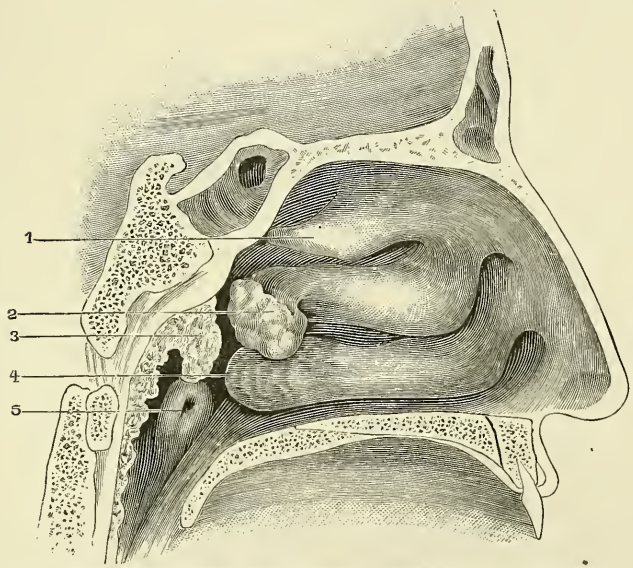
Diseases of the Nose.

The subjective and objective symptoms previously described are due in general to inflammations, malformations, morbid growths, and foreign bodies. They are recognized by their subjective and objective signs, by rhinoscopic examinations, and by bacteriological and microscopical

research. The inflammations may be acute or chronic, primary or secondary. When secondary, both acute and chronic inflammations may be due to infections. To the acute varieties belong the acute catarrh of measles, glanders, hay fever or influenza; to the chronic belong syphilis and tuberculosis.

Simple Acute Rhinitis. Acute Coryza, "Cold in the Head." Ushered in with a feeling of lassitude, aching in the back and limbs, and feverishness, a sense of fulness is felt in the nostrils, with sneezing. After twenty-four hours an irritating discharge begins. During this time the malaise has increased. The pain in the forehead and cheeks has become more pronounced, and a nasal twang is given to the voice. The feverishness continues, reaching 101° in the more pronounced

Fig. 112.



Vertical section through nasal cavities. (Diagrammatic.) (Seiler.)

1. Superior turbinated bone. 2. Middle turbinated bone, with posterior hypertrophy. 3. Section of hypertrophied pharyngeal tonsil. 4. Inferior turbinated bone. 5. Orifice of Eustachian tube.

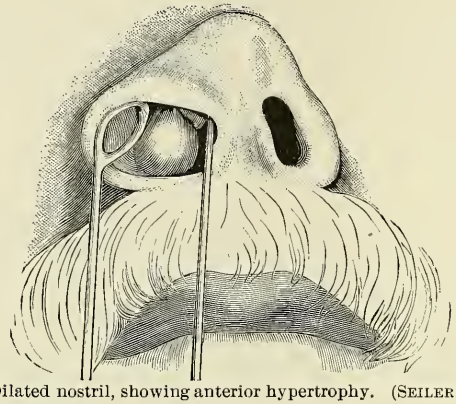
cases, with thirst and loss of appetite. At the height of the fever, in twenty-four or forty-eight hours, a crop of herpes very often develops on the lips. The general symptoms then subside and the local symptoms change. The discharge becomes thick and purulent, the fulness continues, but the pain is diminished. The inflammation often extends up to the tear-ducts and to the eyelids. The latter are congested and smart very much. Very frequently, also, the inflammation extends to the pharynx, causing soreness of the throat and stiffness of the neck, and the larynx even may be involved. A slight deafness may result from the inflammation extending into the Eustachian tube.

Chronic Rhinitis. Four varieties are distinguished, to all of which the term *nasal catarrh* is applied. In one there is hypertrophy of the

turbinated bones ; in the second there is extension of the disease to the post-pharynx—chronic post-nasal catarrh ; in the third there is absolute dryness of the mucous membrane—rhinitis sicca, or dry catarrh ; in the fourth there is atrophy of the mucous membrane—atrophic rhinitis, or ozæna.

Chronic Hypertrophic Rhinitis. The affection comes on gradually after repeated acute attacks of coryza. The only symptoms may be

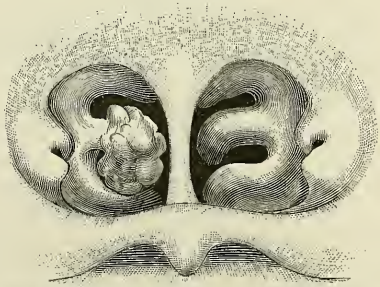
FIG. 113.



Dilated nostril, showing anterior hypertrophy. (SEILER.)

slight fulness in the nose and a little hoarseness of the voice. In more advanced stages the symptoms of stenosis are marked with oral breathing, snoring, and nasal sound. There is a constant discharge of muco-pus backward into the pharynx, causing hawking. The hearing is frequently impaired, as well as the taste and smell. The discharge

FIG. 114.



Rhinoscopic image from a case of posterior hypertrophy on the middle turbinated bone. (SEILER.)

often affects the larynx, causing an irritating cough. The hypertrophied tissue on the turbinated bones, and the pressure of the bone on the septum, may lead to reflex attacks of asthma.

Chronic Post-nasal Catarrh is an extension of the rhinitis into the pharynx. It is distinguished by discomfort or pain in the soft palate and posterior nares. There are tingling and a sense of fulness at the root of the nose, with frontal headache ; the patient complains of a

bad taste in the back of the mouth and of constant flow of thick secretion into the pharynx, causing snoring and hawking. The same perversion of the senses of taste, smell, hearing, and of the voice occurs as in acute rhinitis. Headache seems to be due to the condition of the pharynx. (See Atrophic Rhinitis, page 425.)

Dry Catarrh, or Rhinitis Sicca, is also chronic in its course, accompanied by tingling and dryness of the nostrils. A faint, musty odor is detected, but there is no discharge or sense of obstruction. In severe cases there may be sharp pain in the nose extending to the forehead.

Syphilitic Coryza is seen in infants and young children affected with hereditary syphilis. The nostrils are swollen and red at the edges, sometimes completely occluded, causing oral respiration and inability to take the breast or bottle.

Pustules, fissures, and ulcers are found in the nose and at the margin of the orifices. They are also seen in the pharynx and larynx. Hemorrhages may occur. Other evidences of hereditary syphilis are present.

The Auxiliary Cavities of the Nose.

The Antrum is subject to abscess, cysts and polypi, parasites, and tumors.

Abscess. An odor somewhat like that of ozæna, a putrid taste, nausea, anorexia, pain in the cheek and root of the nose, often neuralgia in the frontal region, and malaise are present. A very characteristic symptom is the discharge of pus from one nostril on leaning the head forward. There is often a bad tooth on the same side in the upper jaw.

The Sinuses. The frontal, ethmoidal, and sphenoidal sinuses are subject to inflammation, abscess, traumatism, and the irritation of foreign bodies, usually parasites.

The frontal sinuses are the only ones which exhibit external symptoms. When these cavities are inflamed the patient complains of pain and tenderness over the frontal protuberances; if the process goes on to the formation of abscess, there may be redness and swelling and finally fluctuation. If the communication is not closed, there is a fetid discharge from the middle meatus.

When the sphenoidal and ethmoidal sinuses are affected there are no external symptoms unless the enlargement is so great as to affect the orbit. There is deep-seated pain. Pus is seen exuding into the superior meatus and flowing backward into the pharynx. Parasites cause intense pain and lead to abscess, caries, and necrosis. *Rhinoscopic* examination in disease of the antrum shows rough hypertrophic enlargement on the under surface of the middle turbinated bone and a flow of pus into the middle meatus. Sometimes a probe can be passed into the antrum from the nose. Often an exploratory puncture is necessary. *When the foramen is obstructed* there is a dull aching pain in the upper jaw, with deformity of the orbit, face, hard palate, and nostril. Fluctuation can usually be found at some point after a time.

The *lacrymal duct* and *sac* are often the seat of inflammation by extension, causing pain, obstruction in the nose, and epiphora. On

examination pus will be seen flowing forward over the inferior meatus. When the lacrymal probe is introduced the ducts are found to be painful and obstructed, and pus exudes.

The Larynx.

The structural composition of the larynx does not differ from that of other parts of the respiratory passage. Mucous membrane, connective tissue, cartilages, and muscle are similar to the same tissues situated elsewhere.

The result of their anatomical association in the larynx is the establishment of the functions of that organ, the formation of the voice and the admission of air. Now, the morbid processes that affect the larynx do not differ from morbid processes elsewhere in which similar tissues are involved. Each tissue is liable to congestion, to inflammation, to degeneration, to new-growth formation ; the joints may become ankylosed, the muscles either paralyzed or the seat of spasm, and we have, therefore, all the symptoms common to morbid processes in each class of tissue. We meet with other symptoms beside, which result from the anatomical position of the larynx and of its functions. The cords cannot vibrate, or the muscles and articulations cannot move, and *dysphonia* or *aphonia* occurs. The narrow chink of the glottis soon becomes occluded, giving rise to *dyspnœa*. Obstruction to the pathway or pain from inflammation or ulceration causes *dysphagia*. The sensitiveness of the mucous membrane provokes *cough* on the slightest provocation.

The larynx is a highly specialized organ, and is well innervated. Large central nuclei, connected by a large nerve which passes over a circuitous route and which anastomoses with other nerve-cords, preside over the function of phonation. Affections of the central nuclei, affections of the nerve-trunk or of adjacent structures exerting pressure upon the trunk, have their expression in disorder of the larynx, particularly if phonation is disturbed. In other words, the phenomena of laryngeal disease may be symptomatic of affections of the brain or of the nerve-trunk, as well as of the larynx. (See Nervous Diseases.)

Owing to the anatomical position and special function of the organ the symptoms of disease of the larynx are very striking, pointing at once to the seat of trouble. Laryngeal affections are not likely to be mistaken for disease of contiguous parts, although retropharyngeal abscess, abscess at the side of the pharynx, disease of the thyroid gland, and inflammation of the lymphatics or cellular tissue in the neck may cause symptoms suggestive of laryngeal disease.

Finally, morbid processes in the larynx determined by the symptoms and physical appearances may be symptomatic of general processes : acute inflammation, of erysipelas, typhoid fever, smallpox, or measles ; chronic inflammation or ulceration, of the rheumatic or gouty diathesis, syphilis or tuberculosis ; scars, of syphilis ; ankylosis, of rheumatic gout. The laryngeal symptoms of brain disease or of affections of the nerve-trunk have been referred to.

The practical point of all this is that affections of the larynx are not

due to *primary* disease of that organ alone, but are often *secondary* either to general processes or to local morbid processes elsewhere.

Therefore, when laryngeal symptoms or lesions are observed, seek beyond the larynx, as well as in it, for their cause.

The Data Obtained by Inquiry.

The Social History. Acute laryngeal diseases are more common in childhood, chronic diseases in late life. Those occupations which compel the inhalation of noxious vapors or excessive use of the voice predispose to laryngeal diseases. Alcoholic subjects and those who use tobacco to excess are liable to laryngeal affections. As with the nose so with the larynx, no special disease is inherited and need be looked for in the *family history.* But we may inquire for a diathetic condition, as gout or rheumatism, which predisposes to a mucous membrane inflammation, or a family type which leads a parent to say his child "has a tendency to croup," a popular expression which has in it an element of truth. That condition or state which predisposes to "colds" belongs also to a family type.

On inquiry as to *previous* disease various acute infections and syphilis and tuberculosis are to be looked for. In a study of the *present disease* it must be borne in mind that laryngeal affections notably may be secondary, and, therefore, the presence of other diseases must be inquired into. Particularly do we inquire for nervous diseases, and in children for rhachitis. One thing is to be borne in mind—one attack of acute laryngitis predisposes to subsequent attacks.

Subjective Symptoms. Pain. Pain in the larynx may be sharp, stabbing in character, or simply a tickling or burning with a feeling of pressure. It is increased by pressure and by speaking or swallowing. Pain is sometimes so intense as to render speaking and swallowing impossible. In acute laryngitis the pain is cutting and burning. In the milder inflammations, in dry catarrh, and in lupus it amounts to soreness only. The pain is severe and sharp in cases of cancer and tuberculosis, rarely in syphilis, and when foreign bodies are present in the structures. The pain may be very severe and intense when there is destructive ulceration. It is a diagnostic symptom of *perichondritis.*

Perichondritis. Inflammation about the cartilages or perichondritis is usually phlegmonous in character, and leads to the formation of abscess. The collateral œdema is so great as to cause some obstruction, with cough and hoarseness. On palpation the larynx is extremely tender. The pain is increased by movement of the larynx, as in speaking or swallowing. If the inflammation involves the arytenoid cartilages, pain extends toward the ear, the vestibule is swollen, the cartilage fixed. On the other hand, when the cricoid is diseased there are pain on swallowing of solid food, on account of interference with the muscular attachments, dyspnœa, and paralysis of the posterior crico-arytenoid muscles.

Inflammation of the thyroid cartilage may open externally or internally. In the latter case the abscess can be seen in the larynx. Discharge of pus and necrosed cartilage confirms the diagnosis. By means

of a sound the bare cartilage can be detected, giving further proof of the presence of the disease. The pain may extend to the ears in carcinoma. The pain is propagated by the auricular branches of the vagus.

Paræsthesia. Peculiar sensations are frequently complained of. They may be *burning*, *tickling*, or *itching* in character, or it may seem as if a foreign body were present in the part, as a hair, or it may seem like a draught of cold air striking the parts. Sometimes after a foreign body has actually been present, the sensation of its presence will continue a long while after its removal. A sense of pressure or fulness, the feeling of a lump in the throat, is frequently complained of, provoking a desire to swallow. The patient will seek advice on account of it. It is known as the *globus hystericus*, and is recognized by the absence of local changes in the larynx, by its association with other phenomena of hysteria, and by its disappearance or aggravation under the influence of excitement. This abnormal sensation is seen in hysteria and hypochondriasis. It is one of the nerve-perturbations in chlorosis and anæmia.

A feeling of *dryness* is frequently complained of, and attends the first stage of acute, and any stage of chronic laryngitis. The sense of fulness, or pressure, or feeling of the presence of a foreign body is complained of in all forms of laryngitis, in croup, in œdema of the glottis, or epiglottis, and in syphilitic infiltration.

Hyperæsthesia and Anæsthesia. When there is *hyperæsthesia* there is constant desire to *cough* (see page 435), and the act is induced by the slightest irritation. The desire to cough, independently of the act, however, is of itself an extreme annoyance. It is a disagreeable sensation present in acute inflammations and in early phthisis. At times of menstruation and during pregnancy both symptoms are frequently complained of. Hyperæsthesia is easily recognized with the probe. In *anæsthesia* particles of food fall into the larynx. The mucous membrane is insensitive to the contact of the probe. Anæsthesia occurs in hysteria, diphtheritic paralysis, paralysis of the superior laryngeal nerve, bulbar paralysis and cerebral softening or hemorrhage, or coma from any cause.

Dysphonia. The most common symptom of affections of the larynx is disturbance of the function of speech. The voice is changed in character, or may be lost in any affection which causes swelling of the mucous membrane, or occlusion of the orifice, or which interferes with the action of the vocal cords. The voice may be *hoarse* in acute and chronic inflammations, in tumors and in specific ulcerations about the larynx, and in paralysis of the cords. From simple hoarseness it may vary in intensity to complete aphonia. Laryngoscopic examination is necessary in order to detect the presence or absence of paralyses. (See Paralyses.)

Chronic Laryngitis. *Chronic hoarseness* may be due to chronic laryngitis. This affection either originates in an acute attack or comes on slowly. Prolonged use of the voice in a higher key than natural, or in the open air, the use of alcohol, constant exposure, are exciting causes. It is symptomatic of syphilis and tuberculosis. It frequently results from inflammation of the upper air-passages, particularly chronic

pharyngitis. It occurs after middle life more frequently, and usually in the male sex. There is discomfort on long speaking, with dryness and tickling. At first the secretion of mucus is very slight, but after hawking and coughing it increases in amount. Hoarseness occurs, and if the patient is careless or persists in the baneful occupation, complete aphonia may result. The voice is clearest in the morning, after expectoration of the mucus that accumulated in the night, but becomes husky toward night. The aphonia may occur in paroxysms, and is relieved by coughing up a dry secretion. The cough is never severe. The sputum is small in amount, glairy, and is often in little balls or crusts.

Lupus. Slight hoarseness, deepening to dysphonia or even aphonia, attended by soreness, and later some dysphagia, is seen in *lupus*. Infiltration and scar-contractions cause dyspnœa later in some instances. Dysphonia from inflammation or œdema is also a symptom of *leprosy*, which, however, is present in other situations as well. The *duration* may be significant. Hoarseness of long duration (years) is said to be prodromal of *cancer* (Ziemssen).

Functional Dysphonia or aphonia may occur after excessive use of the voice and in hysteria. Hysterical aphonia occurs in women and young girls; the laryngoscope reveals nothing; the acts of coughing, laughing, and sneezing are normal, and a sound may be created in either act; it appears and disappears suddenly.

Tone of the Voice. The character of the voice may change. When one-sided paralysis of a cord is present the voice is flat and toneless. In cases of paresis of the tensors of the cords a falsetto voice results. Diplophonia occurs in one-sided paralysis, and in some cases in which small tumors lying between the cords come up during the act of phonation and form nodes. Two tones are formed at the same time. Frequently only certain tones are doubled.

Dyspnœa. This is one of the frequent symptoms—and the most serious—of laryngeal disease. It may be due (1) to obstruction by inflammatory or œdematous swelling; (2) to spasm; (3) to tumors or foreign bodies in the larynx; (4) to the cicatrization of ulcers after syphilis or lupus; (5) to paralysis of the abductors or adductors of the larynx. It may be, therefore, organic or spasmodic.

Duration. Dyspnœa from disease of the larynx may develop gradually and continue over a long period of time, or it may be acute in onset, depending upon the character of the morbid process which has brought about the obstruction. *Acute* paroxysms of dyspnœa, one of which may end in death, sometimes occur in the course of affections in which *chronic* dyspnœa is present; thus sudden œdema may occur in cases of syphilitic or tuberculous ulceration.

Laryngeal Dyspnœa must be distinguished from other forms of dyspnœa: 1. Dyspnœa from diseases of the heart and lungs. 2. Dyspnœa from pressure upon the trachea. The larynx is not markedly moved during the respiratory acts, and the patient bends the head forward instead of backward. 3. Dyspnœa from pressure on the larynx. Cellulitis of the neck, tumors of the lymph-glands, goitre, and retro-pharyngeal abscess are provocative of this form of laryngeal dyspnœa. Examination of the respective localities by inspection and by touch

reveals the cause. It may be worthy of remark that dyspnœa in diphtheria, frequently thought to be due to internal occlusion, may be due to pressure of enlarged glands on the bronchus and larynx.

Inspiratory Dyspnœa. Dyspnœa may vary in degree from slight inconvenience in breathing, noticeable to the patient, to the violent struggling for breath which is seen in cases of extreme stenosis of the larynx. If carefully observed in either case the larynx is seen to rise and fall. In extreme forms of obstruction the head is bent back, the neck stretched, the muscles of the neck contracted. The spaces above the sternum and at the sides of the trachea are drawn in with inspiration, and the alæ of the nose work vigorously. Further evidence that sufficient air does not enter the lungs is found in recession of the epigastrium and drawing in of the ribs at the base of the chest during the act of inspiration. The countenance is dusky or ashy-gray, the lips become cyanosed, and the nails bluish as the dyspnœa persists and increases. A cold perspiration breaks out on the forehead, and finally, from exhaustion, the respiration becomes slower and slower until mere gasps are seen. The heart's action increases in frequency as the stenosis increases. Death usually takes place from asphyxia, the child first falling into a stupor, on account of carbonic-acid-poisoning.

Sounds attend the act of inspiration, the character depending on the nature of the obstruction. In obstruction from simple spasm, or from intense inflammation of the larynx, without secretion, the sound of inspiration is *harsh* and *stridulous*. In obstruction from œdema or from exudation, as in laryngeal diphtheria, the sound of the inspiration is *loud* and *stridulous*, but not shrill. The expiration is usually noiseless and prolonged. The short, stridulous, or gasping inspiration is followed by prolonged gentle expiration. In spasmodic croup the expiration is like snoring. The interval between expiration and inspiration is lessened, the respirations are hurried.

Laryngismus Stridulus. In this form of dyspnœa the act of breathing ceases in the midst of inspiration, and is attended by a characteristic sound. It is seen usually in poorly nourished children. It is of frequent occurrence in *rickets*, its presence suggesting that disease when other manifestations of it are obscure.

The symptoms occur suddenly and are very alarming. The child awakes in the night, and suddenly stops breathing after a few short whistling inspirations. The child is seized with terror, which is depicted on the countenance; the eyes stare; the face is pallid at first, but rapidly becomes livid. The alæ nasi are extended, the head is thrown back, and the spine arched. A cold perspiration breaks out over the forehead. Carpo-pedal spasms may occur and the urine and feces be discharged involuntarily. In a few seconds, or, at most, two minutes, the child draws two or more deep, noisy inspirations, each one lessening in depth and sound, when color returns to the face, the cyanosis gradually disappears, and the child becomes tranquil.

In mild forms the child "catches its breath." It holds its breath, and then makes a noisy inspiration.

Attacks of laryngismus stridulus are more rare in adults. They may occur in *hysterical* subjects. In the attack there occurs a series

of long, harsh, whistling or stridulous inspirations, followed by short, noisy expirations. Rarely is there complete closure of the glottis.

In both children and adults general convulsions may occur during the attack, or carpo-pedal spasms alone may be seen. Among adults the convulsions occur only in hysterical subjects.

The *diagnosis* of laryngismus stridulus is based upon the absence of laryngeal symptoms prior to the attack, the absence of cough or hoarseness, and complete disappearance of all laryngeal symptoms when the attack subsides. The absence of pain and fever and of laryngoscopic signs is noteworthy. This applies, of course, to spasm that occurs independently of laryngeal disease.

Expiratory Dyspnœa. In some forms of laryngeal obstruction the *exit* of air is interfered with, as in a movable tumor below the vocal cords. We have expiratory dyspnœa. The act of inspiration is complete, the act of expiration is suddenly checked by the obstruction, on account of which the lungs become overfilled with air and an emphysema develops.

Dysphagia. Difficulty in swallowing is most marked when destruction of tissue in the larynx takes place, or when there is acute inflammation about the muscles or their attachments; hence, when ulcers, tuberculous or malignant, are present, or perichondritis arises, the difficulty is so great as to prevent the taking of food.

Dysphagia is recognized by *pain* and by the falling of particles of food into the larynx, exciting cough. It must be distinguished from the dysphagia of pharyngeal affections by ocular examination, the location of the pain, and the non-association of rheumatism.

Inflammation of the Epiglottis. When the epiglottis is the seat of acute inflammation there is great dysphagia on account of pain, or on account of the obstruction. The sensation of a lump in the throat at the base of the tongue or the top of the larynx is complained of, and there is pain on swallowing. The pain becomes very intense at times. Fluids cannot be taken, for the fluid enters the larynx when the patient attempts to swallow, because the epiglottis does not protect the glottis. The voice is usually clear throughout the attack, and the general symptoms are not marked.

When the epiglottis is fixed or ulcerated, and in some forms of ulceration of the larynx, the food enters the larynx, and hence produces dysphagia.

Mis-swallowing, or "swallowing the wrong way," occurs in all conditions in which food is allowed to enter the larynx. Although conditions favorable for its occurrence are present, it may not take place unless the patient is off his guard during the act of swallowing, as when he is laughing. It may then occur even in normal cases. It is associated with anæsthesia of the larynx, and occurs in central nerve affections which cause that condition.

Cough. (See Diseases of the Lungs.) Sometimes valuable information is derived from the character and severity of the cough. Several forms are noted:

First, the *dry* cough, as seen in acute laryngitis. It is almost constant, and is aggravated when the patient speaks, takes fluid, or inspires

deeply. In children it is abrupt, brassy, or metallic, stridulous or whistling, so-called "croup-cough," as seen in cases of "false croup" and laryngitis with œdema.

Second, a *dry hoarse* cough occurs in the course of chronic laryngitis.

Third, *cough* with *whoop*. With the act of coughing a whooping sound may be heard in inspiration. After rapid violent expiratory acts the whoop takes place with inspiration. It is spasmodic and convulsive, and is followed by retching, and often by vomiting. (See Pertussis.)

Fourth, the cough is of such a character as to give one the idea that it is *suppressed* in membranous and œdematous laryngitis.

Fifth, a cough frequently occurs without any local anatomical changes in the larynx, which seems to be purely of *nervous* origin. Two forms are seen: *a. Paroxysmal.* Severe coughing occurs suddenly, and cannot be controlled by the patient. It ceases without cause, returning in a few hours. There is no expectoration. *b. Continued and rhythmical.* It is not so severe as in the paroxysmal form, but consists in a regularly recurring cough more or less loud. It does not occur while eating or speaking and ceases entirely during sleep. It is usually worse when the patient is under observation. Examination with the laryngoscope reveals absence of disease. This form of cough is seen after diphtheria, when sexual disturbances are present, at puberty, in cases of anæmia and chlorosis, or of neurasthenia or hysteria. The tone is usually high.

Hemorrhage. Hard coughing or an unusual straining of the voice may lead to the occurrence of slight hemorrhage. Only after injuries are hemorrhages from the larynx at all copious. Moderate hemorrhages occur in scurvy, hæmophilia, hemorrhagic smallpox, typhus fever, and leukæmia.

Disturbance of Co-ordination. Several forms of such disturbance are seen. Spasm of the glottis may occur with each effort to speak, causing either serious interference or complete inability to utter a word, as in stuttering. Sometimes, instead of the glottis opening to complete the act of inspiration, it may close. Sudden inspiratory dyspnœa, therefore, occurs, and is attended with stridor.

Spasm of the glottis is a frequent complication of disease of the larynx. It is seen in "crises," as in locomotor ataxia.

The Data Obtained by Observation.

Objective Symptoms. The objective symptoms are determined by *inspection* and *palpation*. Inspection of the *exterior* of the larynx reveals the presence of swelling, and the movements of the organ as a whole. Local swelling of the tissues over the larynx may occur in inflammations of the cartilages; they are usually of syphilitic origin, but may attend carcinoma or tumor. There is more or less marked swelling in inflammation of the cartilages, which after a time fluctuates, and, when opened, discharges pus and necrosed cartilage. The objective signs of inflammation are noted.

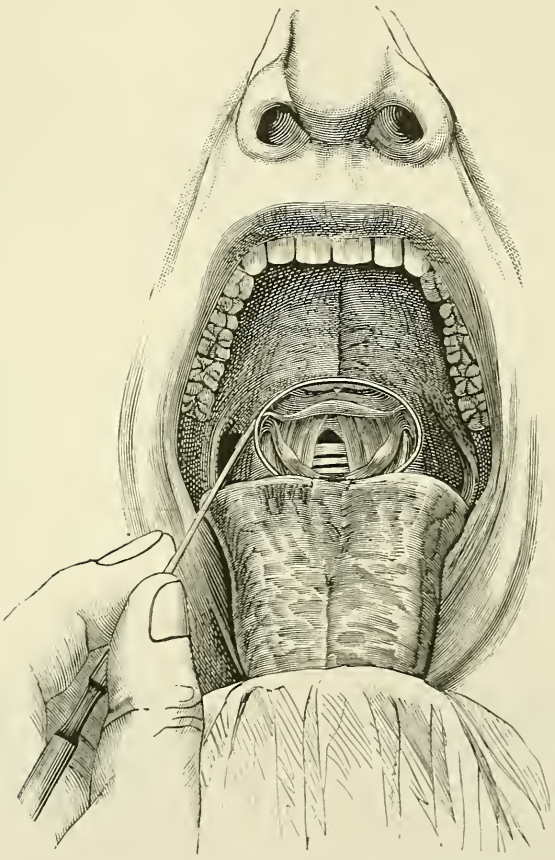
The movement of the larynx is increased in cases of dyspnœa. It

is accompanied by recession of the spaces above the sternum and the clavicles, with clonic contraction of the sterno-cleido-mastoid muscle.

The interior of the larynx is studied by inspection (laryngoscopy), and by palpation (probe or fingers).

Laryngoscopy. The first requisite is a good light, sunlight, a good student's-lamp, or an Argand or Welsbach gas-burner; the electric light is not satisfactory. Second, a good reflector is required. It may be attached to a head-band or a spectacle-frame. It should be concave for artificial light, plain for sunlight, and should be pierced in the centre. Third, laryngeal mirrors of different sizes and a curved probe complete the instruments necessary for examination of the larynx.

FIG. 115.



Laryngeal mirror in position, displaying the laryngeal image. (COHEN.)

EXAMINATION. The patient is seated with the source of the light at one side and behind him; the head and shoulders are brought well forward and the head slightly raised. The operator takes a seat in front at a proper distance for the focal length of the reflector, and focuses the light on the patient's mouth, warms the laryngeal mirror

over the flame and tests its temperature on the back of the hand. It should be moderately heated, so that when it is placed in the mouth the vapor of the breath will not precipitate on its surface. The patient must open the mouth and protrude the tongue, which is grasped between the folds of a napkin by the thumb and fingers of the operator. The tongue should be gently but firmly grasped. The mirror is then inserted carefully and quickly, face downward, into the pharynx. Care must be taken not to touch the tongue or palate, otherwise the patient may be made to retch and become alarmed. The mirror is passed to the posterior wall of the pharynx, and so directed that the image of the larynx is reflected to the eye of the operator. The patient is made to phonate "ā" or "ee," not "ah," and then to respire. The various structures and the action of the cords are observed. The appearances of the mucous membrane are studied during quiet respiration.

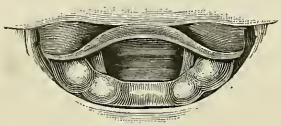
The epiglottis is very dependent, so that often the larynx can only be seen by having the patient stand while the operator remains seated. The patient's head is bowed on his chest and the examination proceeds.

The first examination may not result satisfactorily, but little being observed on account of the spasm of the pharyngeal muscles. Repeated sittings may remove apprehension and accustom the mucous membrane to the presence of the instrument. This object may be attained by administering bromides, or by applying cocaine to the pharynx.

The probe is needed only to ascertain the consistency of tumors and growths. Cocaine must be applied before it is used.

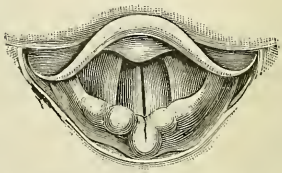
Appearance of the Larynx in Health. Fig. 115 shows the larynx as it is seen in the laryngoscopic mirror. Above (upper part) is the arched epiglottis, below it the cavity of the larynx. In the centre are the vocal cords, white and glistening; on each side of these the pink folds of the false cords. At the bottom of the mirror are the arytenoid bodies, and between them the folds of the inter-arytenoid space. Below and outside the arytenoid bodies are the fossæ. The mucous membrane is pink throughout except on the cords. In respiration

FIG. 116.



Laryngeal image during respiration.

FIG. 117.



Laryngeal image during phonation.

the arytenoids separate, carrying the ends of the cords which are attached to them with them, and leaving a triangular opening—the glottis—through which the rings of the trachea can be seen. (See Fig. 116.) In phonation the arytenoids approach each other, obliterating the inter-arytenoid space; the inner edges of the cords come in contact and close the glottis. (See Fig. 117.)

Appearance in Disease. A note must be made of the color of the various parts, of the presence or absence of swelling, of ulceration, of new growths, and of alterations of the movements of the parts concerned in phonation, particularly of the cartilages and the cords.

Color. The color is an indication of the degree of congestion. *Anæmia* of the larynx may be merely a part of a general anæmia from any cause. In chlorosis it is seen before the external appearance is marked. An intense anæmia of the larynx is an early and valuable symptom of pulmonary tuberculosis. The mucous membrane is pale.

Hyperæmia may be active or passive. It is readily recognized by the intense redness.

Active hyperæmia occurs in acute laryngitis, either of the primary or secondary forms.

Passive hyperæmia occurs in general obstruction to the circulation, as emphysema or valvular lesions; pressure on veins by tumors; forced expiration and holding the breath; in paroxysmal cough, especially whooping-cough. Active hyperæmias lead to catarrhs, passive to œdema.

Swelling and Infiltration. *Swelling* of the epiglottis and of the aryteno-epiglottidian folds is seen in œdematous laryngitis, in acute, submucous, and chronic laryngitis. In *œdema* of the glottis the swelling is below the vocal cords. The swelling may be circumscribed and undergo suppuration. Swelling and œdema is also seen in *perichondritis.*

Tuberculosis. Swelling and infiltration succeeds the primary anæmia or catarrh of the first stage of laryngeal *tuberculosis.* At first there is slight intumescences of tubercular infiltration, not well outlined, and gray in color. They are most frequently found in the interarytenoid space, less often on the false cords and arytenoid cartilages, rarely on the epiglottis.

1. A hill-like prominence between the arytenoid cartilages either in the middle or on one side. In phonation it presses between the cords.

2. When a false cord is affected the whole of it is usually infiltrated, forming a tumor-like swelling which often hides the vocal cords.

3. Vocal cords. Usually only one cord is at first affected. It is thickened and the free border is red. Sometimes the free edge seems split. The infiltration may extend to the subcordal region and cause a hypoglottic laryngitis.

4. Epiglottis. Infiltration of the epiglottis is rarer than œdema after ulceration, and care must be taken not to confound these conditions. The whole epiglottis, or only portions of it, may be affected. It is thickened and curled upon itself, and not freely movable.

5. Arytenoid cartilages. They appear enlarged and puffy, and often fixed from perichondritis.

Syphilis. In *syphilis* we have three forms of swelling:

1. *Mucous Patches.* These are flat elevations of 3 to 7 mm. diameter, oval or circular, and of a whitish-gray color. When the epithelium is lost they appear yellow and purulent. There is no tendency to ulceration, and the patches soon disappear, even without treatment. They occur usually from three to nine months after the infection.

2. *Infiltrations.* Usually these are overlooked, as they produce no symptoms. They are diffuse thickenings in various parts of the larynx, most often on the epiglottis. This may be uniformly thickened or only in part around the edge. The cords may be so swollen as to cause dyspnœa. Usually an ulcerated spot is seen in the centre of the infiltration. The mucous membrane is either normal or reddened. Infiltrations appear three to four or more years after infection.

3. *Gummata.* They appear as round prominences of the same color as the surrounding tissue. They occur on either side of the epiglottis, on the aryteno-epiglottic folds, often in the inter-arytenoid space, on the false cords, and on the under surface of the vocal cords. If they break down, deep ulcers form, leading to extensive destruction of the parts.

Lupus. In *lupus* isolated or grouped nodes are seen flowing together into patches, situated on the epiglottis. The disease is usually present on the face or in the pharynx and mouth. In leprosy the epiglottis is swollen, and nodes from the size of a pin-head to that of a pea are seen on the epiglottis, arytenoid bodies, and false cords.

Fissures. *Fissures* and *erosions* are present in chronic laryngitis.

Ulcers. *Ulceration* is seen in tuberculosis, syphilis, carcinoma, leprosy, and lupus.

Tuberculosis. Ulceration occurs in tuberculosis in the—

1. Inter-arytenoid space. The mucous membranes are notched with irregular projections. When the ulcer is visible it is irregular and of a dirty-gray color.

2. False cords. The ulcers are flat and aphthous, with a pale-white base and a membranous deposit. The mucous membrane sometimes appears sieve-like.

3. Aryteno-epiglottic ligaments. The ulcers are superficial and run lengthwise of the ligament.

4. Vocal cords. The ulcers are either on the upper surface or on the edge of the cords. The former are superficial and seldom destructive. Those on the edge are either small separate ulcers or long ones, affecting the whole border. The circumscribed ulcers occur usually at the posterior portion of the cord and on the processus vocalis. The ulcers of the whole border are often very destructive.

5. Epiglottis. Tubercular ulcers of the epiglottis occur only on its laryngeal side. They are either aphthous and superficial, or deep, and arise from the breaking down of previous infiltration. Sometimes tubercles can be seen at the edge of the ulcers, but they are of no diagnostic value, as similar nodes are seen with non-tubercular ulcers. The epiglottis is usually thickened and œdematous.

Syphilis. Syphilitic ulcers are circular, deep, with a sharp border and inflammatory areola, and overlaid with a whitish-yellow deposit. They develop from an infiltration or a gumma, and not on an unchanged surface. Ulcers on the upper surface of the epiglottis are always syphilitic.

Tumors. Papilloma. The most common form of the benign growths is the *papilloma*. The growth may spring from the true or false cords, the aryteno-epiglottic ligaments, rarely the posterior surface

of the epiglottis. The tumor has a broad base. There may only be one, or it may be multiple, and may vary in size from a split pea to a walnut. Three varieties are met with: 1. Small warty growths, usually on the cords, dark red in color, and seldom larger than a bean. 2. Groups of raised white papillæ on a broad base, also growing on the cords. 3. Large, red, mulberry-shaped or cauliflower-shaped growths, partly villous, partly warty, which fill up the whole larynx.

Fibroma. It appears as a hemispherical, pedunculated tumor of dirty-white, reddish, or dark-red color, more or less dense in consistency. It is usually single, and grows most frequently from the cords. When seen in its smallest size it is known as the "singer's node." It may be as large as a hazel-nut.

Malignant Tumors. In addition to the symptoms indicated in benign tumor, pain and hemorrhage occur. Both *carcinoma* and *sarcoma* are found; the latter is very rare.

Carcinoma. The most common form is the epithelioma, although the medullary and scirrhus have been described. The epithelioma is seen as a circumscribed, hemispherical, warty, or cauliflower-like formation, varying in size, or as a knotty infiltration projecting into the larynx. The medullary form is larger, soft and bloody, and rapidly ulcerates. Scirrhus is firm and hard. The structure of the larynx is gradually invaded, with necroses of the tissues. Perichondritis and abscess frequently ensue.

In carcinoma of the *cords* two kinds of growth are seen.

In the *polypoid* form the tumor develops on the cord like a warty growth, sometimes papillary and of a reddish-gray color. In *diffused* cancer of the cord the structures are red and knotty, and invade the surrounding tissue without distinct demarcation.

Sarcoma. The tumor has a broad base, is shining in appearance, and sometimes lobulated. Sometimes the structure is dark red or yellow.

The Epiglottis. The epiglottis is swollen and red in inflammation of that structure, and may then be palpated with the finger.

Sputum. The sputum from the larynx is generally scanty; it is not frothy, and is colorless and transparent; it is often discharged in small globules; it may be streaked with blood. Sometimes pseudo-membranes are coughed up. It is doubtful if purulent sputum ever comes from the larynx, excepting in cases of perichondritis in which the abscess bursts into the larynx. Laryngeal sputum is found in catarrh and malignant tumors. It is blood-streaked when the catarrh is very intense, or after injuries.

Fever. Fever is present in acute laryngitis and tuberculous ulceration. It is high in acute laryngitis with stenosis; in tuberculosis it is of a hectic type.

Acute Laryngitis.

Acute laryngitis is an inflammation of the larynx, characterized by a sensation of fulness and dryness, with cough, hoarseness, and at times dyspnœa. Several varieties are observed: Simple acute laryngitis,

laryngitis with great stenosis, laryngitis with membrane, laryngitis with spasm.

It is caused by exposure to cold or by the inhalation of acrid vapors. Overstrain, as in singers, excessive use of the voice, particularly in the cold air, may excite an attack. It may be symptomatic of the eruptive fevers, as measles or smallpox, or erysipelas. Its occurrence in the course of chronic diseases must be looked upon with alarm, particularly in cases of Bright's disease, if dropsy is present in other situations.

The attack begins with a feeling of chilliness, followed by *fever* of varying degree, but usually mild. The patient complains of a feeling of pressure and dryness in the larynx, or as if a foreign body were present. Some *pain* gradually develops in the height of the attack, never so severe as to require an anodyne. From the first there is *cough*. It is dry and hacking, and slightly painful. In the more intense forms the cough is continuous, disturbing the patient night and day. Paroxysms occur when the patient speaks or takes food. First the cough is dry; within a short time it becomes moist, and expectoration of clear, transparent mucus takes place. The mucus may be tinged with blood. At the end of forty-eight hours expectoration becomes more yellowish and opaque. The voice may be merely hoarse, or may be lost entirely. Sometimes *aphonia* without general symptoms occurs in acute laryngitis. In *laryngitis sicca* cough and dyspnœa occur in paroxysms and are not relieved until a dry secretion is coughed up. The paroxysms take place at night or in the early morning, and may cause retching and vomiting. It is seen in adults.

Acute Laryngitis with Stenosis No doubt some of the cases of so-called membranous croup in children are cases of acute laryngitis, with swelling and occlusion of the glottis by congestion and by tough secretion. Œdema may or may not be present. The attack begins with catarrhal symptoms. The child is languid, refuses to eat, is thirsty and has some chilliness and rise of temperature. With the slight cough, which may be shrill, there are hoarseness and some difficulty in breathing, but no pain on swallowing. On the second day, or after the lapse of four or five days, during which time mild fever continues, the catarrhal symptoms become more marked. The voice is more hoarse or may be suppressed. The harsh, clanging cough becomes toneless, and soon the sound is suppressed. Dyspnœa is most severe, and the aspirations are hurried and noisy, attended by loud whistling inspiration, and snoring expiration. The stenosis is inspiratory, and during the day or in the succeeding twenty-four hours may become very intense. It is attended with violent efforts at breathing and the occurrence of cyanosis in its most aggravated form. The larynx moves up and down, the head is thrown back. There is recession at the root of the neck and along the margins of the ribs and the epigastrium. The lower portion of the sternum may be drawn in. Duskiness of the extremities and of the lips is observed as the stenosis becomes more marked, finally deepening into cyanosis. It may be relieved from time to time by removal of the obstruction, which occurs after cough, vomiting, or change of position. A paroxysm soon recurs. With each paroxysm lividity becomes more and more marked, the res-

pirations continued hurried. The face becomes pale, the extremities cold, and a cold sweat bathes the brow. Restlessness is characteristic. The child tosses about in the bed or from the bed to the arms of the nurse. The heart's action is increased each hour in frequency as the stenosis advances, and becomes weaker. As exhaustion ensues and the symptoms of obstruction become more marked, stupor deepening into unconsciousness develops. Convulsions may occur at the end. The attacks rarely recur if the patient once recovers. They follow exposure to cold.

If recovery takes place, the child usually becomes more free from dyspnœa, the cyanosis fades, and the restlessness disappears. A prolonged sleep follows relief, although the voice may remain hoarse or suppressed, and the cough continue many days.

Laryngeal Diphtheria. The same symptoms are seen in membranous croup and laryngeal diphtheria. In the latter affection there may be a history of exposure or of infection. At the commencement of the attack the diphtheritic patches may be seen in the fauces or nares. If a membrane can be secured and a bacteriological examination made, the diagnosis of diphtheria with stenosis is positive. Enlarged glands in the neck, with marked physical depression, a moderate degree or entire absence of fever, and the occurrence of early albuminuria, also point to diphtheria. The distinction between the two affections is nevertheless quite difficult, and as long as there is a shadow of doubt, for prophylactic reasons the case should be considered one of diphtheria.

Acute Laryngitis, with Spasm. False Croup or Spasmodic Laryngitis. In children, in addition, another form of laryngitis associated with spasm of the larynx is seen. The catarrhal symptoms are mild, so that the child seems to be well during the day. Fever is absent, and a slight cough or huskiness alone calls attention to the larynx. After the first three or four hours of quiet sleep the child suddenly awakes with a barking cough, sits up and struggles for breath. The dyspnœa continues from a few minutes to an hour or so, gradually lessening, to disappear entirely as the child lapses into sleep. Throughout the next day the child seems as well as on the previous day, and the succeeding night is again seized with another attack of "croup." This may occur once or twice during the night. It seems to be influenced by the weather. Damp days and an east wind are provocative of an attack. It recurs frequently during the same season.

Œdema of the Larynx.

This condition arises in the course of acute laryngitis; frequently occurs in chronic diseases of the larynx, particularly if ulceration is present; and as a complication of erysipelas and diphtheria. In some cases of Bright's disease it may develop suddenly.

In the course of the above-mentioned disease symptoms of laryngeal stenosis may occur suddenly. The voice becomes husky and suppressed, the dyspnœa is very extreme, so that in a few hours grave symptoms of obstruction arise. There is no cough. The patient complains of the sensation of a foreign body, and tries to grasp it.

The Diagnosis of Acute Diseases of the Larynx.

Acute affections of the larynx are distinguished from other diseases without much difficulty. To recognize the various forms of acute laryngitis, however, is not easy. In all there is laryngeal stenosis to a certain degree, and practically the question to answer is, Which form of stenosis is present? The accompanying table shows the differential points for diagnosis. It is seen that the age, occurrence of previous attacks, the character of the general symptoms, the existence of previous laryngeal disease, the association of faucial disease, the presence or absence of membrane, and the results of laryngoscopic examination must be considered before making a positive diagnosis.

Simple Acute Laryngitis.—"Catarrh of Larynx."	*Acute Laryngitis with Stenosis.*
Gradual onset of laryngitis, with dyspnœa very slight or absent.	Gradual onset of laryngitis, but dyspnœa develops to great severity.
All ages.	Children.
Fever of varying degree.	Fever of varying degree.
Dry irritating cough.	Dry cough, often paroxysmal.
May be hoarseness.	Hoarseness.
Pharynx reddened.	Pharynx reddened.
Gradual increase and decline.	Gradual increase, and either death of patient or decline of dyspnœa.
Larynx red and slightly swollen, as seen by laryngoscope.	Same, but swelling much greater.
Acute Laryngitis with Spasm.—Spasmodic Croup.	*Laryngismus Stridulus.—"Child-crowing."*
May be slight hoarseness or cough, or none. Suddenly, in night, child wakes with intense dyspnœa and crowing inspiration.	No laryngitis. Sudden attacks of dyspnœa with crowing inspiration, either day or night. Very severe. May be general convulsions.
Children.	Children or hysterical adults.
Temporary high fever.	No fever.
Slight brassy cough during day.	No cough.
May be slight hoarseness in day. Very hoarse in attack.	No hoarseness.
	Occurs often in rhachitic and hysterical cases.
Lasts a few minutes to one hour. May recur, or no attack until next night.	Ends suddenly, in at most two minutes, and occurs often.
Slight redness, or nothing seen by laryngoscope.	Nothing seen in larynx.
Œdema of Larynx.	*Membranous Laryngitis.—Croup; Diphtheria.*
Some inflammatory disease of larynx exists.	Epidemic.
Rapid development of dyspnœa, increasing to great severity.	Gradually developing hoarseness and croupy cough, with low fever and lassitude, then development of dyspnœa, gradually and without intermission, as a rule.
All ages.	Children
Depends on cause.	Low fever and depression.
No cough.	Croupy cough, later suppressed.
No hoarseness.	Very hoarse.
	Fauces red and often with membrane; albuminuria; paralysis.
Increases steadily to climax, then death, or decline of dyspnœa.	Increases steadily, broken by intense paroxysms. Either death or gradual improvement.
Epiglottis and aryteno-epiglottic folds swollen, pale, and waxy.	Red, swollen, with membrane.

Foreign Bodies.	*Pertussis.—Whooping-cough.*
During eating or while holding object in mouth sudden dyspnœa, varying in intensity according to object.	Epidemic.
	Bronchitis, with cough developing in from one to three weeks. Then dyspnœa caused by severe paroxysm of coughing —absent between them.
All ages.	Children.
No fever.	Only the fever due to bronchitis.
Irritative, expulsive cough.	Intense paroxysm of coughing.
May be hoarseness or not.	No hoarseness.
	Hemorrhages in various places from strain or emphysema.
Cough persists till removal of body, or occasionally the larynx becomes accustomed to its presence, and cough ceases.	May be death from exhaustion, or gradual improvement.
See the foreign body.	Nothing seen, unless slight laryngitis.

Acute Submucous Laryngitis. The inflammation extends to the submucous cellular tissue. It arises in the course of acute laryngitis, and is the form seen in traumatism, or from burns and scalds. The symptoms are those of intense laryngitis, with stridor. They increase in severity until stenosis arises. If the under surface of the cords is affected, death will occur from asphyxia. Sometimes the inflammation is circumscribed and is followed by development of an abscess.

The *chronic form* of *submucous inflammation* of the larynx is usually seen in drunkards, and is recognized usually by the laryngoscopic examination. The symptoms are those of slight stenosis.

Paralyses of the Laryngeal Muscles.

They are divided for convenience into groups. The *symptom* is dysphonia, which, with laryngoscopic appearances, leads to the recognition of the paralysis.

1. Paralysis of the Tensors of the Cord. The crico-thyroid muscle is paralyzed; the superior laryngeal nerve which supplies the muscle is concerned. The *voice* is deep and rough, and incapable of producing high tones. Usually, the whole nerve is involved, and the result is *anæsthesia* of the larynx and *paralysis* of the *epiglottis*.

Laryngeal Examination. The *epiglottis* is fixed, and falls back against the tongue. The *glottis opening* is a wavy line.

Causal disease. The condition described occurs almost exclusively after diphtheria.

2. Paralysis of the Closers of the Glottis, or Adductors of the Cords. The muscles involved are the crico-arytenoideus lateralis, arytenoideus transversus, and the thyro-arytenoideus internus and externus. The nerve is the recurrent laryngeal.

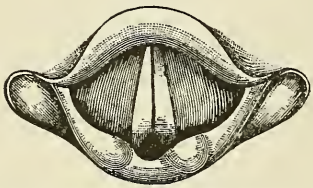
The symptoms are *complete aphonia*, coming on suddenly, and often disappearing as suddenly.

Laryngeal Examination. During phonation the cords remain in the inspiratory position. The paralysis may affect one or both sides.

Sometimes the arytenoideus transversus alone may be affected. Then there is hoarseness or aphonia. The anterior portions of the cords come together in phonation, but the posterior portions do not, leaving a triangular opening posteriorly. (See Fig. 118.)

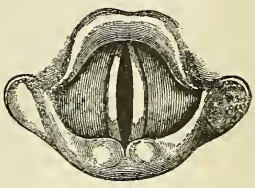
Or, the thyro-arytenoideus internus alone may be affected. There is then dysphonia or aphonia, as before, but the cords come together at both extremities and remain apart in the middle, forming an oval opening. (See Fig. 119.)

Fig. 118.



Paralysis of the arytenoideus transversus in phonation. (Gottstein.)

Fig. 119.



Paralysis of the thyro-arytenoideus internus in phonation. (Gottstein.)

Causal Disease. These paralyses occur in hysteria, catarrh, or severe overstrain of the voice.

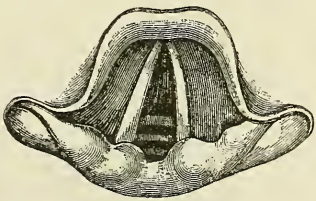
3. Paralysis of the Openers of the Glottis, or Abductors of the Cords. The muscle affected is the crico-arytenoideus posticus, and the nerve is the recurrent laryngeal.

Symptoms. When one side is affected the respiration is free, but there is stridor or forced inspiration. The voice is harsh.

Laryngeal Examination. One cord remains in the middle line. (See Fig. 120.)

When both sides are affected there is gradually developing inspiratory dyspnœa with stridor. The voice is nearly normal.

Fig. 120.



Paralysis of the left recurrent nerve; inspiration. (Gottstein.)

Laryngeal Examination. The glottis is a narrow cleft which becomes still narrower on inspection.

Complete Paralysis of the Recurrent Laryngeal Nerve. Symptoms. Unilateral Paralysis. A weak, toneless voice which breaks into a falsetto when the patient endeavors to speak loud.

Laryngeal Examination. The cord and arytenoid body are in the cadaveric position—viz., half-way between the phonating and the inspiratory positions. In phonation the other cord passes beyond the middle line, and the glottis is slanting. The edge of the paralyzed cord is excavated.

Bilateral Paralysis. Aphonia and inability to cough and expectorate.

Laryngeal Examination. Both cords are in the cadaveric position and their edges excavated.

The adductors are usually paralyzed before the abductors, and one can see all the intermediate stages by close watching.

Causal Disease. The conditions which give rise to the paralysis are numerous. It may arise from simple catarrh or from hysteria. More often it is due to pressure on the vagus or recurrent laryngeal, or some disease affecting these nerves or their roots.

The causes of pressure are: Aneurism of the subclavian or aorta, mediastinal tumor, tubercular bronchial glands, the apex of a tubercular lung, cancer of the œsophagus, goitre, or carcinoma of the pleura.

The diseases are: Diphtheria, tumor, softening or hemorrhage into the brain, bulbar paralysis, neuritis, typhus, cholera, variola, articular rheumatism, toxæmia (?), sclerosis of the cord, progressive muscular atrophy, and paralytic dementia.

Tumors of the Larynx.

Both benign and malignant growths are seen. At first *dysphonia* or *aphonia* takes place. The impairment of voice may continue for a long period of time before *dyspnœa* arises. This develops very gradually, and in some few cases is attended by an irritative *cough*. The general symptoms are not marked in benign cases. In the malignant forms they are pronounced, but characterized by the development of cachexia later than in carcinoma elsewhere.

The *diagnosis* of malignant disease of the larynx is based upon the association of symptoms of laryngeal disease with pain, and with the characteristic appearances found on inspection, on its occurring after the middle period of life, and lasting from six to nine months only, with the development of cachexia and emaciation without fever. Enlargement of the cervical glands points to cancer. Simple and syphilitic perichondritis must be excluded.

Tuberculosis of the Larynx.

The existence of primary laryngeal tuberculosis is doubtful. It cannot be proved clinically, and the majority of cases, at least, are secondary to tuberculosis of the lungs. The manifestations of tuberculosis of the larynx may be either a simple persistent catarrh, an infiltration, or an ulceration. (See pages 439 and 440.) The symptoms vary according to the lesion.

a. Catarrh. There is a slight hoarseness and the voice tires easily. Often paræsthesia or peculiar sensations in the larynx are present. Cough, when due to this alone and not to the process in the lungs, is short and dry.

b. Infiltration. At first the symptoms are those of simple catarrh, then the alteration of the voice increases even to aphonia; there is a feeling of dryness or soreness in the larynx, and dysphagia. The cough is very slight and is usually wholly disguised by the cough due to the disease in the lungs. There is some difficulty in expectoration.

c. Ulceration. The symptoms are the same as those of infiltration, but the dysphagia and pain are greater.

Diagnosis. Tuberculous ulcer occurs most frequently in the male sex, and during the period ranging from eighteen to thirty years of age. If the symptoms develop in the course of phthisis, or in case that affection cannot be recognized, if there is a history of infection, or exposure, and if bacilli are found in the sputum, the diagnosis is not difficult. A portion of the diseased mass may be removed for microscopic examination or inoculation. In examining the secretion for tubercle bacilli, it is to be remembered that the exudation may have been brought up from the lungs. The examination in cases of phthisis is of little practical value, except to determine whether the ulceration present may be syphilitic and grafted upon a tuberculous disease of the lungs. Enlargement of the glands of the neck is often present, but is not diagnostic.

Fever is present, and, indeed, may be an important diagnostic feature in doubtful cases. The temperature should be taken every two hours, for the morning or evening exacerbations may not be present. Emaciation ensues, and sooner or later the hectic phenomena and signs of tuberculosis in other structures arise. When tuberculous ulceration of the larynx occurs in the course of local pulmonary tuberculosis the disease runs a much more rapid course.

The laryngeal symptoms are not diagnostic. Pain may be the most distinct. The appearances observed by the laryngoscope are more characteristic. Local anæmia with paræsthesia, paresis of the cords, and short cough, or an obstinate diffuse catarrh, are suspicious symptoms. The peculiar ridged infiltration between the arytenoids is almost invariably tubercular.

Isolated thickenings anywhere in the larynx that taper off gradually into the normal tissue can only be tuberculous or syphilitic. The regularity and number, with anæmia and lack of inflammatory signs, will usually distinguish the tuberculous from the syphilitic. The ulcers are non-erosive. Syphilitic ulcers do not often occur, except on the edge and lingual side of the epiglottis and on the cords. They extend more rapidly than the tuberculous, and may be continuous with ulceration in the pharynx. The area of ulceration may extend to the base of the tongue, which is very infrequent in tuberculous disease. In syphilitic ulceration scars or cicatrices are seen, but they are absent in the tuberculous form. Laryngoscopic examination in tuberculous ulceration is difficult, as it causes great pain ; in syphilis comparatively little pain attends examination. (See the Infections.)

Syphilitic Affections of the Larynx.

Mucous patches, papules, infiltrations, or gummata may be present in the larynx for some time without exhibiting any symptoms. Usually a change in the voice is the first symptom noticed, due either to the catarrh or to ulcers, scars, infiltrations, or gummata affecting the cords. There is often a feeling of pressure or a tickling sensation. Pain is not usual, and, when present, is very slight. Dysphagia

occurs only when the epiglottis is extensively ulcerated. There is little or no cough.

The *diagnosis* rests upon the history of infection, the objective signs of syphilis indicated by pigmentation or recent eruption, scars, periostitis or nodes on the bone, and enlarged glands. The laryngeal symptoms are not diagnostic, save that pain is absent in spite of extensive ulceration, while difficulty of deglutition, on account of food entering the larynx, is of frequent occurrence. The laryngoscopic appearances, as indicated above, are characteristic of this affection. In obscure cases the distinctions spoken of in tuberculosis are of diagnostic value.

Although the patient may be broken down and cachectic the febrile range is not high, unless perichondritis occurs, or pneumonia sets in, on account of food in the air-passages.

The Larynx in Other Diseases.

Laryngeal symptoms due to lesions of the nervous system are found under the following circumstances. (See Cerebral Localization.)

Cerebral Hemorrhage. 1. Aphasia. The movement of the muscles is normal, but they cannot be controlled by the will. Caused by hemorrhage in the cortex or along the course of connective fibres.

2. Recurrent paralysis. Due to hemorrhage in the medulla.

3. Symptoms of bulbar paralysis. Same cause.

Encephalomalacia. (Softening.) When in the brain, aphasias result; when in the medulla, bulbar symptoms.

Tumors of Cerebrum. The symptoms are, according to location, aphonia, aphasia, or paralysis of the cords.

Bulbar Paralysis. We have, of course, the other symptoms of the disease. The voice becomes weak and monotonous without modulation. High tones are impossible. It progresses to hoarseness and finally aphonia. Particles of food and drink enter the larynx. Paresis or paralysis of the cords.

Multiple Sclerosis. The speech is low, uncertain, and scanning, later hoarse. Laughing and crying are accompanied by peculiar yawning inspirations. Laryngoscopical examination: Slight paresis of the cords is seen.

Posterior Sclerosis (*Tabes*). The muscles act very slowly. Sometimes symptoms of irritation, as tickling or burning in the larynx, with a dry cough, occasionally severe paroxysms of coughing, even to spasm of the larynx, occur. "Laryngeal crises." In rare cases a phonetic spasm has been observed. Less often paresis or paralyses of the various muscles occur, most frequently the posticus, next the recurrent. Sensibility may or may not be disturbed.

Amyotrophic Lateral Sclerosis. There is a mixture of bulbar with spinal symptoms. (See Sclerosis.)

Progressive Muscular Atrophy. The same mixture of symptoms occurs very late.

Paralytic Dementia. There may be disturbances in articulation, with paresis and paralysis of the cords.

Chorea. There may be a tremor of the cords from under-tension, but probably no true choreic movements.

CHAPTER II.

DISEASES OF THE LUNGS AND PLEURÆ.

The lungs are composed of a relatively small amount of tissue. They are made up of tubes and canals. The tissue which composes the structure of the lungs independently of the canals, the connective tissue, is liable to the same morbid processes that affect it in other situations. But, curiously, it is not often subjected to irritants which cause acute inflammation, while chronic inflammations occur secondarily, in the large majority of cases, to processes in the channels. Diseases of the lungs are really the disease of its channels, and the symptoms that arise are due to morbid alterations of them (1) by processes common to the structure of such channels and (2) by obstruction of them. There are three sets of channels: First, for the passage of air; second, for the flow of blood; and, third, for the flow of lymph. The symptoms, therefore, are due to the morbid process or to obstruction of the channels just mentioned.

Physical Classification. The various affections of the lungs occur without any change in the volume of air in the lungs, or are attended by an increase or diminution in the amount of air.

I. Diseases with Normal Amount of Air.

Affections of the Bronchial Tubes, except Asthma.

II. Diseases with Increased Amount of Air.

Enlargement of the Chest. The enlargement with increased amount of air may be unilateral or bilateral. It seems paradoxical that the more air there is in the thorax, the greater is the need for air, and hence the occurrence of dyspnœa.

1. Asthma.
2. Emphysema.

III. Diseases with Diminished Amount of Air.

A. The Consolidations. The consolidations may be local, unilateral, or bilateral.

1. The congestions.
2. Pulmonary embolism and thrombosis.
3. Pneumonia.
4. Bronchopneumonia.
5. Chronic interstitial pneumonia.
6. Pulmonary tuberculosis.
7. Abscess of the lung.

8. Gangrene of the lung.
9. Collapse of the lung.
10. Cancer and other new growths of the lung.
11. Hydatid disease of the lung.

B. Diseases of the Pleura.
1. Diminished amount of air from inhibition of movement, on account of pain.
2. Diminished amount of air from the physical condition within the thorax.

The Morbid Processes.

Affections of the lungs may be divided into the neuroses, the congestions, the inflammations, the degenerations, the morbid growths and gross parasites. Influences operating through the pneumogastric and phrenic nerve may be responsible for respiratory neuroses. The congestions are so intimately associated with vascular phenomena that the latter may be included in the process. The inflammations are limited to the bronchi, to the alveoli, and to the connective tissues surrounding both. The intimate relation of the small bronchi, the alveoli, and their surrounding connective tissues implies their conjoint involvement in many processes.

A. The Neuroses.
B. The Congestions.
1. Active, including *hemorrhagic* infarct.
2. Passive.
Subsidiary: *hemorrhage.*
C. The inflammations, chiefly *infectious.*
1. The Bronchi.
Acute.
Chronic.
2. Bronchi and alveoli.
Bronchopneumonia (an infection).
3. Bronchi, alveoli, and connective tissue.
Pneumonia.
Tuberculosis.
Abscess of the lung.
Gangrene.
Chronic interstitial pneumonia—pneumonokoniosis.
Syphilis of the lung.
D. The Degenerations.
Emphysema.
Bronchial dilatation.
E. Morbid growths.
F. Gross Parasites.
Hydatid disease.

Symptoms Due to the Morbid Process. The air-tubes are lined with mucous membrane, which is subject to morbid processes that attend any such lining—*congestion*, or acute and chronic *inflammation*

—with a flux as the characteristic symptom. The muscle and elastic tissue of the canal become involved in the process. The former undergoes spasm, with or without mucous membrane inflammation (asthma). Grave consequences do not arise until degeneration takes place, then the power of confining the air or driving it out is lost, and emphysema results.

In the blood-canals, hyperæmia (congestion), embolism and thrombosis, and secondary œdema take place; in the lymph-canals, inflammation (acute and chronic pleurisy), and transudation (hydrothorax or hæmothorax). Now, the symptoms that arise in each or all of the above processes—pain, local discomfort, mucous or purulent discharge, serous or purulent exudation, and fever—are not different from those which are found in diseases of similar tissues in other localities. (Compare with affections of mucous membranes in other organs or of serous membranes).

Symptoms Due to Obstruction of Channels. In addition to these, however, there is a group of symptoms due to obstruction of the various channels, and hence, interference with the function of the lungs. The symptoms are purely mechanical.

1. Dyspnœa occurs from obstruction of either the bronchial tubes or bloodvessels in addition to causes mentioned below. It is as pronounced in asthma or capillary bronchitis as in embolic obstruction (fat-embolism) or congestion and stasis in the bloodvessels. It occurs when the canals are occluded by extrinsic causes—foreign bodies in the bronchi or pleural effusions.

2. Cyanosis. As a sequence of the above symptoms we have another vivid picture—the development of cyanosis from interference with aëration.

Symptoms Due to Altered Muscle or Nerve Mechanism. Other structures (the bony thorax and its muscles) are required for the performance of the function of the lung, the aëration of blood. Of these we have more particularly: first, muscles, to hasten the movement of the air; and, second, a nervous mechanism to control the movement of the muscles. 1. Inactivity of the former, from pain, from debility, or from paralysis through disease of the nerves, practically occludes the canals, for the normal contents slacken or cease their movement, and therefore the amount of air is lessened—hence dyspnœa. 2. The nervous mechanism not only controls the large muscles of the exterior, through a centre stimulated or depressed by various influences, chiefly the blood, but also receives and sends impressions to the muscles of the tubes, giving rise to (*a*) *cough* or (*b*) *bronchial spasm* with dyspnœa. This nervous mechanism by its centre of control is in relationship with higher and lower centres, and the nerve that connects it with the bronchial tubes supplies other organs or anastomoses with other nerves. Hence, we may have: *A*. A *central affection*, causing pulmonic symptoms from the following causes: 1. Because higher centres influence the lower pulmonary centre, as we see in hysterical cough, or emotional cough, and in asthma—*respiratory neuroses*. 2. Disease affecting the region of the centre, as in tumor or in bulbar or glosso-labio-laryngeal paralysis. 3. Irritants acting upon the centre, as urea, exciting uræmic asthma. *B*. An affection of the

nerve-trunk, as from the pressure of an aneurism or morbid growth. *C. Reflex influences* through the pneumogastric and correlated nerves. The asthma of nasal disease, or of peripheral irritation, and reflex cough (neuroses) are of this nature. *Corollary:* Lung symptoms, chiefly dyspnœa and cough, may be due to local causes (affections of the muscles), or to causes at a distance, operating directly through the pneumogastric centre, or the nerve-trunk, or by anastomoses in a reflex manner. The practical deduction is to look further than the lungs in the investigation of pulmonic symptoms. Lung symptoms are not often expressive of disease in other parts, nor are diseases of the lungs symptomatic of disease in other organs.

Affections of the Pleura. In diseases of the *pleura*, one side is usually affected. Simple inflammation and inflammation with exudation into the pleural cavity occur. In both forms there is diminution of movement, and hence less air entering the affected lung, although the cause is different in each case. In *acute inflammation*, the diminished amount of air is for *physiological* reasons: the movement of the affected side is inhibited by pain—hence diminution of expansion and lessened ingress and egress of air. Enfeeblement of breath-sounds and fremitus, with diminished expansion, alone indicate the diminution. On the other hand, in acute inflammation *with exudation*, the amount of air is diminished for *physical* reasons. The effusion encroaches upon and causes diminution of the air-space, and hence lessens the amount of air. It will be remembered that the physical signs of diminution in the amount of air from effusion are quite distinct from the physical signs due to consolidation.

The Lungs and Heart. The relationship of the pulmonary vascular channels to the remainder of the circulation is very close. Overfilling of the pulmonic bloodvessels, and hence dyspnœa, may be due to alterations or changes in the central pump, the heart; or in the vessels between—as from the pressure of an aneurism. The nature and importance of lung symptoms cannot be appreciated without an investigation of the heart and the blood-ways. Many pulmonic congestions are due to dilatation of the heart, and are relieved by digitalis. At the other end of the beam, it may be noted that lung diseases cause heart disease; from backward pressure of blood-columns in overdistended vessels, a dilated right heart follows.

Space forbids tracing out the effects of the blocking of channels, but it is suggestive that all the aëration of the body takes place through the first set of tubes, that all the blood of the body passes through the second, and that the third is an enormous drainage-area of lymph. The student can readily appreciate how profoundly diseases of the lungs must affect the general system. Apart from the nerves, the tie that binds the other organs to them is the blood. In proportion as the lungs enrich them with oxygen, the other organs act with vigor. Imperfect oxygenation soon causes diminution of all function, with the secondary effect on the blood of the production of *anæmia*, which, with its long train of symptoms, is seen in all chronic lung affections.

Relative Value of Subjective and Objective Symptoms. The subjective symptoms are few, and, as will be seen later, are common

to so many pulmonary diseases that they are of little diagnostic value. The objective symptoms are more decisive, and the laws of physics as applied to the lungs aid in the distinction. The effect of the occlusion of channels is mechanical or physical, and hence a physical change in the lung follows. The objective symptoms occur (1) because of the physiological movement of air. Sound attends the movement of air in health; if the air-movement is checked, no sounds occur, or abnormal breath-sounds and new sounds (râles) are created. They also occur (2) because of physical changes in the structure. Air is replaced by solid structure; the physical condition of the lung changes. The objective signs of these conditions are determined by inspection, palpation, percussion, and auscultation.

Diagnosis. The diagnosis of disease of the lungs is attained by the collection and consideration of data obtained both by inquiry and by observation. By observation the objective phenomena are secured, first, by physical examination; second, by an examination of the sputum; and, third, by an examination of the fluids secured by puncture.

It is not usually difficult to distinguish diseases of the lungs from affections of other structures. It is true, pleurisy and pleurodynia are often distinguished with difficulty. We are called upon, also, to decide between pleurisy and subdiaphragmatic inflammation, a pleural and hepatic inflammation, a pleuritis and pericardial inflammation, and between cardiac and pulmonary disease, especially when both are present and it is desirable to determine which is the primary affection. The contiguous relations of the organs make this necessary, and with care in ascertaining the history and the subjective and objective symptoms the distinction may not be difficult.

In chronic disease, affections of the lungs, of the mediastinum, and of the great vessels must be distinguished from one another. An aneurism or mediastinal disease may simulate chronic phthisis.

Infections. It often happens in a pulmonary disease that some of its pronounced symptoms may strongly point to an infection other than that of the lungs; thus the cerebral symptoms of pneumonia may be held to be due to meningitis, or the fever thought to be due to typhoid fever. On the other hand, the presence of a pulmonary affection, as tuberculosis, may explain the nature of the morbid process in other organs or structures. Hence, in all cases in which there is a possibility of secondary tuberculosis the lungs should be examined to determine if they are the seat of the primary disease. In this way the true nature of a meningitis, a peritonitis, or other tubercular affection may be recognized. So, too, in secondary anæmia and in protracted debility of unknown source the lungs should be examined. It must be borne in mind also that in chronic diseases, as chronic renal disease, chronic arthritis, diabetes, etc., pulmonary tuberculosis may set in most insidiously. In the same class of diseases pneumonia is frequently a terminal infection, and likewise runs an insidious course. Finally, in the extremes of life pulmonary infections, as pneumonia, present symptoms out of the usual run. In infancy and childhood the cerebral symptoms may mask the pulmonary symptoms; in senility the ab-

sence of cough or expectoration may lead to the dismissal of all thought of pulmonary disease. In short, the lungs should be examined in all affections.

This injunction is particularly to be observed, as lung diseases are often secondary to other diseases; phthisis, to tuberculosis elsewhere, pneumonia or pleurisy to all infectious disorders, to Bright's disease, cancer, and diabetes. Above all, the possibility of a hydrothorax, secondary to causes of transudations, must be borne in mind.

The Data Obtained by Inquiry.

The Social History. A glance at the various processes which take place in the lungs readily lead one to infer the social history.

Age. In the earlier and later periods of life bacterial invasion is more likely to take place; hence, at these extremes streptococcus and pneumococcus infections are common; tuberculosis, on the other hand, is more common in early adult life, although it does not respect age. The degenerations are more common later in life, as we may say of the morbid growths, both obeying the usual rules concerning the course of these processes. The *sex*. As the infections predominate and as one at least is more liable to develop in those whose resistance is lessened, it follows that tuberculosis is more frequently seen in the female sex. That sex which follows occupations compelling the inhalation of irritating particles—the male—is more liable to have fibroid and other inflammations of the lungs.

The Occupation. From this we gather little of diagnostic value, save that the chronic inflammations are more prone to occur in those who inhale solid particles, as miners, stone-cutters, etc., while tuberculosis attends those whose occupations are debilitating and require in-door duties. Nor does a knowledge of the *habits* lend much aid save as they depress the system and render it more vulnerable to bacterial action. It is needless to say clothing, exposure, residence, and the diet may be hygienic factors in the life of the patient. The amount of exercise, etc., must be inquired into in each case.

Infections. It is readily seen, however, that the facts in the social history of diagnostic importance are just those facts which are predisposing factors in many infectious disorders. Most lung diseases are, therefore, correlated in their antecedents with the infections. It must be borne in mind it is always well to trace the source of the infection if possible.

The Family History. Heredity plays a serious part, and hence the family history should be sought for, particularly in the study of those affections which are of tuberculous origin. The tendency of this infection to follow in successive strains is well known. In like manner we inquire in cases of asthma and other neuroses for evidence of their occurrence in previous generations—a well-known clinical fact. Then emphysematous changes seem to be a peculiarity of certain families.

The Occurrence of Previous Diseases is to be inquired for. Pneumonia is likely to be followed by other attacks. Pleurisy is

related to, and may be an expression of rheumatism; it may be preceded by other rheumatic phenomena; it may be the earliest expression of tuberculosis, and may precede the latter by two or more years, an interval of health separating the two. Then it must be borne in mind pulmonary tuberculosis may succeed a long antecedent joint or glandular tuberculosis—a history of which should be inquired for. The state of the circulation should be studied, and the occurrence of previous heart disease sought for. In affections of the pleura we must inquire for previous infections and note the presence or absence of disease of contiguous structures, as the ribs and muscles of the chest and the viscera below the diaphragm.

The Subjective Symptoms. Dyspnœa. Dyspnœa, in its true sense, means difficult breathing. The respirations are deeper than natural, but of normal frequency, or they may only be more frequent than they should be, or they may be both deeper and more frequent. The patient is usually conscious of suffering or of some distress in breathing. *Lung disease* without *dyspnœa:* While a common, indeed almost constant symptom of lung disease, it does not follow that because a patient has extensive disease of the lung he need suffer from difficult or hurried breathing. This is because the system requires no more air than the capacity of the lung is able to supply. The change takes place very gradually, but many persons with chronic fibroid phthisis, or with emphysema, in both of which the disease may be extensive, may not have dyspnœa, unless an unusual demand is made upon the system. The subjects are under-weight, move slowly, and otherwise show that they are deprived of an essential to active being.

Varieties of Dyspnœa Depending upon Cause.

I. Anything which cuts off or lessens the normal amount of air required for oxygenation of the blood. A. Obstruction of the air-passages. B. Diminution of air-space from causes within and outside of the thorax. C. Interference with the action of the muscles concerned in breathing.

II. Affections which lessen the amount of blood, as obstructive heart disease. Rarely, tumors pressing upon the bloodvessels.

III. Affections in which the red blood-corpuscles are diminished—anæmia.

IV. Pulmonary embolism and thrombosis. In cases of weak heart the vessels become occluded. After labor a clot of blood may escape from a uterine sinus, be carried to the right heart, and thence to the pulmonic veins. The clot may arise from inflammation of the veins in any situation.

V. Fat-embolism. Foreign substances in the blood, as fat, occurring in parturient women three or four days after labor, after fractures, and in diabetes.

VI. Dyspnœa due to interference with the nervous mechanism of respiration. *a.* Tumor, hemorrhage, or degeneration about the respiratory centre in the medulla. *b.* Irritation of the centre by toxic agents, as in uræmia, diabetes, auto-intoxication from gastro-intestinal disorder. To this class belongs "heat dyspnœa," which occurs in all febrile conditions. The warm blood acts as a direct irritant to the

respiratory centre in the medulla oblongata (Landois). This explains the dyspnœa of fever and the curious fact pointed out by Cohnheim, that the respirations in pneumonia lessen as soon as the fever disappears, notwithstanding the persistence of the physical condition, which may have accounted for the dyspnœa. Reflex dyspnœa (asthma, *q. v.*) belongs to this variety. The dyspnœa of hysteria is of the same class.

Anything which cuts off or lessens the normal amount of air required for oxygenation of the blood causes more or less dyspnœa.

A. Obstruction of the Air-passages.

1. Occlusion of the nares, unless compensated by mouth-breathing.

2. Enlargement of the tonsils, retropharyngeal abscess, or any obstruction in the throat, from diphtheritic or œdematous swelling.

3. Disease of the larynx, causing stenosis, also causes a characteristic form of dyspnœa known as *inspiratory dyspnœa.* (See Disease of the Larynx.)

4. Obstruction of (*a*) the trachea or (*b*) the bronchus from external pressure or from a foreign body. It must be distinguished from dyspnœa, the origin of which is higher up in the air-passages, by careful inspection.

a. Tracheal Obstruction. In this form of dyspnœa there is no increased movement of the larynx. There is no change in the voice, except that it may be weakened, and the sonorous quality diminished. The voice will be modified, however, if there is at the same time disease of the larynx from syphilis, or paralysis of the muscles from pressure on the recurrent laryngeal nerves by the same cause as the tracheal stenosis. If so, on laryngoscopic examination the tumor pressing upon the larynx can be seen at times, especially if the larynx is healthy.

Expert operators can secure quite an extensive view of the windpipe, particularly if the head is bent slightly forward and the patient is seated in the upright posture. A mirror must then be placed against the soft palate, with the surface more horizontal than usual. By this means an aneurism may be seen bulging into the trachea. It must not be mistaken for pulsation of the lower end of the trachea, due to transmission of the impulse of the aorta to the trachea, which has been shown to occur in healthy persons.

The dyspnœa is *expiratory,* and is never so extreme as in laryngeal stenosis. The lower ribs are therefore not sucked in during inspiration until late in the disease. A stridor attends the dyspnœa, which is heard with the stethoscope over the trachea, as well as over every part of the chest. Sometimes a point over the trachea can be determined at which the sound is heard loudest. The point may indicate the seat of a stenosis. Sometimes the sound is more marked over the larynx than over the sternum, when the lower part of the trachea is obstructed. Demme has pointed out that in cases of prolonged obstruction in the lower air-passages the upper portion of the thorax may diminish in size. Not only is the dyspnœa constant, but paroxysms may take place in which the distress is very severe. These paroxysms of dyspnœa may be due to spasm of the vocal cords; but it is very likely that they are due, as Bristowe has shown, to swelling of the mucous mem-

brane, or to mucus which has accumulated at the point of obstruction and cannot be dislodged, or to spasm of the muscular tissue of the trachea itself. In addition to the subjective symptom of want of breath the patient may complain of pain or oppression behind the sternum, or possibly only of a slight soreness. Cough usually attends the dyspnœa, with expectoration of mucus. Sometimes the mucus is blood-tinged, and even streaks of blood may be expectorated after a considerable time, in cases of leaking aneurism.

If the obstruction is due to a *foreign body*, the dyspnœa is of the same type, but occurs *suddenly*.

b. Bronchial Obstruction. Laryngeal movement is not increased and the voice is not changed. If a bronchus is obstructed, the lung of the unobstructed bronchus becomes the seat of emphysema. When obstruction takes place gradually, compensatory emphysema occurs, developing slowly, not rapidly as in the former instance, the degree depending upon the amount of obstruction in the opposite bronchus. The physical signs over the lung of the obstructed bronchus are pronounced. The vesicular murmur is absent, the fremitus is absent, the movement of the affected side is impaired. With these changes the percussion-sound is normal at first, although its limits are influenced less by forced inspiration and expiration; later, it progresses from impaired resonance and dulness. As the case advances, the affected side may fall in and measure less than the opposite side. A snoring or whistling sound may be heard over the root of the lung, between the scapula and vertebræ, or moist râles may be present.

The causes of tracheal and bronchial obstruction are: (*a*) External pressure. First, tumor of the *thyroid gland;* second, *thoracic aneurism;* third, *mediastinal tumor* from other causes than aneurism, as disease of the glands, cancerous or tubercular, or mediastinal abscess; fifth, *cancer* of the *œsophagus;* and, finally, in rare cases, a *dilated auricle*. (*b*) Diseases of the walls of the trachea. They cause obstruction by narrowing the calibre. *Syphilis* is the most frequent cause of such obstruction. (*c*) Foreign body. The presence of a *foreign body* within the lumen causes obstruction. The foreign body may remain free for a time, moving up and down as the patient coughs, and, indeed, it may be felt against the side of the trachea when the finger is placed outside the neck. Later, the foreign body usually becomes fixed in the right bronchus, or one of its main divisions, because the opening of the right bronchus is more direct than that of the left. In some instances the body may be dislodged and fall into the opposite bronchus. Rarely it falls first into the left.

B. Diminution of the Air-space in the Lungs. All forms of pulmonary disease attended by consolidation, by compression of the lung, or occlusion of the small bronchi, are included under this subdivision. The degree of dyspnœa, of course, depends upon the extent of the diminution in the air-space. In *pleural effusions* from any cause the air-space is lessened and dyspnœa occurs. In bilateral effusions it is more marked than in unilateral. The severity of the dyspnœa depends somewhat upon the rapidity with which the effusion takes place. In cases of sudden effusion of air, as in *pneumothorax*, the dyspnœa is

very alarming at first, but, as accommodation takes place, it is gradually relieved. In rapid effusion of serum it is also serious.

The characteristic form of dyspnœa due to lessened air-space is seen when obstruction of the air-tubes takes place on account of spasm.

Asthma.

Asthma is a chronic disease caused by spasmodic narrowing of the bronchial tubes, and characterized by paroxysmal attacks of *dyspnœa*, diminished respiratory movement of the chest, prolonged expiration, attended by a wheezing sound and sibilant râles, and ending abruptly with the expectoration of tenacious mucus. The attack may be limited to a single night, or may be prolonged for days, with nocturnal exacerbations.

Premonitory symptoms are said to occur in about one-half the cases. These are for the most part nervous, such as headache, neuralgia, irritability of temper, vertigo, drowsiness. Hyde Salter found that there were premonitory symptoms in 111 out of 226 cases collected by him. In 63 they were nervous, in 8 there was profuse diuresis, and in 14 they were connected with the digestive system.

The attack itself usually begins during sleep, and often at a regular time. It may, however, begin during the day, and at a certain hour, independently of sleep. The onset is manifested by tightness across the chest and more or less difficulty in breathing. This dyspnœa increases rapidly and often reaches an extreme degree. The face becomes pale and anxious, and may be covered with a cold perspiration; the lips are dusky from insufficient oxygenation of the blood. The patient feels smothered, and makes frantic efforts to get his breath, rushing to an open window, no matter how cold the weather, or, if unable to leave the bed, sitting up with the hands pressed upon the bed so as to give purchase to the accessory muscles of respiration. Notwithstanding that great respiratory efforts are made, the chest moves but little, because the lungs are already distended to the extent of a full inspiration. The patient is unable to expel the contained air, owing to the spasm of the bronchial tubes.

The frequency of respiration is diminished, sometimes to one-half the normal; the rhythm is also altered, inspiration being short and gasping, and followed without pause by expiration, which is much prolonged and accompanied by a wheezing sound audible to bystanders.

There is an increased amount of air in the thorax, and inability to remove it. The chest is enlarged—barrel-shaped—the movement of the chest is lessened and strikingly out of proportion to the muscular exertions. The diaphragm is lowered.

The *physical signs* are hyper-resonance on percussion; on auscultation, faint, short inspiration, prolonged expiration, and sibilant and sonorous râles, more marked on expiration.

The duration of an attack of asthma varies from half an hour to a day or two. In patients with chronic bronchitis it may be prolonged for a week or two, with remissions during the day. It may subside abruptly or by degrees.

Subsidence of an attack is marked by expectoration, the sputa having special characteristics. (See under Sputum.) At first it is made up of rounded gelatinous masses, which, when unfolded in water, are seen to be made up of spirals. Later it becomes mucopurulent.

Curshmann's spirals and the Charcot-Leyden crystals are nearly always found. The leucocytes are increased, and 25 per cent. of them are eosinophiles.

The causative factors in asthma are various. About twice as many males as females are affected, and there is a marked hereditary tendency in some families. There is probably some special peculiarity in asthmatic patients, but just what it is has not been determined. It may reside in the lungs, and may be part of a general constitutional irritability (Salter). Bronchitis, emphysema, and heart disease act as causes, and so do syphilis, malarial poisoning, and chronic Bright's disease.

The above description applies to that form of dyspnœa treated of in the text-books as spasmodic asthma, a respiratory neurosis which for lack of knowledge is classified as a disease. Up to this time the dyspnœa is paroxysmal. Sooner or later it becomes constant. When the dyspnœa associated with asthma becomes constant other changes have taken place in the lungs. First, there is persistent bronchitis; second, the presence of emphysema. Indeed, in many cases it is difficult to ascertain the exact sequence of affections. In emphysema of the lungs dyspnœa is constant, but, on exposure to cold or on account of an attack of indigestion, more severe paroxysms may occur, as well as asthmatic attacks, although the patient is not an asthmatic. On the other hand, a patient may have had asthma for a number of years, during which attacks of dyspnœa occurred only in paroxysms. As time passes the paroxysms become more and more frequent, and emphysema develops. With the advent of emphysema the dyspnœa becomes more constant.

Asthma, as above described, is a type of dyspnœa of nervous origin. It has just been said that it is due to spasm of the bronchial tubes. This may occur from a number of causes: (*a*) It may be of central origin, from irritation of the pneumogastric centre; (*b*) it is just possible that some disturbance of the trunk of the pneumogastric nerve will also cause asthmatic dyspnœa; but what concerns us most is (*c*) the paroxysmal dyspnœa which arises reflexly from irritation of the terminal endings of the pneumogastric nerve, or of nerves intimately associated with the pneumogastric, in the medulla. (1) Disease in the upper air-passages, as polyps, or a hypertrophy of the turbinated bones, or adenoid growths, are the most frequent source of paroxysmal dyspnœa. Not only in permanent disease of this character do we have such dyspnœa, but temporary irritants applied to the nares likewise produce it. Various odors, the irritation of micro-organisms, or of pollen, or emanations from vegetable life, provoke attacks of nasal congestion and reflex dyspnœa. The irritation is propagated through the ethmoidal and posterior nasal branches of the nerve, the Vidian and nasopalatine nerves, to the septum, and the anterior palatine to the middle and low turbinates. (2) Irritation in the fauces and larynx is not so likely to cause dyspnœa, yet there is no doubt that the presence

of a constant irritant in these situations tends to provoke, or keep in a state of excitability, the respiratory tract, so that asthma is more likely to persist. (3) To this class of cases belongs the irritation of the terminal branches of the pneumogastric nerve in the stomach. Peptic asthma, or the asthma of indigestion, may owe its origin to these causes. Often the irritation is central, due to the irritating influence of an abnormal product of indigestion upon the respiratory centres in the medulla. (4) For the same reason we have asthma due to other poisonous substances circulating in the blood, as the poison of uræmia. The dyspnœa due to this condition usually occurs in paroxysms, but may become constant. Sometimes it is the first intimation of the presence of renal disease. The dyspnœa of diabetic coma may occur from the same cause. The nature of both is recognized more particularly by their associate symptoms. The condition of the urine, the odor of the breath, and the exhalations, the presence of hypertrophy of the heart and of an accentuated second sound, point to a uræmic origin. The history and symptoms of diabetes, the odor of acetone on the breath, the presence of sugar in the urine, the absence of organic pulmonary disease, point to diabetes. The dyspnœa of uræmia cannot be distinguished from other forms of dyspnœa, except by the exclusion of cardiac and lung disease. It is often difficult to do this, because uræmia so frequently develops after the hypertrophied heart has failed, so that the physical signs of dilatation may be sufficient to explain the dyspnœa. The dyspnœa of diabetic coma, known as "air-hunger," is characterized by slow and deep respirations. Cheyne-Stokes respiration is due to the same cause—namely, irritation in the medulla, as in other forms of nervous dyspnœa. It must not be forgotten that the dyspnœa of uræmia may present the Cheyne-Stokes phenomenon.

Diminution of Air-space from Extrapulmonary Causes. Anything which crowds upon the thorax, interfering with pulmonary expansion, causes dyspnœa. This is notably the case in affections below the diaphragm. Hence, in enlargements of the various organs of the abdomen, as the liver, spleen, kidneys, pancreas (cystic disease), and uterus, dyspnœa always occurs. In accumulations of gas (flatulency), or of fluid (ascites), the diaphragm is pressed upward and encroaches on the thoracic capacity. In abdominal tumor, as of the ovary, the omentum, and of the organs above mentioned, dyspnœa is a distressing feature.

C. Interference with the Action of the Muscles. Practically any derangement of the action of the respiratory muscles diminishes the air-space, as expansion of the lungs is interfered with. Nevertheless, the cause of the dyspnœa is extrapulmonary. It is due to weakness or paralysis of the muscles concerned in breathing, or to inhibition of their action on account of pain, or to interference with their action on account of obesity, myxœdema, or œdema, or on account of actual disease, as in trichinosis or myositis.

1. Phrenic dyspnœa is a peculiar form due to *paresis* of the *phrenic nerve* and consequently to interference with the action of the *diaphragm*. It may not be observed as long as the patient is at rest. Upon slight exertion the effort distresses him and causes an increase in frequency

of the respirations. After a few steps a sense of suffocation ensues, or upon ascending an elevation the patient must stop frequently to take breath.

Other physiological processes are affected in phrenic dyspnœa. In the act of sighing the patient feels as though the abdominal organs were drawn up into the chest. Any straining effort, as defecation, is rendered difficult. The voice is weak, and there is difficulty in coughing and sneezing, because a full inspiration cannot be taken. A slight attack of bronchitis may be very serious on this account. On inspection during inspiration, instead of the natural expansion of the ribs and chest, the epigastrium and the hypochondriac regions are drawn in. During expiration they are pushed forward. The thoracic movements are reversed. The abnormality may be detected on palpation with both hands below the cartilages of the ribs, even better than by inspection. Unilateral paralysis of the diaphragm causes drawing in of the corresponding hypochondriac region.

In progressive muscular atrophy, in general lead-poisoning, and in multiple neuritis from other causes, paralysis of the diaphragm may take place. It is said to occur in hysteria, and Walshe states that he has seen it after diphtheria. In fatty degeneration of the diaphragm, on account of inflammation extending from the peritoneum to the pleura, the same phenomenon has been seen. It may occur in trichinosis.

Paralysis of the diaphragm must be distinguished from inaction. If during the act of inspiration one or both hypochondriac regions are drawn in, it is diagnostic of inaction rather than of paralysis; whereas paralysis of the diaphragm is always accompanied by paralysis of other muscles.

Dyspnœa due to paralysis of other respiratory muscles can be recognized on careful inspection and palpation. The atrophied groups of muscles are readily observed. Electricity may aid in the diagnosis.

2. Pain inhibits muscular action. The source of the pain may be in the pleura, the muscles, or the intercostal nerves. Frequently it is below the diaphragm, as in peritonitis, hepatitis, etc., interfering with the action of that muscle. The dyspnœa that occurs from pain, as pleuritis, or inflammation of the chest-wall, is recognized by the posture which is taken in order to relieve the affected side, by local tenderness, and by the physical signs of pleurisy or of pleurodynia.

Clinical Varieties. We observe whether dyspnœa is (*a*) influenced by exertion; (*b*) modified by the frequency of respiration; or (*c*) by the respiratory rhythm; and (*d*) is constant or paroxysmal.

(*a*) Influenced by Exertion. 1. *Shortness of breath* may be apparent on *exertion* only, as in cases of simple debility, or of interference with respiratory action on account of obesity. It is the form of shortness of breath seen in anæmia and in moderate cardiac debility. It may not be observed by the patient unless he walks hurriedly or ascends a flight of stairs. 2. *Shortness of breath independent of exertion* is of more serious import, and is due to a number of causes. It is the shortness of breath that is seen in severe cardiac and pulmonary disease. To the latter belong asthma and emphysema, bronchial obstruction, pulmonary consolidation and compressions (by effusions).

(*b*) The Frequency of Respiration. Dyspnœa varies clinically in the frequency of the respiration. In its most extreme form it is known as *orthopnœa*, when the upright posture of the trunk is assumed. (See Posture.)

1. *Respiration Slow or Normal.* *a*. Dyspnœa may be characterized by deep inspirations, the frequency of respiration being less than normal. This is one of the forms of dyspnœa seen in diabetic coma—"breathlessness without dyspnœa." It is most characteristic, and associated with nausea, vomiting, and coma, while the breath and urine smell of acetone. *b*. The breathing may be slow and stertorous. Such breathing is likewise associated with coma, but the coma is of central origin, due chiefly to apoplexy or tumor. It may be observed that respirations with dyspnœa are usually central or toxic.

Toward the end of life the respirations, even though hurried before, become slower from carbon dioxid intoxication.

2. *Respirations Increased.* The respirations may be hurried and create distress in simple nervousness alone, and hurried respiration is quite common in cases of hysteria. In the latter affection the frequent breathing is often attended by distress. The respirations are quickened, and are half the normal pulse-rate or even as frequent as the pulse. The term "panting" is applied to such respiration. The same character of breathing is seen in exophthalmic goitre. The rate of respiration is increased in all forms of dyspnœa upon exertion (see above), and in all forms due to heart or lung disease.

(*c*) The Rhythm. Alternately slower and shallower breathing, and then quicker as well as deeper, is seen in the peculiar form of breathing known as Cheyne-Stokes respiration. It includes a period of apnœa, with simultaneous alterations in the size of the pupils. (See Uræmia and Diseases of the Brain.)

(*d*) Dyspnœa may further be divided clinically into *constant* and *paroxysmal* dyspnœa. Constant dyspnœa implies a persistence of the cause. Paroxysmal dyspnœa does not include the form that is increased by exertion—a form which in one sense may be paroxysmal. It is seen in its most typical form in asthma. It is often of cardiac origin, but may be due to central or reflex causes. It occurs usually at night. Constant dyspnœa is frequently subject to aggravations paroxysmal in occurrence. Asthma is the type of true paroxysmal dyspnœa.

Diagnosis. While dyspnœa is usually easy of recognition, it must not be forgotten that attacks of acute indigestion, with thoracic symptoms of oppression, may simulate the oppression of dyspnœa. This form of dyspnœa is temporary, however, and not associated with increased rapidity of respiration. Dyspnœa is recognized by increase in rapidity of chest-movement, with increased action of all the muscles of respiration, both the essential and the auxiliary muscles. At the same time the expression is characteristic. The alæ nasi move, the eyes and countenance are indicative of more or less agony, the pupils are dilated. As the dyspnœa continues cyanosis develops, and frequently a cold sweat breaks out. This may be limited to the forehead and face and to the extremities, or may become general. The hands

and feet become cold. Stupor sets in, carpo-pedal spasm or general convulsions follow, the respirations become slower, and death takes place in coma or from heart-failure (asystole).

The dyspnœa of emphysema is characteristic; it is due to inability to empty the chest of air (*expiratory* dyspnœa). The inspiration is short and quick; the expiration is prolonged, and all the auxiliary muscles are called upon to complete the act. The powerful abdominal muscles are seen to contract vigorously, and thus aid in pressing up the diaphragm. The quadratus lumborum and serratus posticus superior et inferior draw down the ribs. The scaleni are strongly contracted, the serratus magnus, latissimus dorsi, and the pectorales all aid in elevating the ribs. Knowledge of the processes involved in forced expiration renders the diagnosis comparatively easy. The contraction of the broad abdominal muscles confirms the diagnosis.

Cough in Pulmonary Affections. (See Larynx.) Coughing is a reflex act. A deep inspiration is taken, followed by closure of the glottis, succeeded immediately by a sudden expiratory effort, during which the glottis is opened, causing a loud sound with the forcible passage of air outward, along with any substances in the air-vessels.

Causes. The pulmonic irritation, on account of which the act takes place, usually begins in the respiratory mucous membrane. The cough is then used to expel accumulations of mucus or pus, or foreign substance. It occurs in all forms of bronchitis and in the lung affections generally in which bronchitis is associated. The cough of phthisis, if not laryngeal, is due to a localized bronchial catarrh. Nodules outside of the bronchi, situated in the lung substance, do not provoke the act of coughing, as we see in the calcareous and fibrous nodules of healed tuberculosis. The irritation is not limited to the mucous membrane of the bronchial tubes, but occurs in the mucous membrane of any portion of the respiratory tract. A foreign body of any kind in the bronchus sets up cough. It is notably present in pharyngeal and laryngeal diseases. The cough of the latter is of peculiar character, which renders it easily distinguished from cough due to other causes.

It must not be forgotten that the presence of an irritant does not always excite cough. Thus, when the sensibilities are obtunded, as in typhoid fever, in disease of the brain, or in the last stages of any disease, the presence of mucus will not excite cough, and yet it is known to be in the trachea, on account of the rattling which takes place. In cases of phthisis sudden checking of the cough and expectoration, on account of weakness, is of bad prognosis and denotes approaching death. It is also a bad sign in pneumonia.

Central and Reflex-cough. Cough may also occur from causes outside of the air-passages. It may be of centric origin. Kohts has found by experiment that irritation of the floor of the fourth ventricle, above the centre for respiration, excites a cough. This centric origin may possibly explain the cough of hysteria, and the short, barking cough which arises in hysterical or nervous states, when the patient is afflicted with the idea that he is about to have hydrophobia. Irritation of nerves which are in anatomical relation with the pneumogastric also excites cough.

Ear-cough. The most characteristic cough of this form is that due to the presence of a foreign body in the meatus of the ear, or to disease of that organ. It is sometimes difficult to examine the external auditory meatus, because coughing is excited. The afferent nerve which receives the irritation is the auriculo-temporal branch of the fifth nerve, according to Dr. Fox, and not the minute auricular twig of the vagus.

Tooth-cough. The same authority points out the occurrence of cough from the irritation of the stump of a tooth, and refers to cough in infants during the first dentition.

Stomach-cough. The popular opinion that cough is very frequently due to the stomach is not substantiated by the experiments of Kohts. Nevertheless, we frequently observe cough in patients who are suffering from mild gastric catarrh, the treatment of which relieves the cough. This is in all probability due to the fact that with the gastritis there is a secondary pharyngitis, and, as the former is relieved, the latter, which causes the cough, disappears entirely.

It will be seen, therefore, that when investigating the cause of a cough in diseases in which this symptom is prominent, it is necessary not only to make examination of the respiratory tract throughout its course, but also to examine the condition of the ears and the teeth, and to bear in mind its possible centric origin.

Clinical Characteristics. The cough may be dry or moist. A *dry cough* occurs when there is an irremovable source of irritation (see dry cough of laryngeal disease). It is seen in the first stage of *bronchitis.* It occurs in the earlier stages of *phthisis.* As a short, hacking, suppressed cough it occurs in *pleurisy* in the first stage. In the second stage it is superficial, as if the sound-waves were checked. It is characteristic and most familiar, although described with difficulty. It is the best type of cough due to irritation outside of the respiratory tract. The ear-cough and tooth-cough partake of this character. In cases of emphysema the cough may be dry and unproductive for a long time, and only be relieved after a small pellet of tough mucus is discharged. In the same category belong the nervous cough, which is nothing but a bad habit; the cough of hysteria, and the cough of a peculiar barking character that occurs at puberty, which Sir Andrew Clark has described.

The *moist* cough is attended by expectoration of a mucus, mucopurulent, purulent, or bloody character, which is comparatively easily removed.

Dry and moist or loose cough may be either *constant* or *paroxysmal,* or both. Constant cough implies a persistence of the cause, which is strictly pulmonary, as in pleurisy, phthisis, bronchitis, and consolidations generally; paroxysmal, a recurrence of cause when pulmonary, or a reflex or central cause.

Under some circumstances the cough is almost constant. The irritation is constantly present. A large amount of secretion is rapidly poured out, keeping up a constant cough. This is seen in *bronchorrhœa* and bronchial dilatation and in the later stages of *tuberculosis.* In these affections the moist cough may occur three or four times in

twenty-four hours, during which time an enormous amount of sputum is thrown off. The cavity is thereby emptied, the accumulation of matter in which excites coughing only after a certain level is reached. In this affection the cough is further characterized by aggravation on change of position.

The moist cough may occur in paroxysms only, each paroxysm being relieved by the removal of the irritation, the subsequent paroxysm not taking place until the irritating secretion has reaccumulated. In cases of *bronchitis* of the second stage paroxysms of cough may occur every few hours, or the cough may take place once in the twenty-four hours, usually in the morning on arising. The accumulated secretions of the night are disposed of, and then the patient remains free from annoyance. Paroxysmal coughs occur in cases of cavities, either of the lung or of the pleura opening into the lung. Cough is excited whenever the cavity fills with secretion. The paroxysm may occur daily or several times a day. The association with retching and vomiting is of some diagnostic significance. It is seen not only in whooping-cough, but also in phthisis. In *pertussis* the character of the cough is of special diagnostic significance; it occurs in paroxysms. The expiratory efforts are frequent and rapid, followed by a noisy, prolonged inspiration, during which the characteristic whoop is created. At the same time the appearance of the countenance is marked. The face is cyanosed, the eyes stare, the appearance of distress is most striking. The labored efforts at coughing frequently terminate in an attack of retching or vomiting.

The *diagnostic* significance of cough is estimated by the character; by the sound; whether constant or paroxysmal; by the frequency of the paroxysm; by its development at particular times or under particular circumstances, as on arising in the morning, or change to a cold atmosphere, or speaking, or upon movement, as in phthisis. By the sound, laryngeal and bronchial, coughs are distinguished. The diagnostic value of cough further depends on a knowledge of its duration and the character of the expectoration. (See Sputum.)

The Sound. The character of the cough sound, however, is usually modified by the condition of the larynx, for which consult the section on Laryngeal Diseases.

Hemorrhage. Hemorrhage of the lungs occurs from disease or from rupture of adjacent bloodvessels into the air-passages. It is not in itself a symptom of lung disease. A hemorrhage may be small in amount and continue over a considerable period of time, or it may be characterized by a sudden profuse discharge, which at once terminates the life of the patient.

CAUSE. *A.* Affections of the lungs.

1. *Congestion of the lungs* will lead to hemorrhage. The amount of blood is small; it may be limited to streaking of the expectoration, or a few mouthfuls may be discharged. In (*a*) *organic heart disease* this form of hemorrhage is seen. It is also a characteristic feature of the first stage of (*b*) *croupous pneumonia.* The rusty-colored sputum is due to the rupture of the capillaries. In (*c*) *hemorrhagic infarcts*

hemorrhage occurs, and is diagnostic if attended by the sudden formation of a consolidated area in the lung. In (*d*) *phthisis* it also occurs (see below).

2. *Tuberculosis.* In tuberculosis hemorrhage may occur either (*a*) as the first symptom of the disease, on account of collateral congestion around infiltrated areas, or (*b*) later, on account of ulceration of an artery when excavation of the lung has taken place. In the early stages the hemorrhage is usually profuse, but not fatal. It may occur repeatedly during a series of weeks, excited, no doubt, by the violent non-productive cough which attends the earlier stages of this disease. In the later stages, when the vessels are ulcerated, the patient may have repeated hemorrhages, varying from a few ounces to half a pint or a pint. They may occur daily, or be repeated at intervals of a week or more for a long period of time. After the hemorrhages that occur at long intervals the patient experiences much relief. Indeed, the dyspnœa, cough, and chest oppression subside in a remarkable degree, and the occurrence of another hemorrhage is often predicted by a gradual recurrence of these symptoms. Death does not usually ensue on account of the large hemorrhage from phthisical ulceration, and yet it may possibly take place. The writer has seen four instances of hemorrhage into a large cavity, three with external hemorrhage, which caused death instantly. Hemorrhage with the expectoration of calcareous masses recurs (*c*) frequently in patients with healed or *quiescent tubercle.*

3. *Cancer.* Hemorrhage recurring frequently is significant of *cancer* of the lungs, in the absence of other causes.

4. *Plastic Bronchitis.* It is of common occurrence in *plastic bronchitis,* when large bronchial casts are expelled.

5. *Gangrene.* In *gangrene* of the lung it frequently occurs, often causing death. The odor and sputum indicate the true nature of the primary lesion.

B. Disease outside of the respiratory tract. (1) Aneurismal disease of the bloodvessels, which are in intimate relation with the trachea and bronchus, frequently causes ulceration into these tubes, with hemorrhage. The hemorrhage is usually profuse and often induces sudden death. Sometimes the profuse hemorrhage may be preceded for days by small hemorrhages. The physical signs of aneurism are sufficient to explain the cause. The bleeding can sometimes be seen in the trachea, when an aneurism of the innominate artery or the aorta presses upon that tube. (2) In diseases of the heart it does not usually take place until the later stages of the disease, and is associated with secondary congestion of the lungs. It may, however, be an early symptom in mitral stenosis. The hemorrhages may amount only to staining of the sputum, or several times during the day an ounce or more of blood may be expectorated.

C. Affections of the blood or bloodvessels, with hemorrhages in other portions of the body. Thus, it may occur in hæmophilia, in purpura, in scurvy, and in anæmia. It occurs in jaundice with hemorrhages in other situations.

D. Gouty endarteritis. In the aged of both sexes, hemorrhages take place independently of disease of the heart or of the parenchyma of the lungs. Sir Andrew Clark and others have spoken of these hemorrhages and attributed them to gouty changes in the vessels as well as to degenerations of lung-tissue, on account of which the rupture took place.

E. Without known cause. In certain instances pulmonary hemorrhages occur in which it is quite difficult to find any cause for the discharge. It is quite common to see hemorrhage occur in females : sometimes at the menopause, in other cases during menstruation, or, again, perhaps vicariously, when menstruation does not occur. A number of cases that have come under the writer's observation have had this tendency for years without the development of pulmonary disease, and, apparently, without much influence on the general health. Indeed, it may be said that recurrent hemorrhage from the lungs in women, in the absence of organic disease, is not of grave significance.

The Symptoms. The only symptom may be the presence of blood in the expectoration, or the discharge of a small amount of blood with slight cough. In either instance, unless the patient's mental condition is rendered obtuse by disease, the hemorrhage is alarming to him. He is much perturbed, and there may be palpitation of the heart, besides other nervous phenomena. Apart from the nervousness excited by the sight of blood, small hemorrhages, and even hemorrhages of moderate amount, do not cause any other symptoms.

The symptoms of a large hemorrhage depend upon the amount of blood that is lost. They may amount to faintness and giddiness only, with or without pallor. If more pronounced, syncope may take place ; extreme pallor develops ; the pulse becomes rapid, small, and feeble ; the extremities are cold, and the face bathed in perspiration. If the patient recovers from the syncope, he is extremely restless, sighing and breathing hurriedly. There may be some nausea. Moderate delirium and mild febrile symptoms often follow the restlessness. If the hemorrhages do not recur and the patient's fears are calmed, the color will gradually return and the heart's action become stronger and slower. These symptoms occur whether the hemorrhage is due to disease of the lungs or to aneurism rupturing into the bronchus. If the hemorrhages are large, they differ somewhat in the two conditions. If a large aneurism ruptures, the blood rapidly wells up into the throat and pours out through the nostrils and mouth with great rapidity. With such hemorrhage the end may come in a few minutes. In pulmonary hemorrhages the discharge is not so profuse, and is attended by coughing. With each cough blood is raised to the amount of a full mouthful at a time. The blood discharged from the lungs is bright in color, very frothy, being mixed with air. There are no clots in the discharged fluid. The blood from an aneurism is also bright red, but is not frothy, unless the discharge is very slow, and becomes mingled with air in the vessels. In rare cases of pulmonary hemorrhage an abundant stream pours out, which is dark in color, free from clots, and not mixed with air (large cavity).

Diagnosis. Hemorrhage from the lungs must be distinguished from hemorrhage from the upper air-passages, the mouth, the stomach, and œsophagus. Thus a discharge of blood from the mouth may occur from cracks in the pharynx, or varicose veins. It is not abundant, and the hemorrhage is mingled with mucus, which is streaked with blood. Hemorrhage from the gums may be taken for pulmonary hemorrhage, unless there is stomatitis, or inflammation of the gums from scorbutus or ptyalism. In stomatitis its color is somewhat different. It is thin, fluid blood, often offensive, of cherry-juice color. Hemorrhage from the lungs is distinguished from hemorrhage from the stomach by the difference in the way in which it is discharged, and the difference in the character of the blood. If from the stomach the blood is vomited. It is mixed with particles of food or other gastric contents. It is dark in color, often of the appearance of coffee-grounds; it is not mixed with air, and hence is not frothy. The rapid hemorrhage from ulceration of an aneurism into the œsophagus, or rupture of varicose veins at the lower end of the œsophagus, cannot be distinguished by the appearance from the hemorrhage of an aneurism which may have ruptured into a bronchus. The recognition is dependent upon the physical signs and the previous history of the patient's illness.

Pain. Pain is rarely a symptom of disease of the lungs unless the pleura is involved. In a case of bronchitis there may be some soreness and oppression behind the sternum, but otherwise pain is absent.

In pleurisy pain occurs before the exudation. It is sharp and lancinating, and so severe as to impede respiration and cause the cough to be short and catchy. It is usually seated at the base of the chest, in the lateral or anterior region. It occurs when the patient attempts to take a full breath. Before the inspiratory excursion is half completed it is checked involuntarily, on account of the pain. The patient's hand is placed upon the affected part and he involuntarily leans to that side. The pain of pleurisy may be increased by local pressure, but general pressure, as from the whole hand, a broad bandage, or a large strap of adhesive plaster, always gives relief. In the pleurisy that attends phthisis pain is quite common. It is of the same character as the pain of acute plastic pleurisy, but varies in situation and in degree. The pain occurs in paroxysms. It follows a slight exposure to cold, undue exertion, or fatigue. It may continue for twenty-four hours, and disappear until a repetition of the cause brings it on again. It must be distinguished from the myalgia of phthisis due to cough and exposure. In myalgia the muscles and fasciæ at the bony attachments are very tender.

The pain of pleurisy must be distinguished from pleurodynia, from intercostal neuralgia, and from the pain due to the disease of the ribs. In *pleurodynia* the muscles are sensitive if pressed between the fingers or palpated. An enlarged area is affected, but physical signs of pleurisy or pneumonia cannot be elicited. Cough is absent, and so, usually, is fever. It is associated with pain in other muscular or fibrous structures. There may be a previous history of exposure to cold and dampness. Usually there is a history of lithæmia or frequent myalgia.

Intercostal neuralgia is sometimes difficult to distinguish. The pain is sharp, localized, and may modify the movements of the chest. General pressure relieves it; local pressure at the points where the terminal filaments of the nerve come to the surface may increase it. The so-called Valleix's tender points are, however, not always present in cases of intercostal neuralgia. The patient is usually anæmic, often the subject of uterine or other exhausting disease, and may suffer from neuralgia in other situations. Cough and physical signs are absent. *Fracture of the rib*, or caries of the rib, may be recognized by the local tenderness and by the signs of these conditions. Localized pleurisy may attend both, however—indicated by more severe pain on cough or full breathing. Caries or fracture is determined by pressure upon the diseased rib, which elicits the crepitus of fracture. An empyema that is about to point will cause pain in some area of the chest. The pain is usually seated at the points of election for the discharge of the empyema, and is soon followed by swelling, with heat and redness of the skin, and the occurrence of œdema.

More or less constant pain at the apices, undoubtedly independent of affections of the muscles, is a suspicious sign of tuberculous disease in that situation. It may be aggravated by pressure.

The Data Obtained by Observation.

The Objective Symptoms. By physical examination of the lungs we ascertain (1) their degree of activity (movement); (2) the physical condition of their parts subjected to examination; but the disease is not diagnosticated. If abnormal signs are detected, they simply indicate an abnormal condition of the part, which condition may be due to any number of diseases. As the lungs in health contain air, any physical change that takes place causes either an increase or a diminution in the amount of air. This may be general (bilateral), or limited to one side (unilateral), or to a smaller area (local). In examining the lungs we might be content to answer the question, Is there an increased or a diminished amount of air in the parts suspected to be the seat of disease? A correct answer to this question, and to an inquiry as to the case of the increase or diminution, would explain any abnormal physical condition. The answer is determined by percussion. Fortunately, however, we have as adjuncts the phenomena that can be elicited by means of inspection, palpation, and auscultation. These methods of examination depend upon the movements of the lungs and the sounds produced in breathing and speaking.

Value of Inspection and Palpation. Too much emphasis has been laid in the past on auscultation and percussion in the study of lung diseases. It is the habit to rely too much on these methods, to the exclusion of the simpler and quite as valuable methods—inspection and palpation. The latter have been employed for a long time in the study of the objective phenomena of disease. The former are comparatively modern methods, and have required special cultivation of senses not usually employed in observation, in addition to exhaustive

comparative research, to put the findings on an accurate basis. Naturally, they have been given undue prominence as methods of diagnosis. The pernicious habit of examining the patient without removing the clothing, either from haste upon the part of the physician or false modesty upon the part of the patient, has unfortunately also led to the neglect of inspection and palpation. It is proper to insist that the data obtained by inspection and palpation are as important and valuable as those obtained by other means. They are even more suggestive or diagnostic of physical conditions. The phenomena observed are more positive and surrounded by fewer qualifications.

The Regions of the Chest. For the purpose of bearing in mind the relations of the organs to the surface of the chest, and the localization and proper recording of the seat of the disease, the chest is divided into regions. The regions correspond to anatomical points on the surface of the chest, and are subdivided by transverse and vertical lines. Knowledge of the landmarks which indicate on the surface the position of the parts underneath is of great importance in diagnosis. The *regions* in the anterior portions of the chest are: The supraclavicular region, above the clavicle; the infraclavicular region, below the clavicle, extending to the third rib; the mammary region, from the third to the sixth rib. In the axilla two regions suffice—the upper and lower—the position of the disease being more definitely determined by association with ribs and interspaces. Posteriorly the regions are the suprascapular, above the scapula; the scapular region, and the infrascapular region; the region between the scapula and the spine is known as the interscapular region. The *vertical lines* are to the right and left of the median line: (1) The parasternal line, which is drawn downward midway between the edge of the sternum and the second line, which is (2) the mid-clavicular line, drawn from the middle of the clavicle, generally passing through the nipple in males; (3) the anterior axillary line, drawn from the anterior fold of the axilla; (4) the mid-axillary line, from the centre of the axilla; (5) the posterior axillary line, from the posterior fold of the axilla. In the back one line is sufficient—the scapular line, drawn through the angle of the scapula when the arm is at rest at the side of the patient. For transverse lines the ribs and interspaces are used. In this way the exact location of a diseased area can be indicated. In order that accuracy may attend its localization, knowledge of the methods of determining the landmarks, and especially of counting the ribs, is essential.

The Angles of the Thorax. The *costal* angle is the angle of the rib. It varies during the act of respiration. In inspiration the rib rises as the sternum projects, and apparently elongates; the angles become more obtuse; in expiration the sternum falls, the ribs become more slanting, and the angle is more acute.

The *epigastric* angle. This angle is formed by the convergence of the ribs of both sides to the xiphoid cartilage of the sternum. On inspiration it is obtuse, increasing as the ribs rise; in expiration it is more acute.

Method of Counting Ribs and Interspaces. The first rib corresponds to the clavicle; the first interspace is the region between the clavicle, or first rib, and the second rib; the subsequent number of an interspace corresponds to the number of the rib above it. The following, from Holden, is of great importance to remember, particularly when the ribs of fat persons are counted:

a. The finger passed down from the top of the sternum soon comes to a transverse projection, slight, but always to be felt, at the junction of the first with the second bone of the sternum. This corresponds with the middle of the cartilage of the second rib.

b. The nipple of the male is placed in the great majority of cases between the fourth and fifth ribs, about three-quarters of an inch external to their cartilages.

c. The lower external border of the pectoralis major corresponds with the direction of the fifth rib.

d. A line drawn horizontally from the nipple round the chest cuts the sixth intercostal space midway between the sternum and the spine. This is a useful rule for localization in tapping the chest.

e. When the arm is raised the highest visible digitation of the serratus magnus corresponds with the sixth rib. The digitations below this correspond respectively with the seventh and eighth ribs.

f. The scapula lies on the ribs from the second to the seventh, inclusive.

g. The eleventh and twelfth ribs can be felt, even in corpulent persons, outside the erector spinæ, sloping downward.

h. One should remember the fact that the sternal end of each rib is on a lower level than its corresponding vertebra. For instance, a line drawn horizontally backward from the middle of the third costal cartilage, at its junction with the sternum, to the spine, would touch the body, not of the third dorsal vertebra but of the sixth. Again, the end of the sternum would be at about the level of the tenth dorsal vertebra. Much latitude must be allowed here for variations in the length of the sternum, especially in women.

It is important to recognize the relation of the ribs to the vertebræ. The first rib articulates with the first dorsal vertebra, which can be located by the position of the prominent spine of the seventh cervical vertebra; even in very fat people this prominence can be recognized. The remaining ribs, except the tenth, eleventh, and twelfth, have facets of articulation on two vertebræ; as the second rib, with the first and second thoracic vertebræ. The eleventh and twelfth articulate with the eleventh and twelfth thoracic vertebræ.

Topographical Anatomy. The following anatomical points are worthy of remembrance:

The top of the sternum is on a plane with the lower border of the second dorsal vertebra behind. The junction of the first and second portions of the sternum is known as the angle of Ludwig. It is opposite the middle of the second rib, and is on a plane with the lower border of the fourth dorsal vertebra. The junction of the body of the sternum to the xiphoid cartilage is on a plane with the lower border of the eighth dorsal vertebra.

PLATE XIII.

Fig. 1. Anterior Aspect.

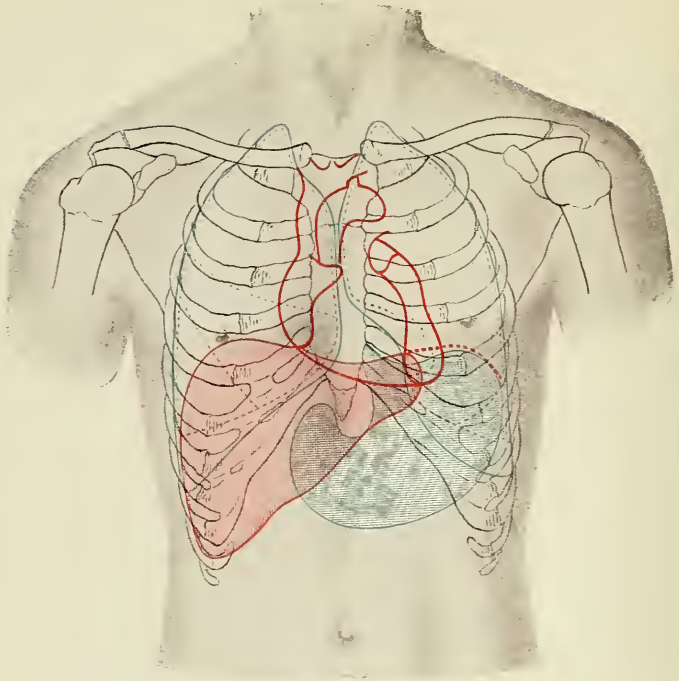
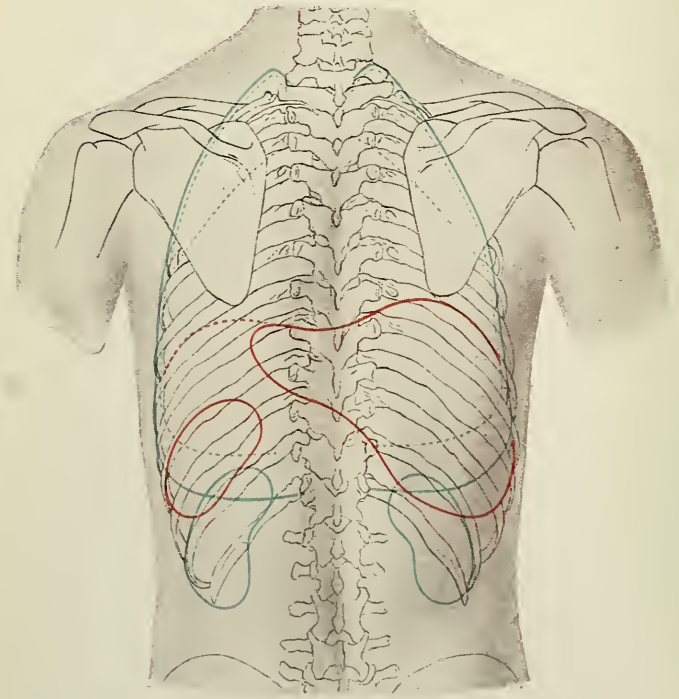


Fig. 2. Posterior Aspect.



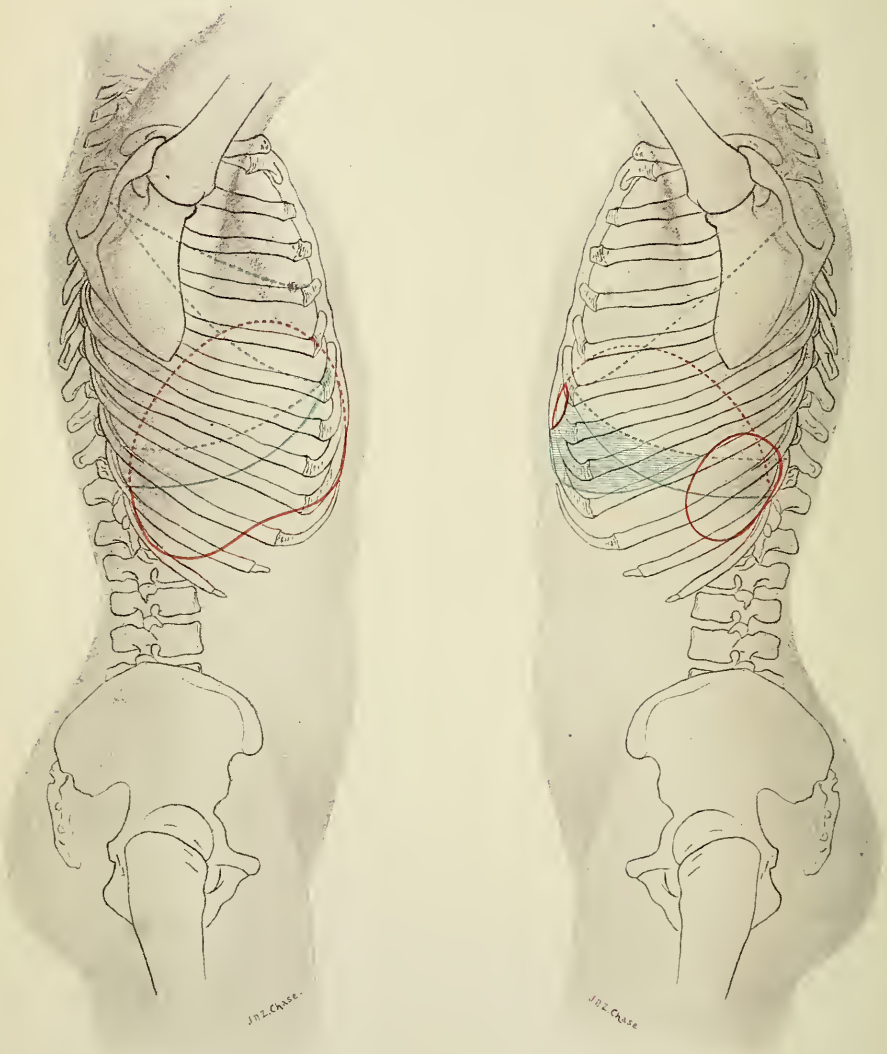
Situation of the Viscera.

Outlines of heart and vessels—broad red lines. Margins of lungs and of individual lobes—dotted green lines. Limits of pleural sacs—solid green lines. Liver—red shading. Stomach—green shading.
(In part after His-Spalteholz and Luschka.)

PLATE XIV.

Fig. 1. Right Lateral Aspect.

Fig. 2. Left Lateral Aspect.



Situation of the Viscera.

Margins of lungs and of individual lobes—dotted green lines. Limits of pleural sacs—solid green lines. Liver and spleen—solid red lines. Diaphram—dotted red lines. Stomach (portion not covered by lung)—green shading. (In part after Luschka.)

The apex of the diaphragm is on a level with the eighth dorsal vertebra.

The trachea bifurcates at the plane which includes the angle of Ludwig and the fourth dorsal vertebra.

Purulent effusions in the left pleural sac frequently point at the fifth interspace, beneath the nipple, because this is the weakest point of the chest-covering. A little external to the inferior angle of the scapula and the eighth and ninth interspaces a similar weak point is found.

Limits of the Lungs. The apices of the lungs reach three to seven centimetres (one and one-fifth to two and three-quarter inches) above the clavicles in front; behind they rise as high as a line drawn transversely through the spinous process of the seventh cervical vertebra. The lower anterior margin of the right lung, when the chest is passive, commences at the insertion of the sixth rib into the sternum, and runs parallel with the upper border of the sixth rib to the axillary line. At this point it descends to the upper margin of the seventh rib. On the left side the lower limit extends as far downward as the right. Posteriorly both lungs reach to the tenth rib. With full inspiration the lungs descend both in front and behind almost the extent of one interspace, while in deepest expiration they are elevated almost to the original position. The "complemental space" of Gerhardt is the space at the lower margin of the lung, and at the point at which the left lung overlaps the heart, in which, during expiration, the surfaces of the visceral and parietal pleura come together. In inspiration the thin layer of the lung in both situations insinuates itself into this space. The heart interferes with the extension of the left lung. The space is triangular in shape, extending in the median line from the fourth to the sixth rib. The left edge of the triangular area corresponds to the edge of the left lung, which, notched for the heart, diverges from the median line and runs along the cartilage of the fourth rib.

Position of the Lobes. Plates XIII. and XIV. illustrate the position of the lobes of the lungs. In the right lung the upper lobe in front extends to the fourth rib, in inspiration laterally to the third, and behind to the spine of the scapula. The lower lobe begins with the spine of the scapula and extends to the tenth rib behind, and from the fourth to the tenth ribs, when fully expanded, in the axillary region. The middle lobe is not seen behind; it extends between the third and fourth ribs in the axillary region in inspiration. In front it extends from the lower margin of the upper lobe to the sixth rib.

The upper lobe of the left lung extends to the sixth rib in front and to the fourth interspace at the side. Behind, a small portion extends above the spine of the scapula, while the lower lobe extends from the spine of the scapula to the base of the lung behind. At the sides it extends from the lowest limit of the upper lobe to the level of the eighth rib.

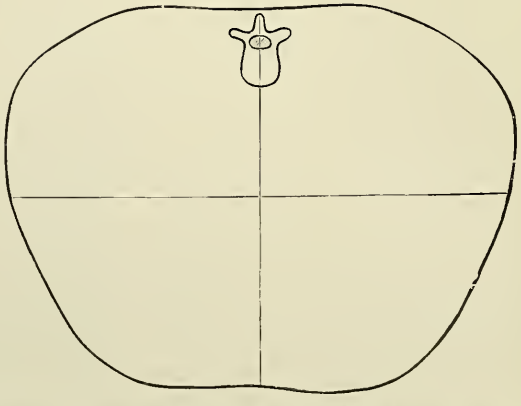
Inspection. By inspection we learn (1) the appearance of the external surface, (2) the shape and size, and (3) the movements of the chest. The second indicates the capacity of the lungs; the last, the

degree of functional activity. The *X-rays* are also employed to conduct inspection.

Methods. The patient must be seated, if possible, in an easy position, with the light falling directly on the part or from the side. He should be viewed by the observer standing, first in front, then behind, and also from the side. To observe the anterior portion it is often well to stand behind the patient and look downward over the shoulders. The arms should fall by the side ; the breathing should be quiet and undisturbed by talking or unusual movements.

The Skin and Subcutaneous Tissue. In health the normal covering should be supple, elastic, and of the color previously described. It is pale in anæmia and wasting diseases ; yellow in jaundice ; pigmented generally or locally from causes previously mentioned. It is the particular seat for the parasitic disease, tinea versicolor, and is the seat of sudamina as well as other non-specific eruptions. The *veins* over the surface of the chest should not be very distinct. They are distinct when there is interference with the circulation in the mediastinum from the pressure of an aneurism or morbid growths obstructing the veins. They, along with the cervical veins, may also be enlarged in dilatation of the right heart. The *capillaries* along the base of the chest are often enlarged or more distinct than usual, and arranged in a bow corresponding to the attachment of the diaphragm. This bow is frequently seen in intrathoracic obstruction. *Œdema* or *subcutaneous emphysema* occurs as indicated under general inspection. If there is too much fat over the surface of the chest, the muscles may be wanting in tone, and an estimation, therefore, of respiratory capacity cannot be made. *Wasting* of the fat and muscles is seen in phthisis, carcinoma, diabetes, muscular atrophy, and paralysis. The degree of *soft-*

Fig. 121.



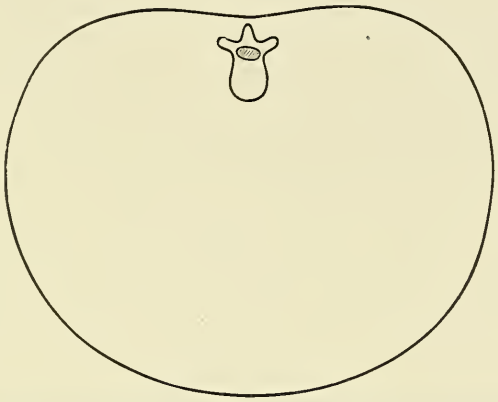
Transverse section of healthy adult chest upon level of sterno-xiphoid articulation. Circumference = 89 centimetres.

ness of the ribs can be estimated in a measure by the undue depression of the ribs at the costo-cartilaginous articulations, and at the base of

the chest (about the sixth rib), during the act of inspiration. It is an indication of rickets. *Rigidity* of the thorax, equal to the senile fixation, occurs in some adults in middle life, and Roberts points out that in young subjects it may be due to congenital syphilis.

The Shape and Size of the Chest. We appreciate the *shape* of the chest in health by an estimation of the relations of the antero-pos-

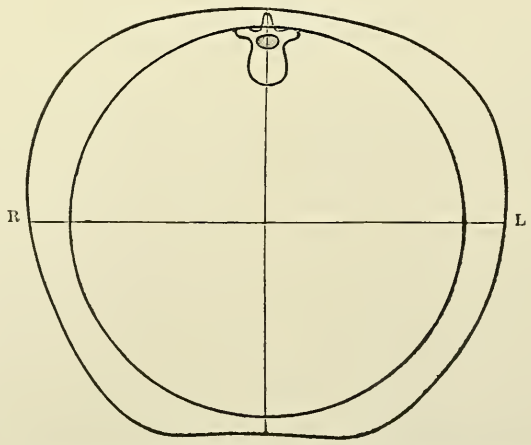
FIG. 122.



Transverse section of healthy male adult chest. Semi-circumference, right side, 16¾ inches; left side, 16½ inches; expansion, 3½ inches. (Ward 6, Philadelphia Hospital.)

terior and the transverse diameters and by the shape of the transverse section of the chest. The latter is an ellipse, and has been described

FIG. 123.



Transverse section of an infant's chest, aged nine months. A circle within shows the similarity.

as reniform (see Fig. 121). The antero-posterior diameter is about one-fourth less than the transverse. Measurement with the cyrtometer

(see Mensuration) verifies the result of inspection with mathematical precision. In children the transverse section is different. It is more circular, and the antero-posterior and transverse diameters are almost equal. (See Fig. 123.) Marked deviations from such section, or in the relations of the diameters, are seen in abnormal types of chest.

It is difficult to describe the *shape* of the chest in health. By repeated practice we readily form a judgment of the true shape. No rule has been applied to the relation of the length of the chest to the length of the body, but it would seem that there is some such proportion. (See Mensuration.) In health the chest should be symmetrical, the right side probably a little larger than the left. In the ideal chest the muscles of respiration should be well developed and there should be a moderate amount of subcutaneous fat. The sternum should project forward from above downward, and the portion joining the manubrium and the gladiolus should be a little more prominent than the other part. It is not unusual to see a clearly marked demarcation between the upper and middle portions of the sternum, or an undue projection of one or more of the upper ribs, and some striking changes about the xiphoid cartilage, none of which are indications of disease. The xiphoid may be depressed, on account of which a crater form or funnel-shaped depression is seen (occupation). The tip of the cartilage is sometimes drawn inward, but more frequently the reverse is noted.

The Movements of the Chest. The frequency, the rhythm, the degree of expansion, and the so-called diaphragm-phenomena are studied. A complete respiratory act consists of two events, inspiration and expiration. Inspiration is active; expiration passive. The latter act is a trifle longer than the former, as may be illustrated by the following proportion—Insp. : Exp. : : 5 : 6. A pause follows the act of expiration. The chest increases in circumference and in vertical length (descent of diaphragm) in inspiration as the lung expands with air. The term *expansion* is applied to the result of inspiration; its degree varies.

The frequency and character of the *movements* in health vary in the two sexes. The respirations are from 16 to 24 in the minute in a healthy adult. In the female they may be 20 to 22. In children the frequency of respiration is much greater—under one year, 44 per minute, and at five years 26. They are *increased* in frequency in the standing position. They are lessened in the horizontal position, increased during bodily exertion, with increased temperature of the air, and during digestion. The hand placed on the epigastrium facilitates counting of the respirations.

The movements of the chest in quiet breathing are more marked in the lower half in male adults, and thus the *costo-abdominal* or *diaphragmatic* type of breathing is seen. The sternum rises, the ribs are elevated, and at the same time are drawn forward and outward. The antero-posterior and vertical diameters increase. The costal angle and epigastric angle become more obtuse. The diaphragm acts conjointly with the external muscles of the thorax, and, as it descends, the epigastric region swells with each inspiratory effort. In expiration the

sternum falls, the ribs become more slanting instead of horizontal, the epigastrium retracts, the angles become acute. The antero-posterior and transverse diameters lessen. The upper half of the chest moves more actively in women, and hence the *costal* or *upper thoracic* type of breathing is seen. The areas below the clavicles and the upper portion above the sternum swell more distinctly during inspiration. The movements of the lower portion, and especially of the diaphragm, are limited.

The *costal* type occurs most frequently in children. The type of breathing is *costal* in both sexes during sleep; the same type is observed during deep respiration.

The Diaphragm-phenomena (Litten). The diaphragm and walls of the thorax approach each other during expiration, and come in apposition at the end of this act. During inspiration they become separated. In persons whose chest-walls are not too thick the movements of the diaphragm are indicated on the surface by the rise and fall of a shadowy line. The patient must lie on his back with his face from the light and head slightly elevated. The light should fall from behind. The observer stands a distance of three or four feet with his back to the light. The chest is scanned at an angle of about forty-five degrees. In the act of inspiration a horizontal shadow or undulation is seen to start on either side about the sixth interspace and passes downward during inspiration over a distance of two or more interspaces, and even to the margin of the ribs. In expiration the shadow begins below and moves upward to the starting-point.

Absence of the phenomena is noted when there is fluid or air in the pleural cavity, when the pleural cavity is obliterated by adhesions, when there is pneumonia of the lower lobe; and in emphysema of the lungs, and intrathoracic tumors low down in the chest. Tumors or fluid accumulations below the diaphragm do not lessen the phenomena.

By this phenomena the volume or vital capacity of the lungs can be estimated. In normal individuals the shadow should move more than two and a half inches. If there is lessening of the extent of movement the respiratory capacity is diminished. In this manner tuberculosis may be suspected. Limitation of the excursion of the diaphragm —X-ray investigations have forcibly taught us—is one of the earliest signs of tuberculosis. This limited excursion can be detected in proper subjects by Litten's method, although it must be remembered that general debility and emphysema lessen the excursion on both sides. In splenic and hepatic enlargements the normal shadow continues, but in a large collection of ascitic fluid it may be detected with difficulty, or may be absent.

The Shape and Size of the Chest in Disease. The chest may be enlarged or diminished in size. Such change may be general or bilateral, unilateral or local.

General or Bilateral Changes in Shape. *Enlargement.* The "barrel-shaped" chest, the type of bilateral enlargement of the chest, is seen in health when it is in the state of full inspiration. All the

diameters are increased, particularly the antero-posterior ; the length is shortened. The diameters are almost equal, and the transverse section approaches a circle. This occurs because in all figures of fixed length, in order that the area may be increased, a change to a circular form must take place. (See Figs. 125 and 126.) The ribs are elevated and almost horizontal, the epigastric angle is obtuse. The sternum and the spine are arched ; the former at the angle of Ludwig. The shoulders are rounded and elevated, and the scapulæ lie flat against the thorax. All the muscles of respiration stand out prominently, the

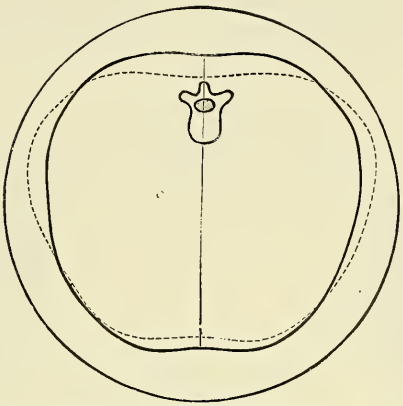
FIG. 124.

Emphysema with enlargement of the chest. The antero-posterior diameter is much increased. (Ward 6, Philadelphia Hospital.)

neck and upper trunk muscles particularly. The individual with bilateral enlargement of the chest presents a striking appearance. The neck is short, the arms are short ; there is undue fulness above the

clavicles. As this enlargement is attended with dyspnœa, the face is drawn and anxious, and the lips usually faintly livid, or purple.

FIG. 125.



Bilateral enlargement of emphysema.

Inner line = emphysematous chest.
Outer line = a circle drawn to show how nearly the emphysematous approaches the circular shape.
Dotted line = natural adult chest.

Actual measurement in centimetres.

Circumference	= natural	89.0	emphysematous,	87.75.
Transverse	= "	29.6	"	27.25.
Antero-posterior	= "	22.25	"	25.4.

—(DR. GEE.)

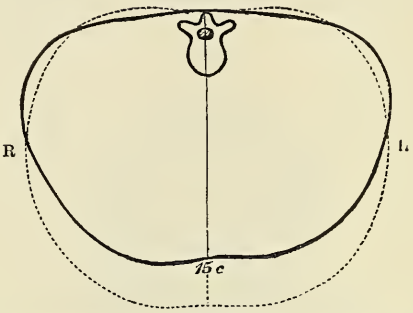
The *movement* of the chest in bilateral enlargement. Expansion is lessened. The respiratory capacity is diminished. The chest is in a state of full inspiration, and the attendant dyspnœa is known as expiratory dyspnœa. The respirations are hurried, the inspirations short, followed by prolonged expiration. While the expansion of the chest in health extends over an area of three or four inches, when the chest is bilaterally enlarged it may be lessened to one and a half inches, or even be as low as half an inch. Both the costal and the diaphragmatic types of breathing are seen in a state of exaggeration. In men the diaphragm acts very vigorously at times. Expiration is three or four times as long as inspiration.

CAUSE. The increase in size is due to enlargement of the normal contents of the chest or to the presence of abnormal contents. In nearly all cases it is due to an increased amount of air within the thorax (normal contents), as in *emphysema*. In a few instances enlargement of both sides is seen in cases of *bilateral pleural effusion;* but, as considerable effusion would be incompatible with life, the enlargement from this cause is never very great. It is said that such enlargement may occur in rapidly growing *cancer* of the *lungs*.

It must be remembered that emphysema can exist without bilateral enlargement of the chest.

Bilateral Diminution in Size. The type is seen in the so-called phthisical or tuberculous chest. The chest is long, the antero-posterior diameter small (see Fig. 126), the transverse relatively very much increased. The angles are acute, the ribs are slanting, the epigastric

Fig. 126.



The flat or phthisical chest, short antero-posterior, long transverse diameter. (Gee)

angle is particularly sharp. The shoulders fall, and hence the scapulæ are prominent—so marked in many cases that the term alar or "winged" chest has been given to it. The anterior plane is often flattened, and hence the term "flat" chest is employed. This change occurs because the curve in the cartilage of the true ribs becomes straight. The *movement* or expansion is lessened just as the respiratory capacity is diminished.

With this type of chest we see the neck long, the larynx (Adam's apple) very prominent, the arms long. The patient is loosely put together; the length of the long bones is increased.

It is known as the phthisical, phthisinoid, or tuberculous chest. (See Figs. 126 and 127.) Although the term tuberculous is applied to the chest of this description, it does not necessarily imply that an individual with such a chest has, or will have, tuberculosis. It is true that in individuals with such type of chest the vulnerability to the action of the tubercle bacillus is more marked, and they are more liable to have the disease. Nevertheless a very large number of individuals go through life with such chests and die of other diseases. If they are not exposed to the infection, they will certainly escape the disease.

Cause. Bilateral diminution means diminution of contents. The extent of air-surface is lessened.

The Chest of Rhachitis. Another type of diminished size of chest is constantly referred to. It is known as the chest of rhachitis (see Fig. 129), and arises in infancy, on account of this disease of the bones. Many other shapes are seen, to which various names have been given. Among the more common is what is known as the

"pigeon-breast." (See Rhachitis, and The Head.) The chest is usually shortened, the sternum is much more prominent than in health, the lower portion projecting to an unusual degree. The portion of the chest at the junction of the cartilages and the ribs is depressed. This tends to throw the sternum further outward. The transverse section of such chest resembles a triangle with the portions where the base-line joins the ribs rounded. (See Fig. 131.) The sternum is de-

FIG. 127.

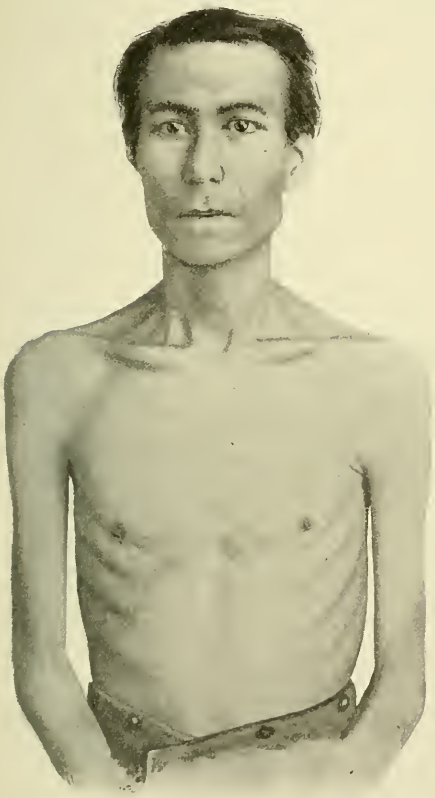


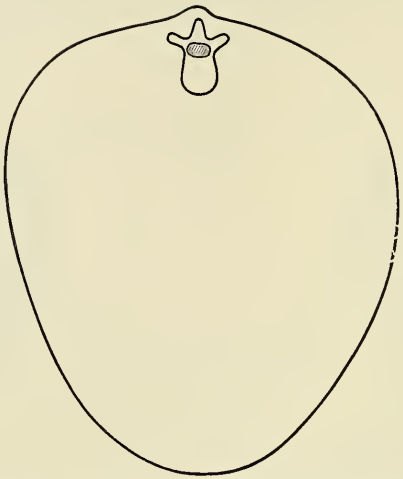
FIG. 128.

The phthisical chest. (Full-blooded Indian, Philadelphia Hospital)

pressed and the osteo-cartilaginous articulations are more prominent in some forms of rickety chest. In others the ribs and sternum from above to the fifth rib are prominent, and from thence downward to the base are drawn in. In the chest of rhachitis the costal angle is usually very acute. (See Fig. 130.) It often looks as if pressure, as by the hands, had been applied to the sides of the chest about the anterior

axillary line, causing the antero-lateral portion to sink inward, while the antero-median portion is projected forward.

Fig. 129.



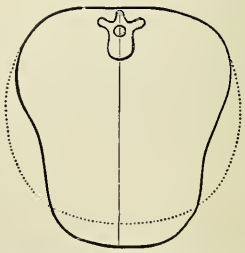
Transverse section of a rhachitic chest at level of sixth thoracic vertebra. Circumference, 32½ inches; right half, 16⅛ inches; expansion, 2 inches.

The chest of rickets is attended by enlargement of the articulations of the cartilaginous and bony portions of the rib—the rhachitic rosary—and by changes in the other bones.

Fig. 130.

Chest of rhachitis. (Eichhorst)

Fig. 131.



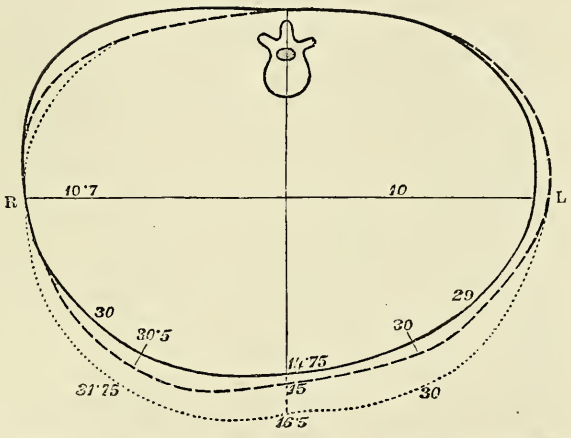
Circumference = 42.75 centimetres.
Rickety chest. Dotted line indicates the shape of chest in an infant about the same age. (Gee.)

The *rhachitic* chest must not be confounded with similar changes in shape due to abnormal conditions of the upper respiratory apparatus in early childhood. In cases of *adenoid disease* of the pharynx (see

Diseases of the Pharynx) the change in shape of the chest has been noted.

The Transverse Groove. This is a depression observed in many individuals. It extends from the median line along the base of the thorax to the axilla; its upper limit is on a level with the xiphoid cartilage. It slopes downward toward the axilla. It is caused in early life by the pressure of the external columns of air on the soft bony thorax when the lungs are not completely filled with air. Hence, it indicates nasal, faucial, or bronchial obstruction in early life, from adenoid disease, bronchial catarrh, or other causes. It may mark the upper limit of the liver on the right side as it was in infancy.

Fig. 132.



Unilateral enlargement of chest (right side), artificially produced by injecting air into the right pleural cavity. Unbroken line: outline before injection. Broken line: outline after moderate distention. Dotted line: outline after extreme distention. Figures at bottom of vertical line indicate the antero-posterior diameter; along horizontal line, transverse semi-diameter; remaining figures, right and left semi-circumferences. (Gee.)

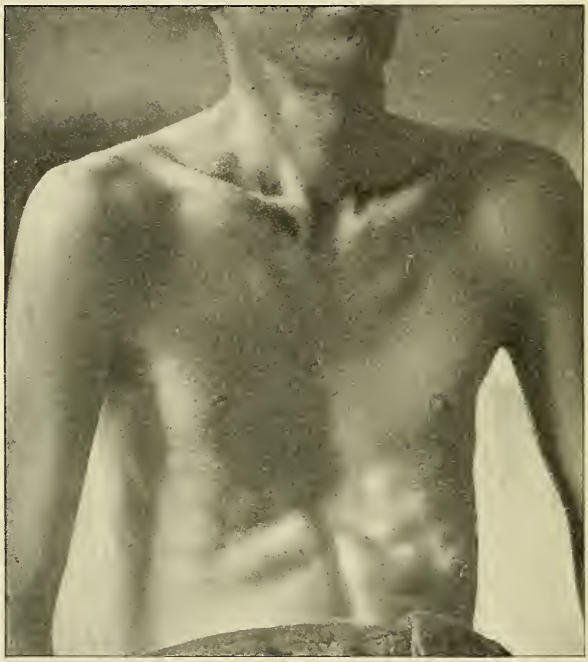
The shape of the chest just described (rhachitic) does not indicate any disease of the lungs; it does indicate deficient respiratory capacity, and is, of course, the tell-tale by which rhachitis of early life or early laryngeal and nasal obstruction are recognized.

Deformities. The rhachitic chest must not be confounded with deformities of the chest which may be congenital in origin, the result of occupation (shoemaking), or of vertebral disease (Pott's disease). The funnel-breast (trichterbrust) is congenital and often seen in several members of a family (Warthin). It is associated with other stigmata of degeneration. The lower sternum forms a deep concavity. (See Fig. 133.)

Unilateral Changes in Shape. *Unilateral Enlargement.* This can usually be seen more prominently at the base. The length is increased. The ribs are elevated, the side more rounded, the costal angle more obtuse. The interspaces are frequently effaced, or fuller than on the corresponding side. The *movement* may be increased or

diminished, depending upon the cause. The nipple is diplaced outward. The scapula of the affected side is also displaced outward, and hence the distance from it to the spine is greater than on the opposite side. (See Fig. 132.)

Fig. 133.



Funnel-breast (trichterbrust).

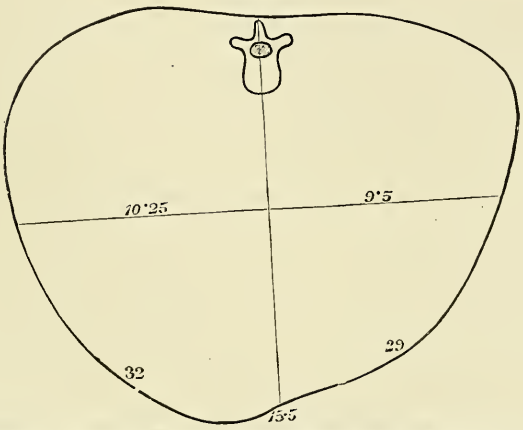
Cause. Enlargement of one side means enlargement of contents. It may be due (1) to increase of the normal contents, as in compensatory emphysema, in which there is an increased amount of air in the lung, or (2) to the presence besides of abnormal contents, as fluid or air in the pleural sac. It is the most characteristic sign of pleural effusion. When the normal contents are increased the movement is increased ; when the pleural cavity is filled it is diminished.

Unilateral Contraction or Diminution in Size. The costal angles are sharper, the plane of the anterior or posterior portion, or of both, is depressed, and approaches the transverse median plane of the chest. (See Fig. 134.) The affected side looks flat before and behind. The semi-circumference is lessened, as well as the diameter through the nipple or any fixed point. The interspaces are lessened in width and may be drawn in. The ribs are closer together, and may almost overlap. The *movement* of the side is lessened.

Cause. Any diminution of contents will cause diminution of the affected side. This may occur from obstruction or compression of the bronchi of that side lessening the amount of air in that portion of the

thorax. Theoretically, it may occur in a case in which there is complete occlusion of the main bronchus. The condition is rare, and is accompanied by marked associate emphysema of the other lung. The unilateral change is most frequently seen in cases of chronic pleurisy and fibroid phthisis. A large portion or even the whole of the lung may be bound down and compressed by thickened adhesions. The pleural cavity of the side thus affected, save where encroached upon by the heart or by invasion of an emphysematous portion of the lung of the corresponding side, is completely obliterated.

FIG. 134.



Unilateral retraction of chest, consequent upon cirrhosis of left lung, in a girl of fourteen years. The figures indicate antero-posterior and transverse diameters and semi-circumferences of right and left half of chest. (GEE.)

Local Changes in Size and Shape. *Enlargement* and *diminution* are also seen.

LOCAL ENLARGEMENT is particularly noted in the region of the heart and great vessels, and will be considered when this division of the subject is discussed. A local enlargement in the lower anterior or lateral region of the chest may occur in cases of empyema, in which the pus tends to be evacuated, or in pulsating pleurisy. Enlargement in diseases of the mediastinum is usually seen in the region of the heart and vessels, to which reference must also be made.

LOCAL CONTRACTION. This may be seen either at the apex or the base. At the apex the local contraction or diminution in size is seen above and below the clavicle. The term *flattening* is applied to this condition. The interspace is sunken and the ribs depressed. It may be more readily seen when looked at from behind. Flattening may also be either in the lateral or posterior region at the base. The anterior and lateral, or the lateral and posterior, region is combined in the local contraction.

CAUSE. The physical condition is the same as in unilateral or general contraction—contraction or diminution in size of the structures underneath. Anything which lessens the amount of air will cause local diminution in size, or *flattening* of the surface. This is notably

seen in *tuberculosis*, in which affection three processes, alone or in combination, lessen the amount of air: First, occlusion of the bronchioles by tubercles and by inflammatory products, causing collapse of the alveoli; second, the overgrowth of connective tissue which attends the more chronic forms of tuberculosis; third, a localized pleurisy. *Local pleurisy*, with organization and contraction of the inflammatory exudate, also causes diminution of the amount of air underneath the part, or diminution of the contents from compression of the adjacent lung structure. In local contractions movement of the part is generally diminished.

General Review. It must not be forgotten that the element of time is necessary to produce changes in shape and size of the chest, with the exception of unilateral enlargement. In emphysema the change in shape takes a long time to develop. The unilateral and local contractions are of slow progress, and hence, it must follow, require more or less chronic disease for their development. The occurrence of pleural effusion may cause unilateral enlargement very rapidly.

The Movements of the Chest in Disease. Bilateral Changes. Frequency. The movements are *increased* in nearly all forms of dyspnœa. (See Dyspnœa.) The frequency of movement varies in many affections. They are more markedly increased in the acute lung affections attended by fever, and are especially more rapid in children. Increased frequency of respiration does not necessarily indicate pulmonary disease. It is always seen in fever, and is a marked phenomenon of hysteria. Conditions outside of the chest increase the frequency, as enlargement of the abdomen from any cause encroaching upon the capacity of the chest. The respirations are *lessened* in frequency in cases of disease of the medulla in which there is pressure upon the respiratory centre, and in some forms of poisoning, as that due to opium.

Alterations in the Rhythm of Movement. Alterations in the character and *rhythm* of the movement are observed by inspection. (See Dyspnœa.) The movements may be (1) slow, and either shallow or deep; (2) rapid and shallow or deep; (3) irregular in rhythm. The relations of the act of inspiration to that of expiration in health are as 5 to 6; in women, children, and the aged, 6 to 8. The expiration is longer. The expiration may be prolonged, so that it is far greater in length than inspiration. *Length of inspiration increased.* The degree of expansion and the duration of inspiration are increased when there is obstruction in the trachea or larynx. Such increased expansion of the upper chest is usually associated with retraction of the soft parts of the thorax, especially at the base. The ribs and the tissues along the margins of the thorax are drawn in with each inspiration. The space occupied by the lung above the clavicle may also be retracted. The transverse groove is more pronounced. If the difficulty in breathing continues, the indrawing becomes very marked, and, if the ribs are soft, permanent. *Expiration prolonged.* Inspiration is short and quick in cases of emphysema. The *expiration* is correspondingly prolonged, and the muscles of expiration are seen to be brought into full action.

In the consideration of dyspnœa we shall describe the appearance and posture of the patient and the action of the muscles of respiration. (See Subjective Symptoms.)

Irregular Rhythm. By inspection the Cheyne-Stokes type of breathing can be noted. "Respiratory pauses" of half to three-quarters of a minute alternate with a short period of increased activity, during which time twenty to thirty respirations occur. The respirations constituting this series are shallow at first, but gradually they become deeper and more dyspnœic, and finally become shallow or superficial again. The acts of respiration are carried on by an alternation of pauses and periods of modified or "tidal" breathing. Sometimes consciousness is abolished during the pause. Often the pupils are contracted and inactive. When the respirations begin they dilate.

Unilateral Changes in Movement. *Increased movement* of one side is seen when the lung of that side is acting vigorously from compensation, the other lung being disabled by disease. The whole side moves more rapidly and vigorously. The increased movement is associated with enlargement of the affected side and hyper-resonance on percussion. Unilateral *diminution in movement* occurs when there is diminution of the respiratory surface, occlusion of the bronchial tubes, or from causes outside of the lung. The air-space is lessened in cases of pneumonia, tuberculosis, or any affection which fills bronchioles and alveoli with inflammatory exudation or fluid. The air-space is particularly lessened by the compression of effusions in the pleura, of contracted and thickened exudations, and of adhesions.

Impaired motion due to *pleural effusion* is almost always unilateral, develops gradually, following an attack of acute pleurisy, is unattended by pain on respiration, but is attended frequently by great embarrassment of the respiration, and sometimes by orthopnœa. Fever is usually moderate in uncomplicated cases. It is to be recognized by the clinical signs mentioned and by the physical signs of fluid in the pleura.

Impaired motion from *chronic pleurisy* is of long standing and gradual development. The chest-wall upon the affected side is retracted, and may be very markedly sunken. In the absence of accompanying lung trouble there is no pain and no fever. It is to be distinguished from other types of impaired motion by the sinking in of the affected side, in sharp contrast with the hypertrophy of the other side; by the absence of fever and pain; by its chronicity; and by the physical signs of thickened pleura and compressed lung. Impaired motion from *pneumothorax* develops suddenly, generally in a person with tuberculosis of the lungs. Its appearance is usually precipitated by coughing, and its sudden development is marked by intense pain, distention of the affected side, great difficulty in breathing, and a very anxious expression of countenance. The escape of air into the pleural cavity is followed by the development of pleurisy with effusion, so that the affection presents the physical signs of air and fluid in the pleural cavity.

The motion of the affected side is sometimes impaired in *pneumonia*, when a large portion or the whole of one lung is involved, and the air-

vesicles are so occluded that very little air can get in. The physical signs in these cases resemble those of pleurisy with effusion very closely, but the diagnosis can be made by noting the acute onset of the disease, with high temperature and frequent respiration, without antecedent pleurisy, and by the presence of cough with expectoration containing the pneumococcus.

Occlusion of the bronchus, with diminution of the movement of the corresponding side, is seen in rare cases in which a foreign body fills the lumen of the tube, or in more common cases of pressure externally upon the bronchus by an aneurism or mediastinal tumor.

Impaired motion from *pressure* on a bronchus by an aneurism or enlarged lymph-gland produces the physical signs of collapse of the lung, coupled with those peculiar to the cause of the occlusion of the bronchus. It develops gradually, the patient having no pain in the lung.

Outside of the lung lessened movement is caused by (1) interference with the muscular activity of that side from rheumatism of the intercostal or respiratory muscles; (2) pain seated either in the ribs or in the pleura. It may be due to *acute pleurisy*, the patient checking the motion of the affected side as much as possible, and breathing with the abdominal muscles, because chest respiration causes acute pain. Impaired motion from this cause or from *pleurodynia* may be suspected when it has come on suddenly, and when respiration causes acute suffering, usually depicted in the face. Pleurodynia and pleurisy are to be distinguished from each other by the presence in the one case of tender muscles, a more constant and less stabbing pain, and absence of fever, cough, and râles; and, in the case of pleurisy, by the occurrence of stabbing pain in respiration, absence of local tenderness, and presence of fine, dry, or coarse râles on inspiration, with cough and fever.

Local diminution of the movement or deficient expansion occurs under the same conditions that produce flattened and local contraction, and for the same reason. Hence deficient expansion is observed in the early stages of phthisis, or in local pleurisies.

Impaired motion, due to consolidation of the lung in tuberculosis, is usually limited to one of the apices, and is accompanied by flattening of the affected apex and emaciation. The condition is of gradual development, and presents the usual signs of tubercular consolidation of the lungs (*q. v.*).

Sometimes the impaired motion and flattening are due to a superficial cavity from tuberculosis or abscess, and when the walls are very thin they may be seen to flap feebly with respiration.

Rarer causes of impaired motion of the lung are cancer and hydatid cyst (*q. v.*).

Fluoroscopic or X-ray Examination. Through the efforts of Williams, Leonard and others the X-ray has become an aid to the diagnosis of pulmonary affections. F. H. Williams has paid especial attention to thoracic diseases. I quote from some of his brilliant studies the results secured by such examination of the lungs:

"In health the lungs are readily traversed by the ray; they appear in the fluoroscope as light areas on either side of the backbone and

the heart. The lower portions of the lungs, bounded by the diaphragm, are seen to move up and down through a distance of about half an inch during quiet breathing, and to descend during full inspiration to a point about two and one-half inches below its level in expiration. The pulmonary is lighter in deep inspiration than during expiration. There are three principal ways in which the fluoroscope may lead us to suspect disease in the chest: (1) The appearance of the dark areas which occur in tuberculosis, pneumonia, carcinoma, diaphragmatic hernia, gangrene of the lungs, and in echinococcus cyst, infarction, pleurisy, empyema, etc., due to the increase in density, which, by obstructing the passage of the ray, diminishes the normal brightness in the chest or changes its normal outlines; (2) the occurrence of abnormal brightness which is found in emphysema and pneumothorax consequent upon decrease in density, which makes the lung area appear lighter than in health as seen in the fluoroscope; (3) the restriction of the maximum excursion of the diaphragm and its altered position and curve from that observed in health."

In tuberculosis the consolidated portion of the lung appears darker than normal in the fluoroscope. The expansion of the lung is reduced. The excursion of the diaphragm downward is diminished during full inspiration, but this muscle is carried up into the thorax as high, or it may be even higher than in health. From time to time the fluoroscopic pictures show the apex of one lung darker, as already stated; the clavicle and upper ribs less marked on the diseased than on the normal side; the darker area extending more and more as the disease progresses. Then the apex of the other lung begins to darken and this area continues to extend. The diminishing excursion of the diaphragm, which is also a characteristic feature of this disease, may likewise be observed, and sometimes may be the earliest sign.

In pneumonia the affected areas are easily recognized in the fluoroscope, and in a central pneumonia may be seen when auscultation and percussion do not reveal them. The excursion of the diaphragm is also restricted, and the heart may be much displaced to the right, if the pneumonia is only on the left side. A secondary empyema, following pneumonia, can be seen by the X-ray. The pleuritic effusion which sometimes accompanies pneumonia may be proved to exist if a dark area and the outline of the diaphragm below the dark pneumonic portion is not visible in the fluoroscope.

In both these affections the outlines of the lower part of the chest are dulled or obliterated, especially the diaphragm line. If the effusion is large the whole chest is dark, and the heart and mediastinum are displaced. In a circumscribed pleurisy or empyema an exploring needle may fail to reach the desired spot, but we may sometimes, by means of the fluoroscope, exactly outline the limits of the fluid.

Lungs that are less dense than normal, as in emphysema, give a brighter area than in health, and the distended lung reaches lower in the chest than normal. The maximum excursion of the diaphragm is much less than in health, as this muscle does not rise so high in expiration. These two signs are characteristic of emphysema. The enlarged ventricles and also the dilated right auricle are seen in late

stages; the heart also lies in a more vertical direction, and its position is not much changed by a deep inspiration.

In pneumothorax the diaphragm is very low, loses its normal curve and movement on the affected side, and the heart and mediastinum are seen to be displaced to the healthy side.

Palpation. By palpation the results of inspection are confirmed, the character and consistence of tumors ascertained, the vocal fremitus determined, and fluctuation detected.

METHOD. The surface should be bared, although the fremitus can be detected through a thin layer of linen or gauze. To detect the fremitus in front, it is often well to stand behind the patient, with the palms of the hands placed over the surface of the chest in front. The opposite position is taken to detect the fremitus behind. The axillary region must also be investigated. The hands should be warmed and applied evenly to the surface. The two sides must constantly be compared, either by simultaneous application of the hands on the two sides, or by applying the hand first on one side, then on the other.

The Vocal Fremitus. CAUSE. The columns of air in the bronchial tubes are thrown into vibration during the act of speaking. The vibrations are transmitted to the hand on the surface of the chest. They are known as the vocal fremitus. In infants the cry must be relied upon instead of the spoken voice.

The fremitus on the right side at the apex is stronger than on the left, because the right bronchus is larger than the left, its angle with the trachea is more acute, and the bronchus going to the right upper lobe is two and one-half inches nearer the larynx than the left (Cary, Ewart). The fremitus is stronger in persons with deep, low-pitched voices, because the vibrations are not so rapid. It is more distinct, therefore, in males than in females, and in individuals with a bass voice. The vocal fremitus is felt more distinctly in persons with thin chest-walls. Thick chest-walls and large mammary glands interfere with the transmission of fremitus. The fremitus is not distinct in children because the vibrations are too rapid.

It is well to become familiar with the vibrations produced by fixed monotones, in order to appreciate the fremitus. The patient is asked to count one, two, three, or to repeat ninety-nine three or four times. It is well to observe a fixed rule as to the words used, in order to have definitely in the mind the character of the vibrations in health, and the departures from the normal in disease.

Vocal Fremitus in Disease. The vocal fremitus may be increased, may be diminished, or may be absent.

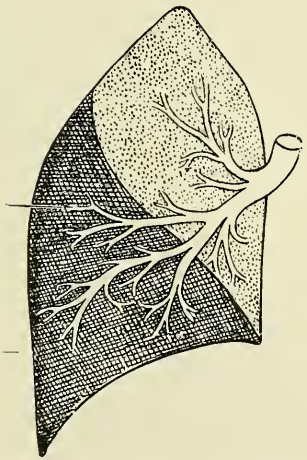
VOCAL FREMITUS INCREASED. When the lung is consolidated, vibrations are transmitted to the hand with greater force. Fremitus is increased in all consolidations, as in pneumonia, tuberculosis, and hemorrhagic infarct. (See Fig. 135.) The fremitus may be absent in rare cases of pneumonia, in which the large tubes are occluded by exudate. The fremitus is increased in the later stages of tuberculosis, when cavities have formed, if the walls are dense.

VOCAL FREMITUS DIMINISHED. Anything intervening between the lung and the surface of the chest which interferes with the conduc-

tion of the vibrations diminishes the fremitus. The fremitus is diminished in cases of thickened pleura, and in thin layers of pleural effusion. The fremitus is lessened if the columns of air in the bronchi are smaller on account of diminution in the calibre, as in bronchitis or in emphysema and asthma. The fremitus is lessened in cavities filled with fluid, or when the bronchus is occluded.

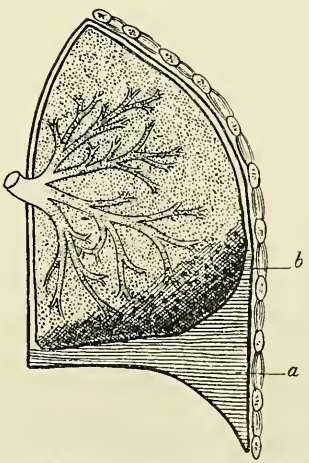
Vocal Fremitus Absent. 1. The vocal fremitus is absent when the columns of air are obstructed entirely by occlusion of the bronchus, as by the external pressure of a tumor, aneurism, or enlarged gland. 2. The fremitus is absent in accumulations in the pleura of air or of fluid, causing interference with the vibrations. (See Fig. 136.) The well-known illustration of striking a stone underneath the surface of the water implies. If the ear of the listener is above the water, the sound cannot be heard. If the ear is underneath the water, the sound is heard a long distance from its origin. Vocal fremitus is absent in pneumothorax, in hydrothorax, in pyothorax, and in hæmothorax. The same physical condition is present when the pleura is greatly thickened, and hence the fremitus is also absent.

Fig. 135.



Consolidation: Pneumonia. Vocal fremitus increased. (Gibson and Russell.)

Fig. 136.



Pleural effusion. Vocal fremitus absent at *a*. (Gibson and Russell.)

The vibrations produced by the passage of air through mucus or fluid in the bronchial tubes are transmitted to the hand when it is laid on the surface of the chest. It is known as the *rhonchial fremitus*. They are felt during inspiration. They may be felt all over the chest in bronchitis, or in asthma, as distinct vibrations, sometimes coarse, or again fine, indicating rapidity of movement. The vibrations may be transmitted over a localized area in phthisis, due to air passing through fluid in the cavity. They are distinct in children in cases of bronchitis, and are often the source of much alarm to the parents.

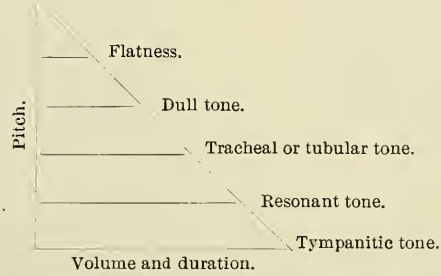
Friction-fremitus. An exudation of lymph on the surface of the pleura often causes a vibration which may be transmitted to the hand.

It is known as a friction-fremitus, and is felt in inspiration. It is usually felt at the base of the chest, in front, laterally, or posteriorly. It is not modified by coughing, and is increased by full breathing. The rhonchi, on the other hand, are influenced by cough and breathing.

Fluctuation is detected by palpation in some cases of effusion, particularly if the intercostal spaces are swollen and tense, or if an empyema is about to point. In rare instances it may be detected by striking the chest opposite the palpating hand.

Percussion. By percussion, (1) sounds are elicited, (2) the degree of resistance to the percussing-finger estimated. When a part is percussed the sounds produced are noises or tones. If a *tone*, the vibrations are uniform and will be in unison with a tuning-fork; if a *noise*, the vibrations produced are without uniformity. We speak of the *pitch*, the *volume*, the *duration*, and the *quality* of the sound. The pitch depends upon the rapidity of vibrations, the number that occur in a definite period of time. It may, therefore, be high or low. In sounds that are high in pitch the vibrations are rapid. In sounds that are low in pitch the vibrations are correspondingly slower in the same period of time. The volume or intensity of the sound depends upon the amplitude of the vibrations, and varies directly as the square of the amplitude. It is modified by the degree of force used in the production of the sound. "Duration" explains itself. These characteristics bear certain relationships. Sounds that are high in pitch are of diminished volume or intensity, and of short duration. The accompanying diagram shows the relation of the characters of the sound. (See Fig. 137.) On the other hand, sounds that are low in pitch have

FIG. 137.



Diagrammatic sketch of the relations of the character of tone. The perpendicular line represents the pitch. The transverse line the volume and duration.

correspondingly greater volume or intensity and longer duration. The three characteristics determine the quality of the sound. The term "clearness" is applied to sounds which have the character of tones. They are low in pitch, of good volume, and long duration. Sounds that are high in pitch, of small volume, and short duration are of a *dull* quality. Noises, highest in pitch and least in volume and duration, are absolutely dull or *flat*. The former are indicative of the presence of air; the latter, of the absence of air. The tones, or clear sounds, are naturally produced over structures containing air. The

production of a tone implies the presence of air in a sac. Structures in which the proportion of air to solid material varies yield sounds which vary between clearness and muffling, to absence of tone or dulness. Resonance and tympany are clear sounds which will be explained later.

Method of Procedure. Due attention should be paid to the presence or absence of tenderness, which necessarily modifies the results obtained by this method of exploration. Definite information can be secured by light percussion, even when there is a good deal of tenderness. In children percussion should be the final step in the examination.

Immediate Percussion. The chest may be tapped by the finger or hand directly. This was the original method of percussing the chest. It is known as the *immediate* method. When the fingers are employed it is known as *palpatory* percussion. One finger is sufficient. The pulp, as most sensitive, may give the blow. Or the tip, the finger bent at a right angle, may be used. By this method the sense of resistance is better appreciated.

Mediate Percussion. The method now employed is that in which a medium is placed between the chest-wall and the instrument used for percussing. This medium is called a plexímeter. It may be a small plate of ivory of suitable size to place between the ribs, or, better still, the fingers of the hand not used in tapping. The plessor is used to create the sound. It may be a small hammer. The one usually selected is of moderate weight, has a firm, light, slightly flexible handle and metal head, the poles of which are tipped with rubber. For purposes of class demonstration a plessor of this character, with an ivory plexímeter, is of value; but for bedside-work the fingers of the physician are better.

The Use of the Plexímeter. The plexímeter must be placed in close contact with the surface of the chest in performing percussion. If the finger is used as a plexímeter, in percussing the anterior portion of the chest, for instance, it must be placed parallel with the ribs. It must not cross them. If it is not in close contact with the chest, the cushions of air between the two will modify the sound, so that accurate data are not obtained. Interspace after interspace should be percussed in this manner from above downward. At the same time, if necessary, the plexímeter may be placed over the corresponding ribs, but parallel with them. With a little practice the method of applying the plexímeter can soon be acquired.

The Use of the Plessor. This requires considerable practice on the part of the student. If a metal instrument is used, care should be taken to acquire the habit of percussing under all circumstances with the same degree of force. If the finger of the operator is employed as a plessor, several points in the procedure must be remembered. It is better to use one finger, preferably the middle finger. Some operators use more than one finger, but with a little practice a sufficient degree of force can be given with one to elicit the sounds essential for distinction. The finger should be bent at right angles and kept in a fixed position. It must be made to strike the plexímeter perpendicularly

to its plane. If the blow is given at any other angle to the part percussed, a true sound cannot be obtained. The blows must be regular and the force even. The character of the part investigated will determine the degree of force that should be used. (See Method of Percussion, page 493.) The force of the blow is to come from the wrist alone, neither the arm nor the forearm must come into play. Beginning anteriorly with the supraclavicular fossæ, and proceeding downward an interspace at a time, *comparison* should be made with the other side at each step. The axillary portions, and the posterior portions from supraspinous fossæ to base, should then be examined in the same way.

Hearing and Feeling Combined. Another excellent plan is to secure information by the sense of touch, as well as by the sound. The second, third, and fourth fingers of the percussing hand are flexed at an angle of 45 degrees. The tips are brought down on the pleximeter finger and kept there for a few seconds, when the blow may be repeated. The perpendicular blow is not used. The sound produced is not loud. It is most useful in diseases of the lungs, spleen, and liver, and where strong percussion cannot be used, as in perityphlitis and cholecystitis.

Position of the Patient. The best position is the standing one, with the arms allowed to drop loosely at the sides, the head straight, not thrown back, and the shoulders allowed to fall a little forward if they are inclined to do so. Any position which throws the chest-muscles into contraction tends to defeat the object of the examiner who seeks to elicit the chest-sounds. In percussing the posterior portions of the chest it is desirable to have the patient stoop forward with arms folded. While this renders the muscles more tense, it has the advantage of exposing a larger portion of the chest.

When the patient is confined to bed he should, if not too ill, be allowed to sit up during percussion, as contact with the bed or with pillows deadens the sounds elicited. This fact should be borne in mind when from any cause it is not desirable to have the patient sit up.

All clothing should be removed, if possible. A thin undershirt may be permitted from motives of delicacy, or parts only of the chest be exposed at one time if there be danger of chill.

The Sounds in Health. Four types of sounds can be produced by percussing over the trunk for the purpose of study. 1. Resonance over the lungs. 2. Tympany over the cæcum. 3. A modified tympanitic or so-called tubular or tracheal sound over the trachea. 4. Dulness over the heart. Modifications of these types represent all sounds produced under every variety of circumstances. They will be considered in the order of their importance. The term *resonance* is applied to the *clear* sound that is produced over the lungs on percussion. It is due to the vibration of the chest-walls and of the air in the bronchi. "Pulmonary resonance" is a term also used to indicate the same sound. While, as stated above, the sound produced is called a *tone*, yet on account of the relation of the air to the solid structure of the lung, the air being confined in innumerable sacs, a true tone is not produced—*i. e.*, the sound cannot be pitched with another tone or made to vibrate in unison with one. For practical purposes, however,

the term "tone" may be used convertibly with "clearness" and "resonance." Its characteristics cannot be defined accurately, and must be learned by repeated practice.

Modifications in Health. The degree of clearness or resonance differs in various parts of the thorax. It is purer in the upper axillary region, at the angle of the scapula behind, and on the anterior surface of the chest, in the second interspace. It is slightly higher in pitch at the right than at the left apex. It is modified by the condition of the chest-walls. Thick chest-walls, accumulations of fat, the mammary gland, and the scapulæ impair the resonance and necessitate deep percussion to bring out the true sounds. In persons with thin chest-walls the resonance is clear and more pronounced. The elasticity of the chest-walls also modifies it. In the aged it is less clear because of rigid chest-walls. In children, in whom the chest-walls are elastic, the resonance is much fuller or clearer, and approaches more nearly the character of a tone. The sounds vary, within certain limits, in different individuals with perfectly healthy, normal chests, as may be seen from the above. Moreover, a sound normal in one part of the chest may in another part indicate disease.

It follows that percussion-sounds do not have an absolute value; their significance depends upon the individual and upon the part of the chest examined. The student should learn from the outset to compare the sounds developed by percussion of symmetrical portions of the chest, and thus determine the normal for the individual. Below the third rib on the left side the dulness of the heart destroys the value of comparative percussion. *Significance:* Excess of clearness or resonance—hyper-resonance—means excess of air, as in vicarious emphysema. Diminution of clearness means diminution of air—increase of solid structure.

Abnormal changes in resonance caused by disease will be considered later.

Tympany. When a single cavity with smooth walls, containing air, is percussed, the sound that is produced is a tone of low pitch, of considerable volume or intensity and of long duration. The term "tympany" is applied to this sound. In health it can be elicited over the stomach when it is free from food, over the large intestine, and at times over the small intestine. In addition to the low pitch and large volume, it possesses a peculiar metallic quality which is characteristic. It may be said to be a "hollow" sound. It is a quality of sound with which the student should become familiar, for variations are characteristic of abnormal physical conditions in the lung and in the abdomen. It must be remembered that tympany can be developed normally over the posterior portions of the lungs of infants and children. The relation of this sound to resonance, or the sound produced on percussing the healthy lung, and to dulness produced over airless structures, may be appreciated by reference to the diagram modified from Gee. (See Fig. 137.) In pitch, in volume, and in duration it is lower than the resonant and tracheal tones. The latter stands midway between tympany and dulness. As intimated previously, all varieties of sounds that may be produced, and which occupy

positions between the extremes noted in the triangle, are dependent entirely upon the proportion of air to solid material.

The *tracheal tone* is a clear tone produced over the trachea when the mouth is open moderately. It is clear, higher in pitch than resonance, and of a tympanite or tubular quality.

Dulness. The sound over the heart is dull, and may be useful to compare with dull sounds yielded over areas usually resonant. If a dull sound has some pitch and duration, some tone is mingled with it. If dulness is absolute, it is without pitch and is a noise. The significance of dulness has been described; it means the absence of air. *Absolute dulness* implies that the airless part underneath is in immediate contact with the surface of the chest. *Relative dulness* implies the interposing of air-containing structures between the airless structure and the chest-wall. The portion of the heart or liver in contact with the chest-wall yields absolute dulness when percussed; the portion overlapped by lung yields relative dulness. Absolute dulness is readily elicited, and with ordinary percussion is a fixed area. All observers will usually secure the same size of absolute cardiac dulness, for instance. Relative dulness depends so much upon the method of percussion, light or strong, and upon the ear of the observer, that for its extent each observer will have a different opinion. The personal equation is a disturbing factor in the estimation of its extent. It must be remembered in disease of the lungs, of the bloodvessels, and mediastinum the location of the lesion is usually made out by the detection of relative dulness, or of changes in the pitch, quality, and duration of the sound, indicating less air in the part percussed. Such changes are more diagnostic if the effects of breathing (respiratory percussion), of the position of the patient, and of the force of percussion (light or strong) are considered.

The Pitch. The estimation of the pitch of the sound is of the highest importance. It is the one distinctive attribute or characteristic which is of special diagnostic significance as to the physical condition of the part. It requires considerable practice to estimate it correctly.

Its significance in relation to dulness and tympany has been mentioned. Although a high-pitched sound may be considered a dull sound, this is not necessarily so. A sound of high pitch need not be markedly dull—indeed, it may be moderately clear. Under the right clavicle in health the pitch is higher than under the left, but not dull in character.

The student may become familiar with the pitch, and with alterations in it, by percussing over a portion of the lung clearly resonant, as in the third interspace and thence downward on the right side. As the interspaces in apposition with the liver are reached the pitch changes. The fulness of the sound is lessened; it becomes more shallow. The increase in rapidity of the vibrations can almost be appreciated, and, as they increase, the heightened pitch caused by them is recognized. This normal increase in pitch is due to a thin layer of lung backed up behind by the solid liver. Change in pitch makes it possible to outline organs and pursue topographical percussion.

The Degree of Resistance. This is estimated by the sense of touch. When organisms containing air are percussed the resistance

appreciated by the finger percussed is small, or, indeed, may be said to be absent entirely. The sensation of the finger is as if the parts underneath bounded away. When the air decreases and the proportion of solid structure increases more resistance is felt. It is of the greatest importance to carefully educate the finger in this sense of resistance. It is often difficult to determine the pitch exactly, and the sense of resistance furnishes an additional means of detecting the presence or absence of solid structure. Palpatory percussion indicates the sense of resistance to a better degree than any other method.

Superficial and Deep Percussion. In superficial percussion the blows are directed lightly over the part percussed, so as to bring out the sound yielded by the portion directly underneath the surface. Hence superficial percussion is applicable over the thinner portions of the lung. It enables one to bring out areas of absolute dulness. Light percussion is necessary in children and in patients with sore chest-walls, or when they have just had a hemorrhage. In deep percussion the blows are given with enough force to influence the structures situated deeply in the lung or overlapped by the edges of the lung. It is necessary, therefore, in cases of deep-seated consolidation, and in cases of aneurism covered by lung, in order to define its limits. It is employed to determine the true height of the liver and the relative area of dulness of the heart.

Auscultatory or Stethoscopic Percussion. This is a valuable means of defining the exact outline of a dull area, as an aneurism or tumor within the chest, or of determining the limits of organs even of similar physical structure. The stethoscope is placed over the organ the border of which is to be defined, and percussion is begun some distance from it. It is conducted toward the stethoscope, and the dull sound of the non-resonant structure is transmitted to the ear beyond limits not determined by ordinary methods. If the tympany of the stomach is to be distinguished from the tympany of the colon, place the stethoscope over either one of the organs. Percuss with the finger-tips directly on the surface by immediate percussion. Begin at the stethoscope and percuss from it. As soon as the limit of the structure percussed is reached a difference of tone or pitch is observed which cannot be detected by other means. In this manner the dulness of the liver can be told from that of pulmonary consolidation or pleural effusion; the dulness of an effusion from a consolidation of the lung which rises higher than the effusion, as in pleuropneumonia. Mediate percussion may also be employed.

Respiratory Percussion. (Da Costa.) The difference in the sound elicited in full inspiration and in full expiration is marked in health. In general it may be said the sound becomes more resonant and higher in pitch in full inspiration. In ordinary bronchitis the same change is observed as in health; on the other hand, in bronchitis with much secretion and in bronchopneumonia the marked difference between inspiration and expiration does not hold. In phthisis the difference between the two sides of the chest can be made more plain by respiratory percussion. By the varying changes in pitch and duration, cavities are detected. (Gerhardt's sign.)

Object of Percussion. The object of percussion is to estimate the proportion of air to the solid tissue contained in the chest. We can thus determine (1) the size of the lungs; (2) the presence or absence of disease causing abnormal physical conditions; (3) the size of the other organs in the thorax (topographical percussion), and (4) in the case of the abdomen the position and size of its organs and the presence of tumors or other solid structures.

The Size of the Lungs. *Increase* in size: The boundaries of the lung have been described. If the resonance extends beyond these boundaries, it may be said that the lungs are enlarged. This is seen in *emphysema*. The area of resonance in this affection extends above the clavicles to a greater height than in health. It encroaches upon, and may altogether displace, the normal area of cardiac dulness; it extends one and a half to two inches beyond the lower limits of the healthy lung. The upper border of liver-dulness is, therefore, lower—instead of beginning in the fifth or sixth space it begins an inch or two below. *Diminution* in size: Shrinkage of the apices (one or both) takes place in phthisis, hence the resonance of health does not extend as high up in the neck. Shrinkage or contraction may take place along the lateral borders or lower edges, on account of phthisis or retracting pleurisy, causing diminution in size of the lung and spurious enlargement of the heart or liver. In diseases below the diaphragm, effusion or enlarged liver, the size of the lungs varies. (For heart and liver, see the special chapter devoted to these organs.)

The Sounds in Disease. It may be said in general that when a sound is produced in the thorax which varies from the normal resonant tone it indicates an abnormal physical condition, or, in a word, disease. Exactly corresponding portions of the two sides must be compared.

Change in tone may be general or local. The areas over both lungs may yield a different percussion-note from the normal (bilateral); the change may be limited to one side (unilateral); or it may be found in small areas (local).

Increased Resonance or Tracheal Tone. The resonance may be increased or diminished. When the resonance is *increased* the sound is abnormally clear. If it is fuller and clearer than in health, without the characteristics of the tympanitic note, it is known as hyper-resonance or exaggerated resonance or a tracheal tone. The physical condition which causes exaggerated or hyper-resonance is increase in the amount of air. This increased amount of air may be general, unilateral, or local. When general (*bilateral*) it gives the characteristic sound heard in emphysema. In this affection the amount of air is so great, and the tension of the chest-walls so exaggerated, that hyper-resonance and sometimes a pure tympanitic sound ("band-box" resonance) are produced over the entire thorax. At the same time normally dull areas are encroached upon. The heart-dulness is effaced, the liver dulness lowered. The same increased resonance may be present in acute miliary tuberculosis. *Unilateral* increase in resonance or tympany occurs when there is an increased amount of air in one lung, on account of compensatory enlargement (vicarious or compensatory emphysema), or on account of an increase of air in the pleura. *Local*

increase of resonance occurs when a local area of the lung is acting in a compensatory manner. This is seen in cases of phthisis in which the alveoli or lobules surrounding small areas of consolidation are very distended. The exaggerated note may aid in the recognition of a deep consolidated area. The same note, hyper-resonance, or skodaic resonance, is obtained over a portion of the lung above the line of pleural effusion, and above the line of consolidation in pneumonia.

FIG. 138.

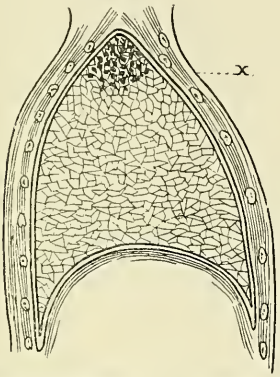


Diagram showing at x moderate dulness over tubercular infiltration. (GIBSON and RUSSELL.)

FIG. 139.

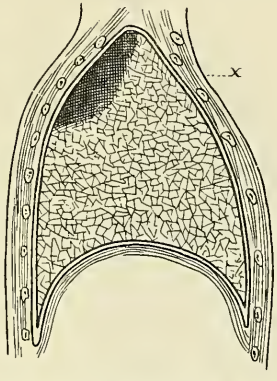


Diagram showing heightening of pitch anteriorly at x from consolidation posteriorly (shaded points). (GIBSON and RUSSELL.)

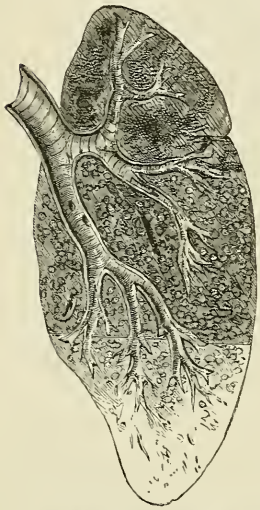
DIMINISHED OR IMPAIRED RESONANCE. The normal tone or resonance is impaired or muffled—that is, the pitch is higher, while the volume is lessened and the duration shorter—in cases of incipient consolidation of the lung, and in small pleural effusions when a thin layer overlaps the lung. It is the first change toward dulness. It is particularly noted in the early stages of phthisis, when the lung area, usually the apex, is the seat of small areas of tuberculous infiltration. The relative amount of air to solid structure is lessened. Impaired resonance is the result. As the disease advances the note changes gradually to dulness.

PITCH. Gibson and Russell have pointed out the change in quality of sound with change in pitch. (See Fig. 139.) If, for instance, the apex of the lung is percussed in front, when there is an effusion of fluid behind, or a consolidation of small area directly on the opposite surface of the lung, the pitch is higher, compared with the sound in the opposite lung at the corresponding point, although the quality is clear. A clear sound of heightened pitch is diagnostic of airless structure behind air-containing structure.

TYMPANY IN DISEASE. SIGNIFICANCE: If a tympanitic note is elicited over a part where in health resonance should be found, it is an indication of disease. It signifies (1) that air is confined in a space (cavity), or that there is an excess of air in many sacs, as in the lungs in emphysema; (2) that the tension of the lungs is less than normal—the lung is relaxed, as it is above the limits of a pleural effusion. A

tympanitic sound from the chest occurs—1. *Bilaterally,* in cases of emphysema. 2. *Unilaterally,* in cases of pneumothorax and compensatory emphysema. In pneumothorax the pitch may be raised if there is much tension; it is then known as a dull tympany. 3. *Locally.* *a.* It is limited to the lobe of the lung in some cases of compensatory emphysema. *b.* It may occur in the early stages of pneumonia, or in the later stages of complete consolidation. In the former it is due to relaxed tension; in the latter, to the air in the bronchus, the lumen of which is free. *c.* In cases of pleural effusion, owing to alteration in the tension of the lung, a tympanitic note is present above the layer of fluid. *d.* In phthisical excavations at the base of the apex, and in bronchial dilatation, if the cavity communicates with the air, and has moderately thin, elastic walls, and is at the same time empty, a tympanitic note is produced. The musical pitch of the note depends upon the volume of air, the size of the opening, and tension of the wall. Large volume of air, low pitch; large opening, low pitch; greater tension, higher pitch. Small volume, high pitch; small opening, high pitch; less tension, low pitch. (For modifications of tympany, see Special Sounds and Cavities.)

Fig. 140.



At the apex complete dulness and bronchial breathing, from tuberculous consolidation; in the middle portion impaired resonance, from disseminated tubercles; below exaggerated resonance, from compensatory emphysema.

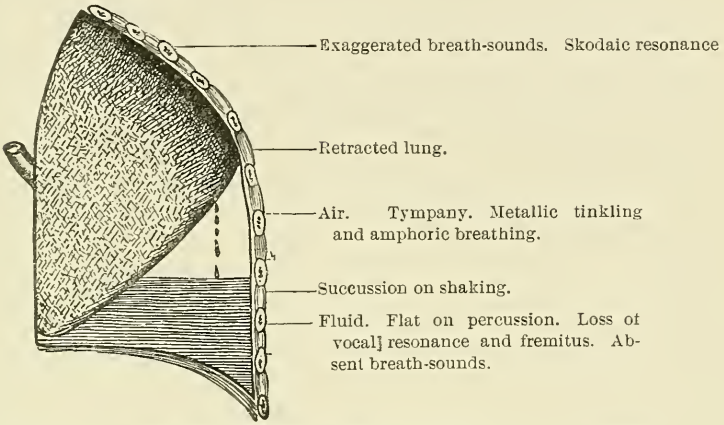
Dulness in Disease. The note is high in pitch, small in volume, and short in duration. Absence of air, or a relatively small amount in proportion to solid structure, is present. The conditions which give rise to it are all forms of consolidation and pleural effusions. The extent and the degree of dulness depend upon the proportionate amount of solid to air-containing material. Moderate dulness is seen in tubercular disease, with moderate infiltration of the lung (see Fig. 138), and in small patches of catarrhal pneumonia, in pulmonary congestion, and in atelectasis and physical conditions in which there is solid material in greater proportion than in health. *Absolute* or *complete dulness* occurs when the air is completely absent, as in the stage of hepatization of acute pneumonia, in hemorrhagic infarction, in condensation from pressure, in pleurisy with large effusion, or great thickness of the pleura, and in tumors. *Flatness* is applied to the extreme degree of dulness. (See Fig. 141.)

We have, therefore, all gradations of the dull sound, from simple impaired resonance in incipient tuberculosis of an apex of the lung, as determined by careful comparison of the two apices, to absolute flatness or deadness.

Method of Percussion: The kind of percussion necessary to bring out the dulness will depend upon the extent and the distance from the surface of the disease. When the consolidation or thickening is super-

ficial, light percussion will discover it, whereas strong percussion would bring out the resonance of the deeper healthy lung-tissue to such an extent as to mask completely the superficial dulness. On the other hand, when the airless consolidated tissue is deep-seated and surrounded by healthy lung, strong percussion is required to discover it.

FIG. 141.



Pneumothorax; resonance over retracted lung. Tympany over air. Dulness or flatness over fluid. (GIBSON and RUSSELL.)

Again, when the airless tissue occupies a small focus and is surrounded by healthy lung, as in pneumonia beginning centrally; and when there are small airless foci, perhaps surrounded by emphysema, as occurs sometimes in disseminated tuberculosis, percussion is often wholly negative.

SPECIAL SOUNDS. Special percussion-sounds, or sounds the quality of which differs from the ordinary tympanitic sound, are present in some physical conditions. Of these the *amphoric*, or *metallic*, and the *cracked-pot* percussion-sounds are most familiar. The *amphoric sound* is tympanitic, but has a metallic clang, or echo, which is an overtone. The prolongation of the sound is compared to an echo. It is like the sonorous ring of the voice when one utters a tone in an empty hall. It can be imitated by percussing an empty vessel. It is heard best in cases of pneumothorax (see Fig. 141) and in phthisical excavation when the cavity is large, superficial, with smooth walls, and when it has open communication with a bronchus. The *cracked-pot sound*, as the name indicates, resembles that produced when a cracked metal vessel is tapped; it is simulated by clasping the hands loosely at right-angles to each other and striking them over the knee. It is heard best over cavities which communicate directly with a bronchus, especially if the chest-wall is thin and yields to the percussion-stroke. The cavity is usually at the apex. In order to elicit the sound the patient should be made to keep the mouth open. The sound should be created at the time of expiration, and the percussing finger should be retained instead of elevated after striking the plexímeter.

In some rare cases this sound can be elicited in health. It may be generated if the chest of a healthy screaming infant is percussed. In this instance it is due to the compressed air forcibly throwing the vocal cords into vibration. The other pathological conditions in which the sound occurs rarely are pleurisy, when the chest is percussed above the effusion, pneumonia before consolidation has taken place, and pneumothorax if there is a free communication between the cavity and a bronchus. In the latter instance the sudden rush of air into the bronchus produces this sound. This is proved by the fact that it can be created when the chest is percussed in a case of empyema, after the fluid has been evacuated by a free incision. It is to be noted that, while corroborative, it is not of itself positive evidence of any single condition.

Auscultation. Sounds are produced in the act of breathing. They are heard by the application of the ear directly to the chest-wall or through some medium. They are created both in inspiration and in expiration. They vary in character in accordance with the situation.

Method. If possible, the patient should sit upright in an easy, unrestrained position. For auscultation in front, the arms should hang carelessly by the side. For auscultation behind, the patient should fold the arms and lean slightly forward. For comparison both sides should have the same freedom of movement, which would not be attained if the patient assumed a lateral or side posture or attitude. Auscultation should be practised in quiet, in full and in forced inspiration and expiration.

Auscultation is practised by two methods: First, the ear is applied directly to the chest, a thin towel or napkin free from starch alone intervening. This is known as the *immediate* or *direct* method. It is of service to ascertain the general character of the sounds. It has the disadvantage of imperfect localization. Second, by means of the stethoscope and phonendoscope the *mediate* or *indirect* method is practised; but it is disadvantageous in infants, because they cannot be kept quiet or are sensitive to its pressure, and in children because instruments are alarming.

The advantages of the stethoscope over direct methods of auscultation are seen when it is necessary to localize sounds. The definite localized area in which the sound is produced can be ascertained, and sounds in close proximity differentiated. Its use is essential in the study of heart-sounds. In addition, the operator is more likely to escape from contagious diseases and vermin. Moreover, on the score of delicacy, the stethoscope is preferable.

The stethoscopes used are single and double, and vary in form with the practice of the operator. It should be an absolute rule with the student to become familiar with and use one form of stethoscope only. The single stethoscope is very good to localize and determine the relation of sounds. It also transmits the shock of an aneurismal vessel or of the heart. The objection to it is that the weight of the head causes pain if the chest is sore, and the pressure of the instrument may modify sounds if bloodvessels are auscultated, or sounds in close proximity to the ear, as a friction. In the use of the single stetho-

scope the student should be particular, first, to see that the portion applied to the chest is perpendicular to the plane of the area over which auscultation is practised. Otherwise slight tilting of the instrument will take place and outside noises be transmitted through the tube. The operator should place himself in an unconstrained position and see that his head is accommodated to the position of the instrument, not the latter to the head. If the parts over which auscultation is practised are covered with hair, an extraneous sound from friction is produced. Oil should be applied to obviate this. The double stethoscope is the most suitable for class instruction. It can even be applied over parts that are quite tender. The rule of application to the chest is the same as for the single stethoscope. The ear-pieces should fit comfortably. The humming sound in the tube is confusing at first.

The Sounds in Health. It may be well to call attention to the confusion that always arises when the student is examining the chest for the first time. The probability is that the coincidence of heart-sounds and lung-sounds in the chest prevents the discrimination of the latter sounds. If attention is paid to the respiratory rhythm they can be distinctly isolated. When the student is auscultating the lungs he should place his hand on the thorax or the epigastrium and fix his attention upon the two acts of respiration—inspiration and expiration. Note the occurrence of each movement, the expansion of inspiration and the contraction of expiration Then analyze carefully the sounds during each event of a respiratory act. Having fixed the attention on respiration, noted its divisions, and excluded cardiac rhythm, note (1) the character of the sound in inspiration ; (2) the character of the sound in expiration ; (3) the relative length of the two. By this means the sounds of respiration are accurately ascertained, and confusing extraneous sounds, as from the heart, distinctly eliminated.

Bronchial Breathing. If the stethoscope is placed over the trachea at the top of the sternum, a sound characterized as follows will be heard : First, it attends inspiration and expiration with a definite pause between ; second, the inspiration and expiration are nearly equal in length ; third, they are of a tubular, blowing character. The expiration is perhaps a little stronger and longer than the inspiration. If the mouth is closed, there is no change except that both inspiration and expiration are harsher and sharper. *Bronchial breathing* is the term applied to the sound which is heard in this situation. It is one of the normal sounds of the chest. It may be heard behind, at or a little below the seventh cervical vertebra, feebler in quality than in the trachea, and in the interscapular space over the large bronchi as they leave the trachea. A sound heard in these areas, bronchial in character, is normal.

Vesicular Breathing, or the Respiratory Murmur. If the ear is applied over the anterior portion of the chest, or, better still, in the upper axilla or below the angle of the scapula behind, a sound is heard both on inspiration and expiration. It differs from bronchial breathing, however, in that inspiration and expiration are changed in length. The sound of inspiration is twice or three times as long as the sound of expiration. The sound of inspiration is soft, breezy, or

sighing in character, increasing in intensity to the end of full inspiration. It is immediately followed by expiration, which diminishes in intensity as the air is expelled, and terminates when one-half or two-thirds of the expiratory act is completed. The sounds can be imitated by breathing with the lips in the position required to pronounce "f" or "v."

Cause of the Sounds. The sound is caused by the passage of air through the nares into the wider pharynx when the mouth is closed. The sounds heard over the bronchi, the terminal bronchioles, and the vesicles are probably created in the upper air-passages and transmitted to the ear through the medium of the bronchi. Bronchial breathing is the sound unmodified, transmitted to the ear, weakened only by its distance from the upper air-passages. The vesicular breath-sound is the same sound modified on account of the intervention of the air-vesicles between the ear and the larger bronchi. The sound is thus smothered or dampened down. It was held that part of the sound of vesicular breathing, if not the whole, was due to expansion of the vesicles and rush of air through the bronchioles. The proof, however, seems to be in favor of the first view given, chiefly because, when the vesicular tissue is removed, as in pneumonia or other consolidation, even far distant from the trachea, bronchial breathing is produced.

Modifications of the Sounds in Health. *Exaggerated Breath-sounds.* Bronchial breathing and vesicular breathing are increased in loudness and sharpness by strong, rapid breathing. In some persons a sound is heard which partakes of the qualities of both bronchial breathing and the vesicular sound. It is noticed in the interscapular region about the level of the spines of the scapulæ, replacing the pure bronchial breathing which is heard in other individuals. Its characters are, first, soft, blowing inspiration, or loud, harsh inspiration; second, slightly prolonged blowing expiration, more exaggerated, louder, but not harsher, than in health. The term *broncho-vesicular* is applied to this kind of breathing. It is due to the fact that the sound produced in the upper air-passages is conducted to the ear less dampened down or modified, because the air-vesicles which surround the bronchus are here smaller in number than are found in the remainder of the lung.

The sounds are increased in children, in whom there are combined greater elasticity of the chest-wall and greater friction throughout the smaller bronchi, which are relatively larger. So distinct and characteristic is the sound in children that the term *puerile* respiration is applied to it. The sounds of inspiration and expiration are both intensified or sharper than in healthy adults; the latter is relatively prolonged.

Feeble Breath-sounds. The sounds are modified by the condition of the chest-walls. If they are thick, or there is an abundance of fat, the sounds are fainter or lessened in intensity. Feeble respiratory power, in wasting and exhausting diseases, causes feeble breath-sounds. The condition of the upper air-passages, even if not pathological, modifies the sound. If the glottis is small, or there is a disturbed relationship between the nose and pharynx, the sounds will be modified. They are usually weakened.

The Sounds in Disease. It is well for the student to bear in mind that sounds heard in the chest which are departures from the normal sounds always indicate disease.

Vesicular Breathing Exaggerated. *Bilateral.* The vesicular breathing or respiratory murmur is increased, first, when there is increase in the force of breathing—when normal respiration is increased and the patient takes full, deep breaths. It is seen in some forms of dyspnœa, as at the acme of Cheyne-Stokes breathing, or in the dyspnœa of diabetic coma. It may be increased or exaggerated in certain forms of bronchitis, particularly when the small tubes are narrowed by inflammatory swelling.

Unilateral exaggeration or increase of vesicular breathing is heard when the lung is acting vigorously or in a compensatory manner. The strong inspiration followed by strong and relatively prolonged expiration of an actively moving lung signifies almost certainly disease of the lung of the opposite side.

Local exaggeration of vesicular breathing, the inspiration harsh, is noted in cases of phthisis in its earliest stages. It should be compared with the sound of the opposite side, when the difference can easily be ascertained. It is heard over the apex, in pneumonia or pleurisy of the base, and *vice versa.*

Vesicular Breathing, Diminished or Absent. *Bilateral.* (1) It is lessened in all cases in which the expansion is interfered with. In feeble persons the respiratory murmur is weak, particularly at the bases posteriorly. If the muscles of respiration are paralyzed or enfeebled, the murmur is also lessened. If the expansion is interfered with, on account of disease of the diaphragm, or pressure upward by accumulations in the abdomen, it is weakened.

(2) Anything which lessens the amount of air supplied to the chest diminishes the vesicular breathing. It is, therefore, lessened in cases of occlusion or obstruction of the nares, the pharynx, or the larynx.

(3) Thickened chest-walls that occur from disease, as œdema, weaken the respiratory sound.

(4) The vesicular breathing is weakened throughout the entire extent of the lung in emphysema. The enfeebled respiratory forces and the short act of inspiration in this affection cause less air to enter the already overfilled chest. Moreover, in the bronchitis that attends emphysema the bronchioles are all more or less occluded, and hence the air-supply is diminished. These conditions lead to feeble respiratory murmur except at the anterior margins of the lungs.

Unilateral diminution of breath-sounds occurs (1) when there is narrowing of the bronchus, as in cases of aneurism or mediastinal tumor; (2) when there is pleural effusion, which (*a*) lessens the amount of air-pressure by compression of the lung and (*b*) interferes as a different conducting medium. (See Fig. 136.) If pain in pleurisy, pleurodynia, or neuralgia is present on one side, the breath-sounds of the affected side will be lessened. Not only in pleural effusions from serum, blood, pus, or air, but also in thickened pleura there is weakness or faintness of the respiratory murmur. It should not be forgotten that effusions and thickenings of the pleura rarely take place bilaterally; when they

do occur the breath-sounds are weakened, but not to the same extent as when an effusion is limited to one side.

Local diminution of breath-sounds occurs in the early stages of phthisis or in the earliest stages of pneumonia.

Alteration of the Rhythm. We take cognizance of the rhythm of the sounds. In health the movement of inspiration and that of expiration are almost equal, but, as previously noted, the sound of inspiration is heard during the entire act, while that of expiration occupies the first third or so of the act. The sound produced during expiration may even be less than half the length of that produced during inspiration. The following proportion represents relative lengths—Ins. : Exp. : : 3 : 1.

Expiration Prolonged. The first notable change in the rhythm of respiration may be prolongation of expiration. When the expiration is prolonged it equals inspiration, or may even be longer. This is due to the difficulty of getting the air out of the chest—expiratory dyspnœa, a physical condition which enables the sound of expiration to reach the ear. Hence, prolongation of expiration all over the chest is seen in emphysema and asthma. In this condition the inspiration is short, the expiration prolonged. Although distinct throughout the chest, it is more pronounced above the clavicles and along the free margins of the lung anteriorly. It is prolonged in bilateral bronchovesicular breathing (*q. v.*).

Local prolongation of the expiration is of great diagnostic significance. It occurs when areas of the lung are partially consolidated and the elasticity thereby impaired. The respiratory murmur is harsh, or puerile, or it may be weak. This condition obtains in tuberculosis, and is one of the first physical signs of this affection.

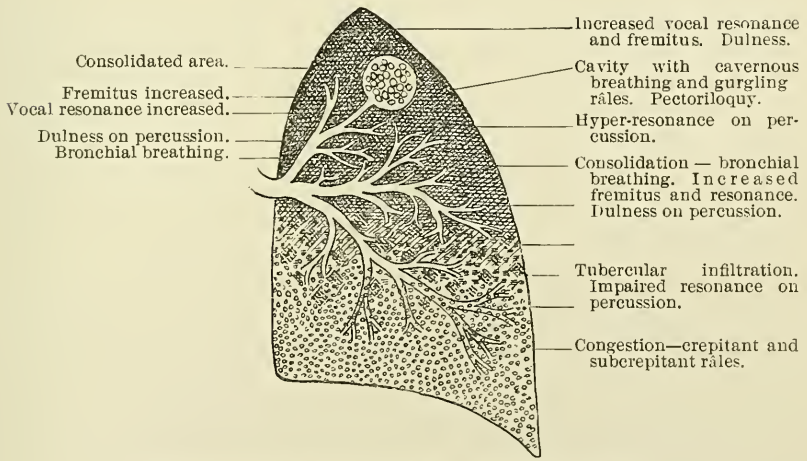
Jerking or Interrupted Inspiration. Instead of the smooth, even, sighing, or breezy inspiration the sound is created in puffs or jerks, so that during the act of inspiration, as the chest expands, a number of successive vesicular sounds are heard until the act is completed. The physical condition which causes jerking inspiration, or cog-wheel breathing, is found in the earlier stages of tuberculosis, when the various bronchioles are more or less occluded by outgrowths of tubercle. The air, therefore, enters different lobules at different periods of time, thereby giving rise to this peculiar broken sound. It must not be confounded with the same character of breathing that is heard adjacent to the heart, due to the pressure of that organ, or of structures in intimate relation therewith, upon portions of the lung, on account of which air enters various areas in puffs. On the other hand, jerking inspiration sometimes occurs in health. It is simulated by the jerky act of inspiration in nervous patients. It is of no significance unless attended by other physical signs.

In cases of adhesion at the apex, particularly of the left lung, the same puffing or jerking inspiration is often heard. It is also present in aneurism, or disease of the aorta, pressing upon a bronchus, causing the air to enter the part in an intermittent manner. When pathological jerking breathing is present, the expiration is prolonged, and, if the case is under observation a sufficiently long time, bronchial breathing

will usually replace the jerky respiratory murmur in progressive consolidations. Small, moist râles, excited by coughing or a full breath, usually attend jerking breathing when it is pathological.

Bronchial Breathing. The normal situation of bronchial breathing in health has been indicated. If the same kind of breathing is heard in any other portion of the lung it is pathological. It is generally indicative of the presence of consolidation. The spongy lung-tissue is replaced by solid conducting material, by which the bronchial sound is conducted to the ear. It is heard, therefore, in all pathological conditions in which consolidation takes place. It is the typical form of breathing heard in pneumonia, in consolidation of the lung due to tuberculosis (see Fig. 142), in hemorrhagic infarcts, and in lung syphilis. It must not be forgotten, however, that cases of pneumonia do exist without this type of breathing. This is the case when the large bronchus supplying the lungs, or the bronchioles, are occluded by inflammatory exudate. In tuberculous consolidation it may be absent for similar reasons. In central pneumonia, where consolidation is deep-seated and surrounded by lung-tissue, bronchial breathing may not be heard, or it may be postponed until the third or fourth day of the disease, by which time consolidation will have reached the surface of the lung.

Fig. 142.



Showing phthisis at various stages. (Gibson and Russell.)

In certain cases of pleurisy with effusion bronchial breathing exists. The accumulation is not great enough to compress the lung completely. The bronchial tubes remain patent, while the vesicular structure is compressed. Low-pitched bronchial breathing is heard under these circumstances. It is more pronounced at the upper layer of the effusion. It is always heard close to the spine posteriorly, where the lung is compressed. Sometimes it is heard above the limit of the effusion, in all probability because of relaxed tension of the lung.

Varieties of Bronchial Breathing. Its special characteristics must be borne in mind. (See p. 503.) It must not be forgotten that

bronchial breathing is not represented accurately in every instance by the sounds heard over the trachea. Its character may be modified and yet approach that type of breathing. The modification occurs in one or both of the two portions that go to make up the sound: (1) The blowing element may not be as distinct in inspiration as in expiration; (2) in rare cases, the characteristic blowing sound may not continue so long during expiration as to equal the inspiratory sound. On the other hand, (3) the bronchial breathing may vary in pitch. At times it is (*a*) high in pitch, both in inspiration and expiration, but with a pure blowing quality (harsh) attending each. It may be (*b*) soft and low in pitch attending both acts. The strong, high-pitched sound emitted by breathing deeply when the lips and tongue are placed in position to pronounce "ch" is termed *tubular breathing*. It is the characteristic sound of croupous pneumonia. (4) The loudness of the sound may also vary. This depends largely upon physical peculiarities of the individual. The condition of the chest-walls and the force of breathing determine it.

When pleurisy with effusion coexists with pneumonia, the bronchial breathing, which should be audible, is feeble and distant. Under the same circumstances a bleating sound replaces bronchophony. (See Ægophony.)

Mode of Determination. Breathing which may, during very quiet respiration, appear to be normal, is sometimes discovered to be bronchial when the patient has a spell of coughing and then takes several deeper breaths than usual in rather quick succession. Sometimes the noise made in nasal respiration obscures the pulmonary sounds. The patient should be instructed to breathe with the mouth open, to take somewhat deeper breaths than usual, and to let expiration follow at once upon the close of inspiration. Many patients when told to take deep breaths expand their lungs to the utmost, and then hold the air in a while, and allow it to pass out slowly. Such a method usually defeats the purpose of the examiner, which is first to note the relative length of inspiration and expiration, and then the quality of the two sounds, first, as compared with each other, and, secondly, as compared with the normal. In listening for bronchial breathing the attention should be fixed more upon the length and quality of the expiratory sound, and it is, therefore, important that the patient breathe so as to bring out its characteristics more clearly; this he can do by taking several moderately deep breaths in quick succession and with the mouth open.

Modifications of Bronchial Breathing. If a case of tuberculous consolidation is watched, it will be found after a time that the bronchial breathing becomes lower in pitch. It is heard in inspiration and expiration, but a more hollow quality attends the sound. From the hollowness of the tone the word *cavernous* has been applied to the breath-sound; it is due to the formation of a cavity in the consolidation, or to a dilated bronchus. It is a sign of a cavity. (See Fig. 142.) Cavernous breathing may have a metallic quality, and is then called *amphoric*. It is analogous to the sound produced by blowing across the open mouth of a jar. A large cavity with smooth walls that com-

municates with the air is the cause of the development of such sound. It is heard also in pneumothorax, when such communication exists. The metallic tone is analogous to the metallic percussion-sound. It occurs under the same physical circumstances. The physical condition which causes it may be so marked that the same character of tone is imparted to râles produced in the cavity, or to the heart-sounds which are transmitted by the solidified area surrounding the excavation.

Bronchovesicular Breathing in Disease. The physical condition is more or less consolidation surrounded by vesicular structure, as in the early stages of tuberculosis. It is found midway in the change from respiratory murmur to bronchial breathing in progressive consolidations. The inspiration is higher in pitch; the expiration prolonged, harsh, and blowing; or the former may be bronchial or tubular, the latter absent. It may, however, be indistinct or masked by râles. It is sometimes heard in the earlier stages of pneumonia, and is the modified bronchial breathing heard over small consolidated areas in capillary bronchitis and catarrhal pneumonia, with collapse of lobules. The term "transition breathing" has been applied to this character of breath-sounds.

New Sounds. The foregoing sounds are modifications of the normal sounds heard during the act of breathing. New sounds or adventitious sounds are created in the lungs or in the pleura. In the lungs the term râles is applied to them, and in the pleura they are known as friction-sounds. Under the same head may be classified the succussion-sound and metallic tinkling.

Râles. Râles are sounds created in the bronchi, bronchioles, and air-vesicles, or in pathological excavations (cavities). They are due (1) to the passage of air through bronchial tubes which are narrowed, either on account of swelling of the mucous membrane or on account of spasm; or (2) the passage of air through fluid (mucus, serum, pus, blood). The former are called "dry râles;" the latter moist râles, or crepitation. When the dry râles are continuous—*i. e.*, heard during both the acts of inspiration and expiration—they are known as *rhonchi*. Dry râles are musical; moist râles are not. When heard over consolidated areas, the latter are, however, usually accompanied by overtones (resonance transmitted from the bronchi), and are then clear and sharp—"consonirende Rasselgeräusche," Skoda.

Dry Râles are divided into (*a*) sonorous and (*b*) sibilant. The former are large râles, the character of which is indicated by the name. They are created in the large bronchial tubes. They are coarse, low-pitched musical sounds. Sibilant râles are created in small tubes, and are high-pitched, whistling sounds. Both are heard only over the areas of their creation, although the sonorous râle may be transmitted all over the chest. They may be heard at the same time. The dry râles are heard in the early stages of bronchitis, when the mucous membrane is swollen and thickened, but has not begun to secrete mucus or mucopurulent matter. They are also heard in asthma in which there is spasm of the bronchial tubes, and in the chronic bronchitis of emphysema. In the latter the smaller râles are more common.

Moist Râles, or Crepitation. They may be divided into large or small râles; the latter are also called subcrepitant. (See Fig. 142.) The *crepitant* râle is a fine râle, said to be created in the alveoli, due to inflation of the cells, the walls of which have been held together by exudation or fluid (œdema). It is a fine râle distinctly localized, resembling the sound produced by rubbing a lock of hair between the fingers or by putting salt on a hot plate. In the early stages of pneumonia and in œdema of the lungs it is said to be pathognomonic. It may, however, be heard whenever there is a small amount of fluid in the alveoli and feeble respiratory action. The *small, moist* or *subcrepitant râles* are created in the smaller bronchioles and the alveoli. They may be general or local. If general, they are due to bronchitis in the second stage. There is an abundance of secretion in the terminal air-passages which is thrown into vibration by the current of air during the act of breathing. The element of moisture is pronounced and gives to them their quality, to which the term "crackling" is sometimes applied. They are found in congestion with outpouring and stagnation of secretion; in œdema; and whenever fluid is drawn into the bronchi, as when there has been a hemorrhage in the upper passages. Small moist râles in local areas are found in phthisis, particularly at the end of the first stage, on account of the local bronchial catarrh, and in the second stage for the same reason. They occur in the early stage of pneumonia, particularly in the area of the lung which is the seat of collateral œdema adjacent to the consolidation. They are also heard in the later stages of pneumonia when resolution has taken place. If this is reached, however, they may be replaced by large râles. They may be heard around any consolidation because of congestion, œdema, or catarrh. It must not be forgotten that cough or forced inspiration must be excited before râles can be definitely excluded.

Large moist râles, or *mucous râles*, occur in the larger bronchial tubes, or in cavities, from the same causes that produce small râles. The fluid, however, is larger in amount, the air-current stronger, and the space for vibration is greater. While sometimes present in bronchitis, they are heard in their most marked form in the third stage of phthisis. They are described as bubbling and gurgling râles, and are very characteristic after a full breath or a cough. (See Fig. 142.)

Râles are to be distinguished from other adventitious sounds. Although in some instances, as when râles are heard over the bases of the lungs, it is almost impossible to distinguish them from friction sounds, they have nevertheless certain marked characteristics. We recognize râles, *first*, by the *qualities* previously mentioned. *Second*, by their *location;* if the adventitious sounds are general, they are due to râles. *Third*, râles are *modified* by *cough* or *breathing*. They may be intensified by either act, or, after the completion of the act, may disappear entirely. On quiet breathing, in the early stages of tuberculosis, for instance, they may not be heard at all. It is absolutely necessary, before excluding them, to have the patient cough and then take a full breath. *Fourth*, they *vary* in *position*. This may occur from hour to hour. If the chest is examined in the morning, they may

be more pronounced, for instance, at the base. At another time in the twenty-four hours they are distinct at the apex. They are more likely to be present at the base if the patient is kept in the recumbent posture. *Fifth*, they *vary* in *character*. At one time small, moist râles are heard; in a short time they are replaced by larger râles. Dry râles are regularly followed by moist râles in the course of bronchitis. In a case of bronchial asthma all sorts of râles may be heard in a few hours. *Sixth*, they are distant. They seem to be further away from the listening ear than are friction-sounds.

Râles in the bronchi must not be confounded with the crepitant or fine crackling sound which is heard at the base of the lung in patients who have been ill with the exhaustive fevers and who have not taken full breaths for some time. They disappear after the patient has inspired deeply half a dozen times.

Râles throughout the lung are not, in themselves, diagnostic of any affection save bronchitis, in which, with the absence of other physical signs, their occurrence all over the chest is significant. In the absence of this affection râles at the base of both lungs are due to congestion. Râles at one apex, with failing health, point to the onset of tuberculosis.

Friction-sound. In health the two surfaces of the pleura rub together without making any sound. If they are inflamed, the surfaces are roughened, as swelling and dilatation of the capillaries produce a more or less granular surface, or because of transudation of fluid or lymph. Under these circumstances rubbing together of the two surfaces creates a sound, to which the term friction is applied. It is heard at the end of inspiration, and may continue during expiration. It is a localized sound, usually at the seat of pain; it is near the ear, and is not modified by cough or full breathing, except occasionally by the latter when repeated. It occurs in "nests" or "bunches." It may be increased by the pressure of the stethoscope. Moreover, it is a fixed sound, in that it does not disappear until effusion takes place. It may reappear again when the fluid subsides. The above characteristics distinguish it from râles. Both, however, may occur together. Although almost always of respiratory rhythm, when the pleurisy is in the neighborhood of the heart, the friction may be of cardiac rhythm. Under these circumstances it is more distinct during the act of inspiration. It is heard as a systolic rubbing, often of respiratory rhythm, along the borders of the heart.

We not only distinguish the friction-sound by the characters just indicated, but also by the presence of pain, which renders its existence more probable. Usually it is heard at the base, in the nipple-line in front, or at the angle of the scapula behind, and frequently in the axillary region.

In addition to the friction-sound of acute pleurisy, dry creaking sounds, not unlike the sounds produced when an old door is swung on rusty hinges, or when new leather is bent, are heard in cases of old pleurisy. Other physical signs of pleural adhesions are present, and a friction-fremitus is often transmitted to the hand. An old or dry friction is often heard at the apex, in the neighborhood of old cavities. It

attends both inspiration and expiration, is not modified by cough, nor has it any of the elements of the moisture that attends moist râles. The patient may be cognizant of the grating or rubbing sensation, and be able to describe the sensation during each breath. It may continue a long time after an acute pleural effusion has disappeared, and is sometimes the source of anxiety upon the part of the patient.

Pyæmic deposits in the lungs, infarction, bronchiectasis with reactive pneumonia, and pleurisy with emphysema, are first revealed by pleuritic frictions (Vierordt). At the base of the right lung they may be the first indication, or at least an early one, of hepatic abscess (Clark). The pleural friction in the hepatic region must not be confounded with peritoneal friction of respiratory rhythm. In a case of secondary cancer of the liver a friction-sound was heard in the seventh interspace from perihepatitis over a cancerous nodule.

Metallic Tinkling. The impression imparted to the listener is that of the falling of some material into fluid in a hollow space. The physical condition is that of a cavity partly filled with fluid, partly filled with air, into which there is dropping from an opening above. It is seen in hydropneumothorax or pyopneumothorax and in a few cases of large cavities. The air-chamber acts as a consonance-box and resonator, and gives a metallic quality to the sound. Other physical signs of cavity and fluid are associated. It may be heard when the patient is breathing quietly, or only after coughing. Sometimes only tinkling is heard, or the sound of a number of drops is transmitted. The latter occurs after coughing.

Bell-tympany. The *bell-sound* is heard when air is confined in the pleura. If the stethoscope is placed over the pleural cavity, and two coins are used as plessor and plexímeter, a distinct metallic or anvil-sound is transmitted to the ear. The cavity containing air can be clearly outlined if the metal plexímeter is moved about. As soon as it passes over a part of the chest under which no air is confined the sound is not heard. Although heard in nearly all cases of pneumothorax, there are some cases in which it cannot be elicited, probably because of the small size of the aperture in the pleura.

Succussion. The ear is placed to the side of the chest, and the patient's body moved suddenly by himself or by the observer. A splashing sound is heard. It can only be produced when there is air as well as fluid present in a cavity. It was first described by Hippocrates, and the term "Hippocratic succussion" has been given to it. It is characteristic of hydropneumothorax, although not present in all cases of this disease. The sound may be audible at a distance. Metallic tinkling can usually be heard at the same time.

Auscultation of the Voice. When the ear or stethoscope is applied to the surface of the chest and the patient is asked to speak, the vibrations of the air in the trachea and bronchial tubes are transmitted to the chest-wall and become audible. The sound is known as the *vocal resonance.* It is a sign which goes hand-in-hand with *vocal* or *tactile fremitus,* and is modified by the same conditions which modify the latter. While, in general, conditions which increase the fremitus increase the vocal resonance also, this is not invariably the case.

Sometimes one is increased and not the other, without there being any evident reason for it.

Vocal Resonance in Health. It varies in health conjointly with the fremitus. The sound is purring or buzzing. It is heard more pronouncedly at the right apex than at the left; in persons with thin chest-walls; in individuals in whom the voice is low in pitch and strong. It is lessened, therefore, in females and children. It diminishes the further away the ear gets from the larynx, and hence is feebler at the bases. It is immaterial what words are selected by the patient to create the resonance. It is important for the student, however, to become familiar with the resonance of a definite series of words which when pronounced do not need any marked change in inflection of the voice. The words "one," "two," "three," or "ninety-nine," spoken repeatedly, are selected. The patient should not raise or lower his voice during the act of speaking. Symmetrical portions of the two sides of the chest must be examined successively.

Vocal Resonance Increased. Increased vocal resonance depends upon the intensity or extent of the cause. When slightly above normal it is referred to as slight increase, or when the voice is transmitted comparatively distinctly to the ear it is known as *bronchophony.* This may be heard in health over the trachea, or over the bronchi behind. When heard over the vesicular structures of the lung, it indicates that the vibrations are transmitted to the ear by some better conducting material. This is usually a consolidated lung, and hence: 1. In all cases of consolidation the resonance is increased, that is, bronchophony is created; but in pneumonia, if the bronchus is occluded by exudate, it is absent. 2. If the lung is collapsed but the bronchi open, the resonance is increased. 3. It is also increased in cavities. Sometimes the resonance is intensified and the sound is even more pronounced than when heard over the trachea.

Pectoriloquy. The voice may be so distinctly transmitted that we have the impression that the patient is speaking into the mouth of the stethoscope. If the patient speaks slowly the words may be distinctly heard. It is more striking when the patient whispers. The term "whispering pectoriloquy" is then applied to it. It is detected over a cavity if it communicates with a large bronchus, and sometimes in consolidation of the lung.

Vocal Resonance Diminished. Vocal resonance is diminished or absent when anything cuts off the supply of air, and intercepts the vibrations from the part over which the observer is auscultating. Fremitus and resonance are absent over the area supplied by a bronchus which is occluded by external pressure, as an aneurism. Diminution or absence of vocal resonance is more marked in cases of pleural effusion (serum, blood, pus, or air) or thickened pleura. The vibrations are impeded because of the difference of conducting material. The degree of diminution depends upon the amount of effusion.

Modifications of Vocal Resonance. 1. At the uppermost limit of the pleural effusions, at which point the layer of fluid is thin, the resonance is transmitted in a modified form. It is tremulous and bleating in character, and is known as *ægophony* because it resembles

the bleat of a goat. It is especially heard at the angle of the scapula, or below it in cases of moderate effusion. It is due to the fact that the fundamental tones are intercepted by the fluid, while the other tones are allowed to pass through and give the peculiar bleating sound (Gee). 2. The vocal resonance may have a metallic character in pneumothorax when there is free communication with the bronchus.

Cavities. Pulmonary cavities are due to destruction of lung by abscess, gangrene, or tuberculosis, or to dilatation of the bronchi.

As there is usually a local increase in the amount of air in cavities, there is in consequence a local area of exaggerated resonance, or tympany, and with it the occurrence of cavernous breathing, or breathing of an amphoric type. The presence of a cavity, however, is often difficult to recognize, because of the relation to the surrounding structure or because of fluid contents. If the lung about it is the seat of consolidation, the physical signs of this consolidation may over-ride the signs of a cavity. If compensatory emphysema surrounds the cavity, it may be almost impossible to recognize it. Moreover, the contents of the cavity render the recognition of its presence difficult. If it contains a large amount of fluid, the signs of consolidation alone may be present. Much attention has been paid to the recognition of cavities, and some methods have been proposed by which it is thought they can be distinguished. While it is a satisfaction to determine exactly the presence and location of a cavity, it is not an essential to diagnosis. To confirm the presence of an excavation, even if the physical signs point to its occurrence, the diagnosis should be controlled by examination of the sputum. If, on such examination, yellow elastic tissue is found, the presence of a cavity is more probable. The methods employed to determine their presence absolutely have been named after the observers who devised them.

First, *Wintrich's change of sound.* If the cavity communicates with a large column of air in the bronchus, and percussion is employed with a moderate degree of force, the note will change as the patient alternately opens and closes the mouth. If the mouth is open wide, the sound is louder and more distinctly tympanitic and higher in pitch. If the mouth is closed, the sound is correspondingly lessened and not so tympanitic. Indeed, sometimes a sound is obtained with scarcely a trace of tympany. This change of sound is in all probability due to change in the resonant cavities in the upper respiratory tract. It must not be confounded with "Williams' tracheal tone," which can be elicited near the junction of the clavicle and sternum on the left side, in cases of consolidation of the underlying portion of the lung, particularly if the force of the blow is directed toward the trachea. Strong percussion is necessary to bring out Williams' tone.

Second, *interrupted change of sound,* also described by Wintrich, is distinguished from the simple change, in that it occurs in different positions of the body. It may be heard when the patient is in an upright position, and disappear when he assumes the recumbent posture; or the converse may be true. The change in position changes the relation of the bronchus to the cavity, on account of which the varying tympanitic sound is produced.

Third, *Gerhardt's change of sound.* This change depends upon the alteration of the level of the fluid when the patient assumes the upright, or the dorsal position. It is not necessary that the cavity communicate with the large bronchus. It is a positive symptom of a cavity, but rarely present. The sound changes in pitch and in the degree of tympany. It may be absolutely dull over the lower part of the cavity when the upright position is assumed, because the fluid gravitates to this portion and comes in contact with the chest-wall.

Fourth, *Friedreich's respiratory change of sound. Respiratory percussion.* (Da Costa.) The pitch of the sound becomes higher at the end of a deep inspiration. It depends upon increased tension of the chest-wall and lung-tissue, and the wall of the cavity, during the act of inspiration. It may be a source of confusion, which is obviated by percussing at the same stage of the breathing each time, or percussing only on superficial breathing.

Fifth, Seitz has called attention to a form of breathing named *metamorphosing.* Inspiration begins harshly bronchial, then becomes faintly bronchial, the latter sound being heard also in expiration. It is said to be a sure sign of cavity.

Résumé. The student must bear in mind in auscultation to note: (1) If the sounds are increased or diminished in intensity; (2) the rhythm of the inspiratory and expiratory sounds; (3) if the respiratory murmur is replaced by bronchial breathing or its modification; (4) the presence of new sounds (râles and friction); (5) the voice-sounds.

Mensuration. By mensuration or thoracometry, the results secured by palpation are confirmed more accurately. The size and the degree of expansion of the chest are ascertained. Hence the circumference and diameter of the chest are determined and the differences in the shape and movement of two sides made manifest. If the measurement is taken from day to day, it can be graphically recorded by tracing sections on paper, and delicate changes can thus be definitely ascertained. The circumference of the chest is measured by means of the ordinary tape-measure or by metal tapes joined together by a hinge. The latter can be made to fit the circumference of the chest accurately, and are essential in order to transfer the section to paper. The middle of the hinge is held firmly over the spinous process of the vertebra, while the two limbs are carried around the chest, moulded to all inequalities, and crossed in front, one above the other; a mark is made on each where it crosses the middle line. Measurements should be taken at about the level of the nipples, and two inches below them, and care should be taken to have the level the same in front and behind. They should be taken in full inspiration and expiration, and in repose. The outline secured by this method need not be disturbed, as by flexion on the hinges we are enabled to remove it intact. The tapes are carefully transferred to a sheet of paper, on which imaginary diameters have been marked. After fixing the corresponding points of the tapes on the lines of the respective diameters, the outline can then be traced.

Woillez's cyrtometer is a chain with links which is used to ascertain the exact circumference. The *diameter* of the thorax is secured by

means of caliper compasses. The antero-posterior diameter should be taken on a level with the nipple and the insertion of the second rib behind; the transverse diameter at the highest points of the axillæ. The length of the chest may be ascertained by measuring in the mid-clavicular line from the clavicle to the border of the ribs. It is important to remember that the right side of the chest measures a little more than the left in people who are right-handed.

The respiratory capacity is estimated by measurement of the circumference of the chest. This is secured by taking the measurement at the end of complete expiration and then at the end of complete inspiration. In health the difference between the two should be from five to ten centimetres (two to four inches). If the expansion is less than two inches, it is considered deficient by insurance companies, and the risk is not regarded as first-class. The expansion is less in women. In taking the measurement the observer must be particular to keep the terminal portion of a tape-measure fixed in the median line of the structure. The other portion is to be held in the hand, so as to move with inspiration and expiration. Always mark in advance the anterior mesial line and note the exact level at which measurements are made when they are taken daily. Deficiency of chest-expansion not only indicates the presence of a local morbid process—notably incipient tuberculosis, but it also indicates lack of strength and of muscular development, of physiological deficiencies, rather than physical, and is an unerring guide to the need of respiratory gymnastics.

Spirometry. By means of the spirometer Dr. John Hutchinson has been able to estimate the quantity of air taken in with each inspiration and discharged with expiration. By it the respiratory or vital capacity is estimated. The data ascertained are not of much diagnostic significance, although if measurements are made from day to day we may be able to estimate the extent of recovery from disease of the lung which was incapacitated. When, however, there is an important diminution of lung-capacity, tuberculosis may be suspected, before subjective and objective signs warrant a diagnosis. We can also estimate the degree of interference with breathing by disease below the diaphragm. Spirometry is of particular value because it shows in a graphic manner the need for respiratory gymnastics. By means of Waldenburg's pneumotometer the respiratory pressure of air on inspiration and expiration is determined. Expiratory pressure is diminished in emphysema, and the degree of diminution may furnish a clue to the severity of the disease or the degree of improvement. It is to be remembered that the expiratory pressure always exceeds the inspiratory pressure in health by as much as 20 to 30 millimetres, according to Waldenburg. It is natural to find that inspiratory pressure is lessened in stenosis of the air-passages, in phthisis and in pleural effusions, although it is not of diagnostic significance.

The following measurements, secured by laborious investigation, are excellent criteria from which pathological inductions can be made.

Measurements of the Chest and Lung Capacity.

(Otis, *Boston Medical and Surgical Journal*, 1895.)

Table I.—Chest Measurements.

	Repose, inches.	*Inflated, inches.*	*Difference, inches.*
Girth, muscular.—Men:			
Average of Dr. E O. Otis, 1000 measurements, between sixteen and forty years of age . .	34.0	36.1	2.1
Average of Dr. Hitchcock, of Amherst College, 8000 measurements	34.6	36.5	1.9
Average of E. Hitchcock, J., of Cornell College, 15,000 measurements	34.5	36.3	1.8
Girth, muscular.—Women:			
Mt. Holyoke and Wellesley students. Measurements of Miss Wood and Dr. Mary Colton .	29.5	31.5	3.0
Chest, respiratory.—Men:			
Average of Dr. E. O. Otis, 1000 measurements .	31.1	33.1	2.0
Chest, respiratory.—Women:			
50 per ct. of 1500 of Wellesley students, Miss Wood	24.6	27.2	2.6
Depth of chest.—Men:			
Average of Dr. E. O. Otis, 1250 measurements in repose and 362 inflated	7.5	8.3	0.8
Depth of chest.—Women:			
50 per ct. of 1500 students at Wellesley, Miss Wood	6.9	....	....
Breadth of chest.—Men			
Average of Dr E. O. Otis, 400 measurements .	9.9	10.8	0.9

Table II.—Capacity of Lungs.

	Cubic inches.
Men:	
Average of Dr. E. O. Otis, 1000 measurements	240.6
Hitchcock, 8000 measurements	230.0
Hitchcock, Jr., 15,000 measurements	236.6
Women:	
Mt. Holyoke and Wellesley students, measurements of Miss Wood and Dr. Mary Colton	145.8
50 per cent. of 1500 Wellesley students, Miss Wood	150.3

Table III.—Comparison of the "vital" or lung capacity and the amount of air expelled after an ordinary quiet respiration.

Average of Dr. E. O. Otis, 150 measurements.

	Cubic inches.
Vital capacity, or the amount of air exhaled after a full inspiration .	230.5
Amount of air exhaled after an ordinary quiet respiration . .	129.3
Difference, or "complemental" or "reserve" air	101.2
Difference as given by Hermann	97.6

Average Lung Capacity for Height (Otis).

Height.	*Lung capacity.*	*Average for each inch or centimetre in height.*
66 to 67 inches inclusive.	231.62 cubic inches.	3.4 + cubic inches.
167.7 to 170.3 centimetres.	3797 cubic centimetres.	22.4 cubic centimetres.
67 to 68 inches inclusive.	237.10 cubic inches.	3.46 cubic inches.
170.3 to 172.8 centimetres.	3903 cubic centimetres.	22.7 cubic centimetres.
68 to 69 inches inclusive.	244.44 cubic inches.	3.5 cubic inches.
172.8 to 175.4 centimetres.	4007 cubic centimetres.	23.06 cubic centimetres.
69 to 70 inches inclusive.	259.34 cubic inches.	3.66 cubic inches.
175.4 to 177.9 centimetres.	4250 cubic centimetres.	24.06 cubic centimetres.
70 to 71 inches inclusive.	261.38 cubic inches.	3.64 cubic inches.
177.9 to 180.5 centimetres.	4284 cubic centimetres.	23.9 cubic centimetres.
71 to 72 inches inclusive.	261.34 cubic inches.	3.5 cubic inches.
180.5 to 183.0 centimetres.	4284 cubic centimetres.	23.03 cubic centimetres.
General average . . .	3.52 cubic inches, for each inch of height.	
	23.19 cubic centimetres, for each centimetre of height.	

Powel lays great stress upon the fact that in phthisis the inspiratory capacity is diminished, but the expiratory power remains normal.

Combination of Physical Signs. In order to determine the physical condition of the lung, it is necessary to draw conclusions from the results obtained by all the methods of physical examination. It is the exception that any one sign is pathognomonic of a physical condition. If the student will glance over the abnormal physical conditions which may take place in the lung, he will find that they may be divided, first, into physical changes in the lung proper, and, second, into physical changes in the pleura. With regard to the lung, it will be further noted that the changes are due to an increased amount of air or to a diminution in the amount of air.

Increase in the amount of air may be general, unilateral, or local, and is indicated by a combination of physical signs which are usually unerring. On inspection (*a*) enlargement, general, unilateral, or local; (*b*) increased action in general emphysema, although with diminished respiratory excursion; when unilateral or local, increased action and increased expansion (compensatory emphysema). On palpation, inspection confirmed, and vocal fremitus diminished when the increased amount of air is general, slightly increased when it is unilateral or local. On percussion in each instance exaggerated resonance or tympany. On auscultation, when general (emphysema), feeble respiratory murmur, with prolonged expiration; when unilateral or local, exaggerated respiratory murmur. The difference in the physical signs of increased amount of air is not due to the difference in quantity, but to the associate physical condition and the force of the movement of the air. The diminished expansion and feeble respiratory murmur in emphysema are due to inability to exhale the air because of the diminished elasticity of the lung, while the bronchioles occluded from bronchitis lessen the fremitus. In cavities—local increase of air—the physical condition of the tissue which surrounds them modifies the physical signs.

Decrease in the Amount of Air. The diminution in the amount of air from change in the physical condition of the lung is due to consolidation or to collapse. The latter occurs when the bronchus is obstructed, the former in congestion, pneumonia, gangrene, abscess, forms of tuberculosis, and hemorrhagic infarct. The physical signs are the same under all circumstances, except in collapse: expansion lessened, fremitus increased, dulness, bronchial breathing. The signs vary with the degree of consolidation as follows: Slight increase to greatly increased fremitus, impaired resonance to complete dulness, broncho-vesicular to bronchial breathing. In tuberculosis there may be flattening of the chest-wall, but otherwise the signs are the same. The presence of new sounds depends upon the amount of secretion or fluid, as is the case when there is increase of air in the part.

Broadly speaking, therefore, in affections of the lung proper, the two conditions just mentioned must be differentiated—air increased, air diminished. We do not refer to bronchitis, because no physical change takes place in the lung, and the signs depend upon the amount of fluid in the tubes.

The Pleura. If satisfied that the physical condition is not due to change in the lung structure, the state of the pleura must be investigated. Here, too, the physical condition may be due to an excessive accumulation of air or to an accumulation of fluid or solid material. In effusion there is enlargement of the affected side, diminished movement, diminution of fremitus and of vocal resonance. When air is present, however, there is tympany; when fluid, there is dulness on percussion.

The problem may, however, be looked at from another side. 1. The percussion-note is tympanitic and indicates that there is an increased amount of air. Is this in the pleura or in the lung? If in the pleura, it can only be unilateral, and is recognized by diminution of the movement and of fremitus, as against increased movement and fremitus when due to unilateral increase of air in the lung proper (compensatory emphysema). 2. The percussion-note is dull and indicates the absence of air. Is this in the pleura or in the lung? A distinction between consolidation and pleural effusion must be made. In consolidation there are increased fremitus, increased vocal resonance, bronchial breathing, and dulness on percussion. There may or may not be contraction. In pleurisy with effusion, diminished or absent movement, absent fremitus and resonance, dulness on percussion, feeble, distant, or absent breath-sounds. The distinction of the two physical conditions seems easy, and yet the physical signs may not be sufficiently definite to warrant a positive conclusion. There are cases in practice in which it is almost impossible to determine which is present. It has been stated previously that bronchial breathing may be present in pleural effusions. To add to the difficulty in certain cases of consolidation it may, however, be absent, and so may the vocal fremitus and resonance. Apart from the associate general and local symptoms, we must look to two methods of corroborative proof of the presence of fluid. First, exploratory puncture; and, second, displacement of organs. The former has been spoken of. The latter includes displacement of the heart to the right or to the left, depending upon the seat of the effusion; dislocation of the liver; and, in cases of left pleural effusion, obliteration of the half-moon space (Traube's line).

Sputum.

This term is applied to all the products of secretion of the mucous membrane of the respiratory tract, and other substances that may be brought up through the respiratory tract. The characters of sputa in disease vary with the part affected, as well as with the pathological nature of the disease. It is always well to examine each specimen both *macroscopically* and *microscopically*.

Method of Collection. Sputum that is to be examined should be collected in perfectly clean vessels, containing no fluid, preferably in glass or white earthenware spittoons, and care should be exercised against the entrance of any extraneous substances, as tobacco or particles of food from the mouth, or from outside sources, or from the stomach through vomiting. Tobacco, prunes, and bread crusts are at

times mistaken for blood. It is also necessary to see that the matter sent for examination is derived from the lungs, and is not simply the oral and faucial accumulation. If practicable, the mouth and pharynx should be first rinsed with a warm alkaline solution. The true sputum is coughed up.

We usually require in the examination one or two glass dishes or plates, a large and a small piece of window-glass, mounted needles, and forceps; for microscopic work, in addition to these, a good microscope and accessories, and certain staining fluids. Sputa which upon examination has been found to contain tubercle bacilli should not be allowed to dry in the air, but should be thoroughly mixed with a 1 : 20 carbolic acid solution, or a 6 per cent. formalin solution should be added to the sputa after the examination is completed.

In describing sputum we note the quantity in twenty-four hours; its color, odor, specific gravity, its composition and consistency, whether mucous, purulent, mucopurulent, frothy, watery, bloody, tenacious or viscid, and whether it is made up of separate layers or is homogeneous.

The *quantity* in twenty-four hours varies from a few c.c. to even 1000 c.c., as in a discharging empyema.

The *color* changes with the composition and the nature of the disease. Thus, in *acute bronchitis* and œdema of the lung it is white; in purulent sputa, no matter what the cause, it is yellow or greenish-yellow; in pneumonia, "rusty;" in abscess of the liver with amœbæ characteristics, brownish-red or like "anchovy sauce."

The *odor* is characteristic in a few cases only. That of bronchiectasis, gangrene, and putrid bronchitis is particularly *heavy* and *fetid*—a characteristic which renders its origin almost unmistakable.

The *reaction* is always alkaline.

The *specific gravity* may vary from 1.0043 (mucus sputum) to 1.0375 (serous). (Von Jaksch).

Varieties of Sputum. Mucus sputum, on account of the mucin, is usually glairy, clear, and tough. It is seen in acute bronchitis in the early stage, and in œdema of the lung. In health a small amount of mucus is expectorated, which in cities and smoky towns is apt to contain black pigment-particles, due to inhaled soot.

Purulent sputum is composed almost entirely of pus. Typical purulent sputum is that from an empyema discharging through a bronchus. It may also occur in bronchiectasis, chronic bronchitis, abscess of the lung, of the liver, or more rarely of the mediastinum, discharging through a bronchus; or it may be the discharge of a tubercular vomica. The special condition can usually be determined by microscopical examination and the accompanying symptoms and signs.

Mucopurulent Sputum. It is most common to have mucus and pus mixed together in varying proportions, and then it is termed mucopurulent. Such sputa may be found in the same conditions as purulent sputa. When flat, coin-shaped masses are formed, sinking to the bottom if the vessel contains water, as in phthisis and chronic bronchitis, it is known as "nummular" sputum; or it may be more spherical, and is then called "globular." At times the sputa may be seen to separate into three distinct layers, the upper frothy, mucopuru-

lent, greenish-yellow, or dirty-green, sometimes lumpy, sometimes composed of shreds; the middle thin and watery, with shreds from the upper layer; and the bottom layer, apparently made up of pus and débris, opaque, and without air-bubbles. It points to *gangrene* of the lung in most instances, but may also occur in bronchiectasis.

Watery or serous sputum is the result of œdema of the lung. Such sputum, also called albuminous expectoration, is discharged after paracentesis of the chest. Beginning during or as late as two hours after the operation, from one to three pints may be discharged in a few hours.

Bloody Sputum—Hæmoptysis. As blood in sputum is always of importance, the entrance of substances as mentioned above, which simulate it in appearance, should be guarded against. It may be seen in greatly varying quantities and have many different sources, and it may be of slight or grave significance. It may come from the gums, nose, pharynx, or larynx, and in all cases such sources should be examined. Again, there may be cases in which bleeding from the stomach (hæmatemesis) or œsophagus simulates hemorrhage from the lungs, but still more often people speak of vomiting blood that really has come from the lungs. Usually that from the lungs is much more frothy and bright-red, while that from the stomach is darker and acid, and may contain particles of food. Diagnosis is more difficult when some blood from the lungs is first swallowed and then vomited.

Usually there is a distinct history of preceding cough, and for some time afterward small amounts of blood continue to be expectorated. (See Lungs; Hemorrhage.)

Small amounts of blood streaking the mucus sputum or appearing in small clots often come from the throat or nose or upper air-passages, but may come from the lungs. Mucopurulent sputum streaked with blood is frequently indicative of phthisis. In pneumonia the rusty sputa are the result of an admixture of mucus and blood, and usually contain small air-bubbles. When the blood-coloring matter is changed there may be a yellowish or greenish tinge. In certain cases of chronic pneumonia, in which the blood remains longer in the lung-tissue, the expectoration has a darker color. The same color may be observed when there is a slight leakage from an aneurism. Pneumonia accompanied by expectoration of large amounts of blood is often of tuberculous origin. Blood may be mixed with the greenish expectoration of gangrene. According to Finlayson, this is especially true in children. In chronic valvular disease of the heart, and in oozing from aneurism, frothy mucus containing more or less blood is commonly seen. "Currant-jelly" sputa are more or less characteristic of malignant growths of the lungs, while the expectoration from a liver abscess with amœbæ is reddish-brown in color, from the mixture of blood, pus, and bile-elements, and is not unlike "anchovy sauce." We may have hemorrhage from the lungs as part of a general hemorrhagic tendency, as in purpura and hemorrhagic smallpox; in so-called "vicarious menstruation" there may be hæmoptysis. But a patient presenting such symptoms should be examined with the greatest care, to exclude actual pulmonary complication. When great quantities of

blood are expectorated we suspect tuberculosis of the lung, aneurism, or cardiac valvular disease.

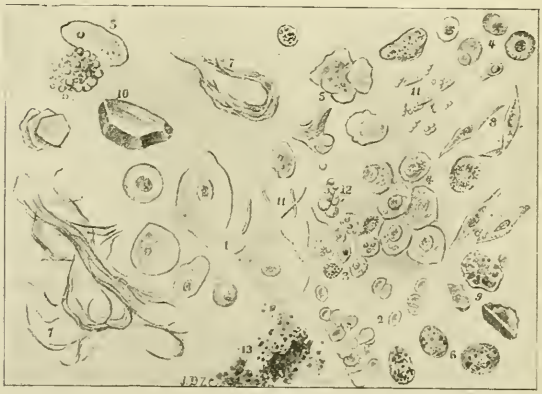
The unaided eye may distinguish other foreign substances, such as fibrinous and spiral casts of the bronchi or trachea ; but full consideration of them will be given further on.

Microscopical Examination of the Sputum. (See Fig. 143.) White blood-corpuscles, usually of the polymorphonuclear variety, are present in all sputa, but in varying numbers and size. They are most abundant in purulent sputa. Often they contain fat-drops and pigment-particles. In stained preparations of sputa in cases of acute croupous pneumonia, influenza, pneumonia, or phthisis, frequently many of the leucocytes contain large numbers of organisms—*i. e.*, pneumococci, influenza bacilli, or tubercle bacilli.

Red blood-corpuscles are to be found in most sputa. They may be so few as not to give a red color. The source is often high up in the respiratory tract. When they are present in large numbers the sputum is more or less tinged, and in hæmoptysis it is almost wholly made up of red cells. Usually each cell is well preserved, but they may appear as pale bodies or as rings, the pigment remaining in the sputum as pigment-particles or as crystals of hæmatoidin, as in pneumonia.

Eosinophile cells are frequently found in large numbers in the sputum in cases of asthma. They are also present in the sputum in acute and chronic bronchitis and in phthisis. Their presence in the sputum in cases of phthisis is considered by Teichmuller to be of favorable import.

Fig. 143.



Various objects from sputum. 1, squamous epithelium ; 2, red blood-corpuscles ; 3, polynuclear leucocytes , 4, alveolar cells ; 5, myelin-cells ; 6, pigment-cells ; 7, elastic-tissue fibres ; 8, squamous cells ; 9, hæmatoidin-crystals ; 10, phosphate crystals : 11, fungi ; 12, fat-globules ; 13, free pigment. (Original observation.)

Epithelium. Two general varieties are found in the sputum—squamous and cylindrical. The former comes from the mucous membrane of the mouth, the tongue, tonsils, true vocal cords, and perhaps from the salivary and small bronchial glands. It has no clinical importance. (See Fig. 143.)

Cylindrical cells in sputum are rarely perfect. It is uncommon to find the cilia intact, and still more so in motion, while the body of the cells is likely to be changed. They are found in inflammations of the trachea and bronchi, or the posterior nasal fossa—a locality where, it must be remembered, ciliated epithelium exists.

"ALVEOLAR" EPITHELIUM, so called, when found in the sputum, is more important than the above, as different observers consider its presence to have more or less clinical significance. The cells are elliptical or round, somewhat larger than white corpuscles, with a single nucleus, which is indistinct without the addition of acetic acid. The protoplasm is granular and contains particles of iron-dust, carbon, or blood-coloring matter, and often fat-drops. The cells may also have undergone complete fatty degeneration, and they have been considered the source of myelin-drops in the sputum.

Bizzozero has shown that alveolar epithelium not only occurs in almost all pulmonary affections, but also at times in normal sputum.

Detection. A small bit of sputum is placed on a microscope-slide and a cover-slip applied. Examine with varying powers, and again, after acetic acid is added, stain the cells with an aqueous solution of methylene-blue.

Frequently in cases of heart disease with failing compensation, especially where the mitral valve is affected, the alveolar cells may contain large amounts of blood pigment.

GIANT CELLS have been found in the sputum of phthisis cases.

ELASTIC FIBRES. As the presence of elastic fibres in sputa is of much import, denoting destruction of the lung-tissue, bronchi, or the larynx or bloodvessels, their presence from food remaining in the mouth must be especially guarded against. They may be mistaken

FIG. 144.

Elastic fibres of lung-tissue obtained from sputa after digestion in caustic soda.
(Drawn by DR. JOHN WILSON.)

for fat-crystals. They are found as single threads in bundles, or showing an alveolar arrangement. They are to be recognized by the double contour and curling ends, and at times by their alveolar arrangement. They may be due to tuberculosis, abscess of the lung, bronchiectasis, gangrene of the lung, pneumonia (von Jaksch), and rarely to destructive diseases of the larynx. In a very great majority of cases they are

due to tuberculosis. It is uncommon to find them in gangrene, probably because, as Traube first suggested, they are destroyed by a ferment. (See Fig. 143.)

Elastic tissue from the alveoli often shows the diagnostic alveolar arrangement; the fibres that form a bronchus are branched; those from eroded artery appear in the form of a network, or the fibres are bound together. (See Fig. 144.)

Detection. The method employed by Osler, modified from Sir Andrew Clark's, is the best. A small amount of the thick, purulent portions of sputum is pressed out in a thin layer between two pieces of plain window-glass, 15 x 15 cm. and 10 x 10 cm. The particles of elastic tissue appear on a black background as grayish-yellow spots, and can be examined *in situ* under a low power. Or the upper piece of glass is slid off till the piece of tissue is uncovered, when it is picked out and examined on a microscopic slide, first with a low power, as the one or one-half inch objective, and then with a higher power. At first there will be some difficulty in distinguishing with the naked eye between elastic fibres and particles of bread or milk globules, or collections of epithelium and débris, but with practice such mistakes can be avoided, and the microscope always reveals the difference. This method is much easier of accomplishment and quite as satisfactory in results as the one generally employed—boiling an equal quantity of sputum and solution of caustic potash (8 to 10 per cent.) for a short time, and then allowing it to stand for twenty-four hours in a conical glass. The elastic tissue remains intact and is found in the sediment.

Connective tissue and cartilage, in fragmentary bits, are rare constituents of sputum. The former may occur with abscess or gangrene of the lung, and the latter when there is ulceration of the larynx.

Fibrinous Coagula. These striking, tree-like bodies are found in the sputa of plastic bronchitis, and at times in that of pneumonia, phthisis, and in diphtheria and croup when there has been an extension into the bronchi. They are usually mixed with mucus, and are rolled up into a mass. Their peculiar form is best seen when they are washed and unravelled in water. They are then seen to be a complete mould of a small bronchus with its ramifications. The size varies greatly. They may be many centimetres long. In fibrinous bronchitis the size and shape of the moulds in different attacks may be exactly similar, as if they came from the same bronchus. They are grayish-white in color, hollow, and on transverse section are seen to be made up of cast upon cast. Leucocytes, blood-cells, and alveolar epithelium are found in the meshes by the microscope, and at times Charcot-Leyden crystals and Curschmann's spirals also. They are almost pathognomonic of fibrinous bronchitis. When they occur in any number in pneumonia they make the prognosis unfavorable. Blood-casts of the smaller bronchi have been found in cases of hæmoptysis. They are rare, and have no apparent connection with the fibrous coagula.

Spirals. Under this name are included spiral bodies that are found in the sputa of bronchial asthma, and occasionally in that of

pneumonia and capillary bronchitis (von Jaksch), and chronic pulmonary tuberculosis (Vierordt). At the beginning of an asthmatic attack tough rounded balls are expectorated—"perles" of Lænnec—which, if freed from the mucus surrounding them and spread out on a glass with a dark background, may be seen by the naked eye to have a twisted spiral form. With the aid of the microscope they are found to be made up of spirally arranged mucin in a more or less tight twist, with many cells from the alveoli and bronchi. In some of these spirals a shining central thread runs through the entire length like a core, remarkable for its clearness and its high refractive index. The fine fibres composing the spiral may be closely arranged or not. Epithelium and Charcot-Leyden crystals may be found lying among the coils. The main constituent of the spirals is mucin, and Osler has suggested that the central thread is made up of transformed mucin. On the other hand, von Jaksch believes it to be chemically distinct from the mucin spiral and to approach rather to the character of fibrin. Vierordt considers it either made of tightly twisted central fibres or to be an optical image of a core-cavity. They are probably the result of an acute bronchiolitis. Why they should assume this remarkable form is still an open question. It has been suggested (Osler) that the ciliated epithelium of the bronchi may have a rotary action, and their action, combined with the spasm of the bronchioles, causes the spiral formation.

Sections for Microscopical Examination. Schmidt (*Zeitschrift f. klin. Med.*, 1892, p. 476) fixes sputum in ½ per cent. salt solution saturated with mercuric chloride, hardens in alcohol, and sections in the usual manner. For hardening sputum Zenker's fluid has been found most satisfactory. After hardening the sputum is embedded in paraffin and cut. In many cases it is advisable to roll up the sputum in a little ball before fixation. For the study of spirals thick pieces should be embedded in celloidin; for the study of the cellular elements, thin sections are embedded in paraffin.

Sections of sputum with mucin swell when treated with watery solutions of the dyes; hence the celloidin should be first removed to prevent folding of the sections. All specimens of sputum, except the very thin ones, can be prepared in the manner described.

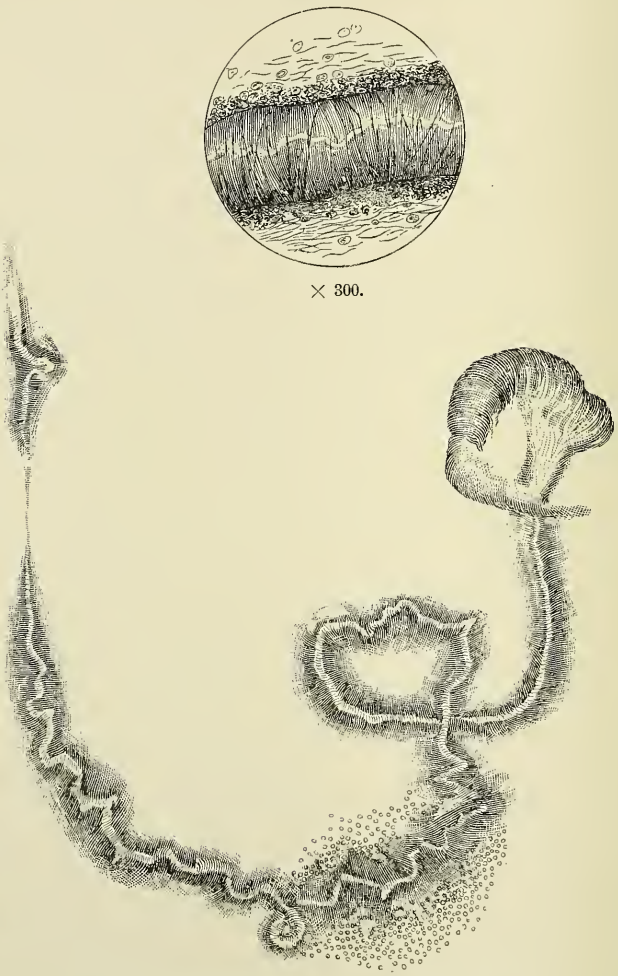
The spirals are best stained with Weigert's fibrin-method; they stain blue. Yet they—*i. e.*, the central threads—are not fibrin: (1) Because they are perfectly homogeneous; (2) they assume a violet color after prolonged staining—fibrin is always blue; (3) unformed blue masses are found which could only be compact mucin masses; (4) their specific mucin reaction with thionin; (5) the greenish color assumed when Ehrlich's triacid stain, as modified by Babes, is used. (See Fig. 145.)

That there is a connection between the spirals and Charcot-Leyden crystals seems very probable, as the latter are absent from the sputum at the beginning of an attack of bronchial asthma; but if a portion of such sputum is allowed to stand for twenty-four to forty-eight hours, taking care that evaporation does not take place, crystals will be found. As has been said, the crystals are often found among the spirals, and

this when they are seen nowhere else. Later on the spirals disappear, but crystals derived from them (?) continue to be expectorated. (See Fig. 145.)

The method of examining for spirals is as given above.

Fig. 145.



× 300.

Spirals from bronchial tubes. × 80. (After Leyden.)

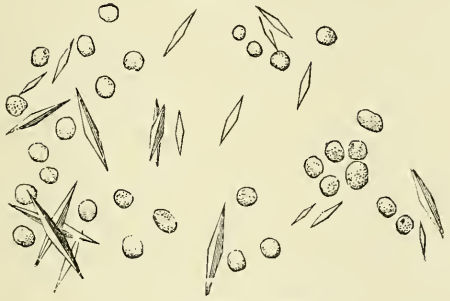
Crystals. Charcot-Leyden, cholesterin, hæmatoidin, fatty, tyrosin, oxalate of lime, and triple phosphate crystals are to be found in sputa under various conditions.

Charcot-Leyden crystals are octahedral, sharply pointed, colorless or slightly bluish, soluble in warm water, alkalies, and acetic and mineral acids. The practised, unaided eye may recognize these as small yellowish bodies, not unlike grains of sand; under the micro-

scope they are unmistakable. Their size varies greatly. They occur most abundantly during (invariably) and after an attack of bronchial asthma; they have also been seen in sputa of acute and chronic bronchitis and tuberculosis. They are identical with crystals found in semen, feces, and leukæmic blood and bone-marrow. Their connection with spirals has been mentioned above. Schreiner considers them to be the phosphate of an unknown base, which Ladenburg and Abel think may be identical with æthyleninim or di-æthyleninim. This identity, however, is disputed by Th. Kohn.

Detection. Examine the sputum of an asthmatic patient a day or two after the beginning of an attack for round, hard, yellowish bodies, and place these under the microscope with different powers. They are readily recognized. (See Fig. 146.)

FIG. 146.



Charcot crystals. (SCHEUBE.)

CHOLESTERIN CRYSTALS. These crystals are similar to those of cholesterin found elsewhere, being thin rhombic plates, often with irregular corners and high refractive index. They are soluble in ether; and, when treated with dilute sulphuric acid and tincture of iodine, become violet, blue, or green, and then red. They may be present in the sputum of tuberculosis, abscess, and hydatid abscess of the lung, and in pus from an abscess of another organ, as the liver. They have but little clinical significance.

HÆMATOIDIN CRYSTALS. Hæmatoidin crystals are at times recognizable by the naked eye as distinct spots of yellowish or brownish-red color. Under the microscope they have a brownish-yellow or ruby-red color, and are either in the form of small rhomboid prisms or of fine needles, single or arranged in bunches of various shapes, or as free pigment-particles without crystalline form; smaller particles may be contained within a leucocyte. Their presence indicates that blood has remained in the respiratory tract for some time before being expectorated, or that an abscess has discharged into a bronchus. They occur in phthisis, following hemorrhage; in thoracic aneurism when blood is oozing into the lung; in gangrene; in abscesses discharging through a bronchus. Von Jaksch states that when the crystals are contained in cells there has been a preceding hemorrhage, but that when there is considerable free hæmatoidin one infers that an abscess of a neighboring organ has discharged into the lung.

Fatty Crystals. Crystals of margaric acid occur as long, thin needles, greatly curved or bent at one end like a fish-hook, and either singly or in bundles. They are found in unhealthy pus—as in gangrene, putrid bronchitis, bronchiectasis, and tuberculosis; in the plugs formed in inflamed tonsils; and in purulent sputum in general which is allowed to stand in a warm place. They dissolve in ether and boiling alcohol; this characteristic, together with the regularity of their curve, should distinguish them from elastic fibres, with which they are sometimes confused by beginners.

Tyrosin crystals have been found in the sputum of putrid bronchitis and empyema discharging into the lung, and usually in conjunction with leucin. They are most abundant in sputum that has been allowed to stand for some time. Under the microscope they appear as fine needles, and can be mistaken for fatty crystals. They are without diagnostic importance.

Oxalate of lime and triple phosphates have been noted occasionally in sputa; the former in a case of diabetes, and also in an asthmatic; the latter occur only in alkaline sputa, as they are soluble in acids.

Uric acid crystals have been observed by Moore in the sputum of a gouty patient.

Concretions are rarely present in the sputum. They arise usually from the bronchial glands or lungs, from foci of tuberculosis which have become healed with the deposition of lime-salts. They may be single or multiple. Hievoilés reports finding tubercle bacilli in the centre of one of these concretions.

Corpora Amylacea. Starch-like bodies have been found in the sputum after pulmonary hemorrhage and in that of pulmonary gangrene. They have the shape of starch-corpuscles, and sometimes give the amyloid reaction with iodine or iodide of potassium. They are at present without clinical significance.

Parasites.

A. Animal Parasites. Echinococcus cysts are to be found in sputum, generally broken into fragments, and only very rarely in a perfect whole, when there is rupture of a cyst of the liver or lung into a bronchus. Scolices and free hooklets from the same may be recognized, and pieces of the cyst-wall will be known by their remarkable formation. Their presence is of great clinical value.

Infusoria have been found in the expectoration from gangrene of the lungs. They belong to the monad and cercomonad varieties.

Distoma hæmatobium eggs may occur in sputa when the lung-tissue is broken down by its presence, the eggs being thrown off in the sputum.

The distoma Westermanii or pulmonalæ is found in the sputum in Japan in certain cases resembling phthisis. Both the worm and the ova may be present in the sputum.

Amœba Dysenteriæ (*Amœba Coli*). Of far more interest and importance is the presence of this parasite in the expectoration. A full description of the amœba will be given in the article on Dysentery.

They are the same in every respect when found in the sputum, except that they are often slightly larger. The sputum containing the amœba is partly diffluent, tenacious, frothy, bright red in color at first, due to the presence of blood, and later brick or brownish-red, sometimes bile-stained. Small yellowish-white cheese-like particles are seen. Upon exposure to the air the sputum becomes thin, syrupy, and oily, and it then looks much like anchovy sauce. The sputa are alkaline and of a faintly sweetish odor, never putrid. Later on they become more purulent, somewhat nummular, reddish-yellow, and contain less blood. If there is a favorable termination, they become more fluid and frothy, with less blood and pus, and, on standing, show the three layers. The quantity varies from 25 c.c. to 500 c.c. in twenty-four hours. Under the microscope will be found, beside the amœba, red blood-corpuscles, leucocytes, alveolar and oval epithelium, and bodies looking like degenerated liver-cells without a nucleus; occasionally elastic fibres, hæmatoidin, leucin, tyrosin, and Charcot-Leyden crystals and bacteria are seen. The cheesy particles are made up of amorphous granular matter and oil-globules. Amœbæ are constantly present in varying numbers, usually not so many as in the stool, but somewhat larger. The number varies from day to day, and diminishes with the disappearance of the cough and expectoration. The sputa should be examined as soon after their discharge as possible, and in the interim should be kept at a temperature of 30° to 35° C. If examined on a warm stage, active movements of the amœbæ will be kept up much longer.

They should be examined under various powers: $\frac{1}{2}$, $\frac{1}{5}$ or $\frac{1}{7}$, and $\frac{1}{12}$ inch objectives. Of these the $\frac{1}{5}$ or $\frac{1}{7}$ inch will be found most suitable for following the movements. They measure from 10μ to 20μ. They will be readily recognized by their size, formation, and movements. That they have important clinical value is true, as cases have been reported in which the observer diagnosticated hepatic or hepato-pulmonary abscess secondary to amœbic dysentery, by the peculiar anchovy-sauce expectoration and subsequent detection of the amœbæ.

B. Vegetable Parasites. Fungi—Non-pathogenic: Moulds. *Oïdium albicans* may be a constituent of the sputum when the bronchi are invaded by it, but usually it is from the saliva. Certain other moulds have lately been considered to cause disease of the lungs by multiplication, but nothing very definite has resulted from the experiments thus far made.

Yeast-fungi. Von Jaksch reports having seen scattered yeast-cells in the pus from a phthisical cavity. Otherwise we have no knowledge of yeast being found in sputa.

Fission-fungi. Leptothrix. Leptothrix occurs alone, in the sputum or in the bronchial plugs, in putrid bronchitis, along with the fatty acid and hæmatoidin crystals. It is probably derived from the mouth, having thence entered the air-passages, or it is taken up from the mouth by the expectoration. It is recognized by its staining blue with iodine and potassium iodide.

Sarcinæ Pulmonalis. Sarcinæ may be seen in sputa. They are larger than sarcinæ ventriculi, with which they have no connection, nor have they pathological significance when present in sputa.

Non-pathogenic bacilli and cocci may occur in all sputa, but are without significance. They are more numerous in fetid sputa. They stain with methylene-blue and other simple dyes.

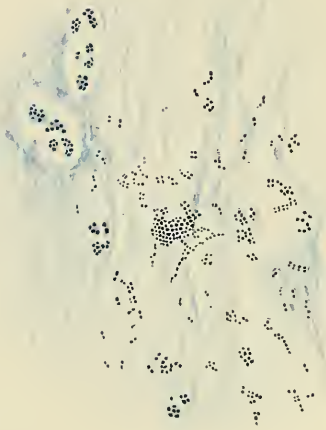
Pathogenic Fungi. Tubercle Bacillus. The organism which is the cause of tuberculosis is a rod, straight or slightly curved, without motion, varying in length from 2μ to 5μ (about $\frac{1}{4}$ to $\frac{1}{2}$ the diameter of a red corpuscle). It usually has a beaded appearance when stained, due to the spores, which do not take up the stain that affects the rod as a whole, and which often bulge slightly beyond the edge. It is probable that this beaded appearance is caused by the contraction and breaking up of the stainable portion, permitting us to see the empty spaces between the fragments and the other membrane. Bacilli presenting this appearance are supposed to be undergoing degeneration. Attention has recently been called to the presence in the sputum of branching forms of the tubercle bacillus. The bacillus of tuberculosis cannot be recognized in the sputum unless stained, and in the staining it shows a peculiarity which belongs to but few organisms—the smegma bacillus, the bacillus of leprosy, and the bacillus of syphilis. As under ordinary conditions these bacilli are not met with, this peculiarity in staining in a vast majority of cases is diagnostic of tubercle bacilli.

Recently, Pappenheim found in the sputum from a case of gangrene of the lung stained by Gabbet's method numerous bacilli which were considered to be tubercle bacilli. At the autopsy no evidence of tuberculosis could be found. Further examination led Pappenheim to believe that these bacilli were smegma bacilli. A similar case has been recently seen where large numbers of bacilli were present in the sputum in a case of gangrene of the lung secondary to a sub-diaphragmatic abscess, which, stained by Gabbet's method, were considered to be tubercle bacilli. The autopsy showed no evidence of tuberculosis, macroscopically or microscopically. Inoculation from the lung into a guinea-pig was also negative. Fraenkel has observed similar bacilli in the sputum when stained by Gabbet's method from patients with bronchiectasis.

Preparation of Sputum and Method of Staining Tubercle Bacilli. A small amount of the purulent portion of the sputum is spread in a thin and uniform layer on a perfectly clear cover-glass by means of forceps, needles, or the "oese," which must previously be held a moment in the flame of a Bunsen burner or spirit lamp; or by pressing a small amount of sputum between two cover-glasses, then sliding them apart. It is then dried in the air, or more quickly by holding the cover-glass with forceps some distance above the flame of a burner or lamp. Finally, it is to be passed three or four times through the flame and so "fixed." The edge of the cover-glass, with sputum side up, is then grasped with forceps and covered with the staining solution, care being taken to prevent the fluid from extending to the under surface, and held in or just above the flame, until the solution boils for a second or two or a bubble rises. When the excess of the solution is washed off in water, the slip is treated with the decolorizing agent until the color is almost or wholly removed. It is again washed in water to remove the excess of the decolorizer, and

PLATE XV.

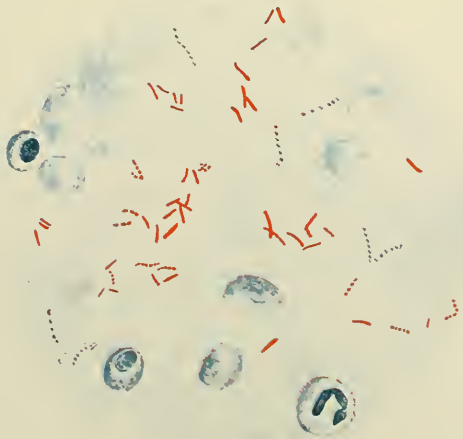
FIG. 1.



Pneumococci from a Case of Empyema.

(Oc. 4, ob. $\frac{1}{12}$ immersion.) Drawn by J. D. Z. Chase.

FIG. 2.



Tubercle-bacilli (red). Streptococci (blue chains).

(Oc. 4. $\frac{1}{12}$ oil immersion.) Drawn by J. D. Z. Chase.

mounted for examination, or given a contrast-stain ; the latter is preferable.

A second rapid method is as follows : Select with the sterilized oese a suspicious yellowish particle from the sputum ; smear it thinly over one end of a slide which has previously been passed several times through the flame of an alcohol lamp or Bunsen burner. Dry by holding over flame ; fix by passing several times through the flame. Cover the dried sputum with the desired stain, and steam gently for two minutes over the alcohol or low Bunsen flame ; the slide can be held in the fingers, or, after heating, can be laid aside for a moment ; wash off the excess of stain with water, then cover the stained sputum with decolorizing agent and counter-stain, which should not remain more than thirty seconds. Wash away excess with water, dry the slide by blowing upon it through a pipette, and cover with a clear cover-glass, using distilled water as a mount. This method is extremely satisfactory for ordinary clinical work, especially with Ziehl's and Gabbet's solution.

If fuchsin has been used to stain the tubercle bacilli, methylene-blue is a good contrast-stain ; while if gentian-violet was selected Bismarck-brown is better in contrast. These contrast-stains are made as needed by dissolving enough of the dye in a few c.c. of water to make the solution as seen through a test-tube of 14 mm. diameter only transparent, and then filtering ; or, a concentrated watery solution may be made for stock just as the concentrated alcoholic solutions of fuchsin and gentian-violet were made, diluting a small quantity of this when needed with enough distilled water to make it just transparent in a similar test-tube. To apply the contrast-stain, place a few drops on the cover-glass that has been prepared as above—stained, decolorized, and washed—allow it to remain thirty or forty seconds, wash off in water, and mount for examination on a glass slip, in water, oil of cloves, or Canada balsam. A drop of water will serve perfectly well for examining when the preparation is not to be preserved. In the microscopical examinations a $\frac{1}{12}$ inch oil-immersion lens and Abbe condenser, or, at the least, a $\frac{1}{7}$ or $\frac{1}{8}$ inch objective is used. If gentian-violet has been used, the tubercle bacilli appear as dark-blue rods, with all other bodies brown, if Bismarck-brown is used for contrast-stain while with fuchsin staining for tubercle bacilli, and methylene-blue as a contrast, the former will be found as red rods in a blue field (background). (See Plate XV., Fig. 2.)

The above rapid method of staining takes much less time than the method usually described, and gives most satisfactory results. The steps in the old method are the same as given above, except that instead of placing the staining solution on the smeared and dried cover-glass, and holding it in or above the flame until the solution boils, the cover-glass is floated in a cold solution, in a watch-glass, sputum side down, for twenty-four hours, or in a hot solution for six to eight minutes, or until moisture appears on the upper surface of the cover-glass. The remaining steps are similar.

Tubercle bacilli do not stain with the simpler dyes, but when stained by solutions of dyes made more penetrating by the addition of aniline

oil, carbolic acid, or like substances, *they retain the color when subjected to decolorizing agents.* In this they differ from all other organisms, except, as stated, the smegma bacillus, the bacillus of leprosy, and the bacillus of syphilis.

The Smegma Bacilli. Pappenheim distinguishes them from tubercle bacilli by staining with a solution of corallin in absolute alcohol saturated with methylene-blue, when decolorization takes place without acid. If fat acids and myaline are present in the sputa, the bacilli are, in all probability, not tuberculous. They are not found in mucopurulent, but in putrid, sputum.

Housell's method of staining them is the best. After the preparations are stained in carbol-fuchsin they are placed in a mixture of 3 per cent. HCl in absolute alcohol for ten minutes. They stain best with an alcohol solution of methylal-blue, which decolorizes the micro-organism.

A number of methods have been devised for the detection of the *tubercle bacillus* by means of its peculiar action toward stains. The most satisfactory are those known as the Koch-Ehrlich, Ziehl-Neelson, Gabbet, and Gibbes. These methods differ chiefly in the solutions used. Slightly modified from the original in execution, they are as follows :

A. Koch-Ehrlich method :

Solutions Used.

I. Concentrated alcoholic solution of fuchsin or gentian-violet.
II. Saturated solution of aniline oil in water.
III. Thirty per cent. solution of nitric acid in water (decolorizing solution).

I. Place in a clear bottle fuchsin or gentian-violet in substance to one-fourth its capacity, and fill with alcohol (95 per cent.) ; shake well and cork and allow it to stand for twenty-four hours. If all the dye has been dissolved, add more and shake, and let stand for another twenty-four hours, and so on until some of the dye remains permanently undissolved at the bottom of the bottle. This solution remains good until used.

II. To about 100 c.c. of distilled water, in a flask or other suitable vessel, add aniline oil, drop by drop, shaking the flask continuously, until the solution is opaque, or drops of the oil float on the surface, then filter through moist filter-paper until the filtrate is perfectly clear. This solution must be made fresh as needed.

III. Mix a few c.c. of nitric acid and water in about the above proportion, never stronger, each time bacilli are to be stained.

The Koch-Ehrlich solution is made by adding 11 c.c. of the fuchsin or gentian solution (No. I.), and 10 c.c. of absolute alcohol to 100 c.c. of the clear aniline filtrate (No. II.). It should not be used after it is a week old.

B. Ziehl-Neelson method :

Solutions Used.

I. Carbolic-fuchsin solution :

Distilled water	100 c.c
Carbolic acid (crystalline)	5 grammes.
Alcohol	10 c.c.
Fuchsin in substance	1 gramme.

This solution can also be prepared by adding saturated alcoholic solution of fuchsin (see above) to a 5 per cent. watery solution of carbolic acid, until a metallic lustre is seen on the surface of the fluid. This solution does not decompose so easily as those made with aniline oil.

II. Decolorizing solution of nitric acid, and
III. Contrast stain of methylene-blue, as above.

The preparation and staining are exactly the same as in method *A*. The tubercle bacilli are stained red, the other bodies blue.

C. Gabbet's method :

Solutions Used.

I. Carbolic-fuchsin solution (as in *B*).
II. Methylene-blue solution :

Methylene blue[1]	2 grammes.
Sulphuric acid	25 "
Distilled water	75 c.c.

This solution is apt to decompose if old.

Preparation of Slips and Staining. The cover-glass is prepared and stained with the carbolic-fuchsin solution and washed in water as in *A*. Then (instead of decolorizing with nitric acid or adding in contrast-stain) the slip is washed for twenty to thirty seconds in the methylene-blue solution, until a faint blue replaces the red tinge in the (slip) sputum ; the excess of the solution is washed off in water, and the slip is mounted and examined as above. The tubercle bacilli are stained red and the other bodies blue. In sputum from gangrene of the lung and bronchiectasis, decolorization with alcohol, in addition, must be employed to eliminate the presence of the smegma bacillus.

The writer has found that this method can be rapidly applied, and that it gives good results ; he recommends it highly.

D. Gibbes' method :

Solutions Used.

I. *a*.	Fuchsin	3 grammes.
	Methylene-blue	1 "
	Mix thoroughly in a mortar.	
b.	Aniline oil	5 c.c.
	Alcohol	20 c.c.

Dissolve and add *b* to *a* slowly, stirring vigorously until *a* is evidently dissolved, then add 20 c.c. of distilled water, and keep in a stoppered bottle, ready for use.

Prepare slip and stain with this solution, as with the others, up to the point of decolorizing. Then wash with alcohol until the dye ceases to come away. Mount and examine as above. Tubercle bacilli will be stained dark red, the other objects dark blue.

When the bacilli are few in number, Biedert proposes that the following preliminary steps be taken : About 4 c.c. of sputum are mixed with 8 c.c. of water and 1 c.c. of solution of caustic soda, and boiled a

[1] An alcoholic solution of methyl-blue should first be made, and then added, drop by drop, with constant stirring, to the sulphuric acid and water.

few minutes, when about 15 c.c. of water are added and the whole again boiled until a homogeneous fluid is formed. This is allowed to stand in a conical glass for twenty-four to forty-eight hours, when the sediment is stained by the Ziehl-Neelson or Gabbet method. Or, the homogeneous fluid can be put at once in a centrifugal machine, and the resulting sediment stained.

Sputa hardened in Zenker's fluid, embedded in paraffin and cut, has proven most satisfactory in the study of the branching forms of the tubercle bacillus, the study of giant-cells in the sputum in phthisis, and in the study of bacteria in the sputum in cases of pneumonia.

It is well to remember that, in the absence of a proper decolorizing agent, hot water applied for some minutes has been shown to decolorize very satisfactorily.

Importance. The greatest importance attaches to the presence or continuance of tubercle bacilli in sputa. It indicates tuberculosis of the lung or larynx ; in the vast majority of cases of the former.

They are often to be found in the sputum when physical signs are not yet present or are indefinite. The number varies so greatly in different cases, and in the same case at different times, that a in recent attack it is impossible to judge of the extent of the disease by the number present in a given preparation.[1]

The absence of bacilli from sputa has no true value unless negative results are obtained after many trials and careful examination by an experienced observer, using good stains. Hence, too great care cannot be taken in each and every step.

Biological Properties. The tubercle bacillus is difficult to cultivate, as it grows readily only in conditions found within the body. The best medium is blood-serum. The cheesy mass from the sputum or the tubercular nodule from a tissue is placed on the surface of the serum and rubbed carefully over it. It is best to make twenty or thirty such inoculations. The tubes must then be sealed to prevent evaporation and drying, and exposed for twelve days to a temperature of 37.5° C. When a pure culture is obtained further cultivations may be made on agar-agar, to which 6 per cent. of glycerin has been added.

The pure cultures appear as dry masses on the surface of the medium, either as flat scales or clumps of mealy-looking granules. They are of a dirty drab or brownish-gray color. (See Plate VII., Fig. 6.) The bacillus is parasitic, aërobic, non-motile (facultative anaërobic).

Pneumococcus. Diplococcus Pneumoniæ. Micrococcus Lanceolatus. The causative factor in most cases of acute croupous pneumonia in its typical form is a paired lancet-shaped coccus, often irregular in size, with a tendency to chain formation. Frequently oval or conical forms are present, and there is apt to be variation in the size of the two cocci forming the pair. The organism has a distinct capsule. In the sputum of croupous pneumonia these pneumococci are usually present in large numbers. Their presence within leucocytes

[1] "A Method for the Examination of the Actual Number of Tubercle Bacilli in Tuberculous Sputum." By George H. F. Nuttall, M.D., Ph.D., Johns Hopkins Hospital Bulletin, May, 1891. The method is of pathological but not of diagnostic interest.

and their tendency to chain formation has been especially noted in such cases.

Pneumococci are stained in cover-glass preparations with the ordinary aniline dyes, as given above. The capsule may be stained and differentiated in the same way, but it more often requires a special method. Welch recommends the following: Spread and dried cover-glass preparations are treated first with glacial acetic acid, which is allowed to drain off, and is replaced (without washing in water) with aniline oil-gentian-violet solution. (See under Tubercle Bacilli.) The staining solution is repeatedly added to the surface of the cover-glass until all of the acid is displaced. The specimen is now washed in a weak salt solution (about 2 per cent.), and examined in the same, not in balsam. The capsule and coccus can then be differentiated. Sputum stained by Gram's method, thoroughly decolorized by alcohol, counter-stained with a watery solution of eosine, or a 1 per cent. aqueous solution of aurantia, has been found satisfactory for microphotographic work. Degenerative and involution forms are constantly met with. There will be variations in size and shape, and the capsule may contain only remains of a coccus, or be entirely empty. (See Plate XV.)

Biological Properties. The pneumococcus is not motile. It stains by Gram. It grows well on blood-serum. The growth is minute, transparent, colorless colonies, resembling drops of dew. A favorable growth of very minute colonies appears in glycerin agar-agar. Bouillon is faintly clouded. Litmus milk will sometimes turn pink and coagulate. Growth on other culture media is usually feeble. The tendency to form chains is especially observed in the water of condensation on blood-serum tubes. The lancet shape of the cocci enables them to be differentiated from the streptococcus. The capsules are not usually observed in the cultures with ordinary methods of staining.

By inoculation into susceptible animals a typical fibrinous pneumonia is developed. The pathogenic power attenuates rapidly in cultures, but recovers its virulence by passing through susceptible animals.

This micro-organism is found in nearly all cases of acute croupous pneumonia, and in many cases of bronchopneumonia. Its presence has also been observed in health in the saliva. It is found also in acute pleuritis, endocarditis, pericarditis, peritonitis, acute purulent meningitis, and otitis media. Its presence in empyema is considered of favorable import. It has also been found in cases of synovitis, osteomyelitis, and abscess formation in various situations. It may cause a general septicæmia—*i. e.*, pneumococcus septicæmia.

Bacillus Mucous Capsulatus. This organism is found in the sputum in health in a certain number of cases. In association with the pneumococcus it can cause pneumonia. It can also produce pneumonia by itself in rare instances.

In three fatal cases of pneumonia due to the capsule bacillus alone, there have been found in the sputa large numbers of capsule bacilli. These were frequently inside of leucocytes, and many alveolar cells were filled with these bacilli.

Bacillus of Influenza. This organism is found in the sputum in cases of influenza or influenza pneumonia. It was first isolated from the sputum by Pfeiffer. The organism appears as a small bacillus with rounded ends. Its length varies somewhat, and thread-like, involution forms may appear. It stains more deeply at the ends than at the middle, and the long forms may show irregularity of staining. It does not grow on the ordinary media. It is best cultivated upon agar-agar slants, upon the surface of which has been smeared a few drops of blood. The colonies appear after twenty-four to thirty-six hours as minute, colorless, watery, clear, dew-like colonies, best seen with a hand lens. In the sputum these bacilli are frequently present in large numbers in cases of influenza, and their presence filling up the protoplasm of the leucocytes and the purulent sputum of pneumonia is not uncommon. Thin smears of the sputum, stained with aniline oil-gentian-violet, somewhat decolorized with alcohol, and counter-stained with a 1 per cent. aqueous solution of aurantia, have shown these bacilli much better than the ordinary methods of staining with Löffler's methylene-blue or dilute carbol-fuchsin.

Whooping-cough. Minute bacilli have been discovered in the sputum in cases of whooping-cough by Czplewski, Koplik, Zusch, and others. At present the results are not sufficiently uniform to prove these bacilli of etiological value in the disease.

Actinomyces. When the lungs or pleura are infected by this fungus actinomyces may be found in the sputum. The disease in these organs is rare. Macroscopically they appear as small kernels, yellowish-white or greenish-yellow, and having the shape of a millet-seed. Under the microscope they are recognized by the rounded, club-like bodies projecting from all sides of an unformed central mass. They are seen better when not stained. (See page 352.)

Chemistry of Sputum. As the chemical examination of the sputum does not aid us in diagnosis, it has but little or no value. Mucin, nuclein, and serum albumin are constituents of sputa in health. Peptone is present whenever there is pus, and is especially marked in pneumonia. Volatile fatty acids, such as butyric and acetic, occur at times, markedly so in pulmonary gangrene. Glycogen has been obtained by Solomon, and a ferment resembling one of the pancreatic ferments has been detected, especially in pulmonary gangrene and putrid bronchitis. Of inorganic substances, chlorides of soda and magnesia; phosphates of soda, lime, and magnesia; sulphates of soda and lime; carbonate of soda, lime, and magnesia; and in a few cases phosphate of iron and silicates have been obtained (Von Jaksch).

SPECIAL DIAGNOSIS.

Pictoric Records of Physical Signs.

In order to draw accurate conclusions from the various data obtained during the physical examination of a patient, the physician must carry in his mind the results of the inspection, the palpation and percussion, and the auscultation of each individual part of the thorax and abdomen. For the beginner the grouping

PLATE XVI.

FIG. 1.—Anterior Aspect.

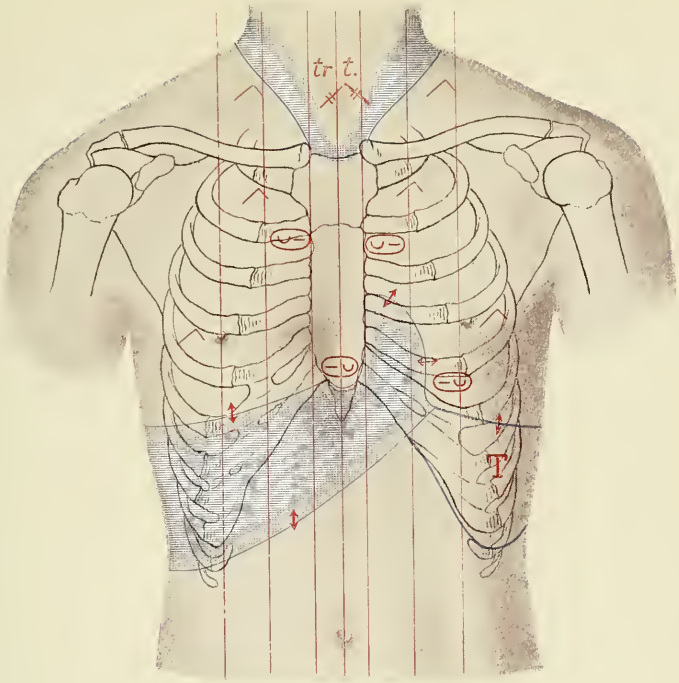
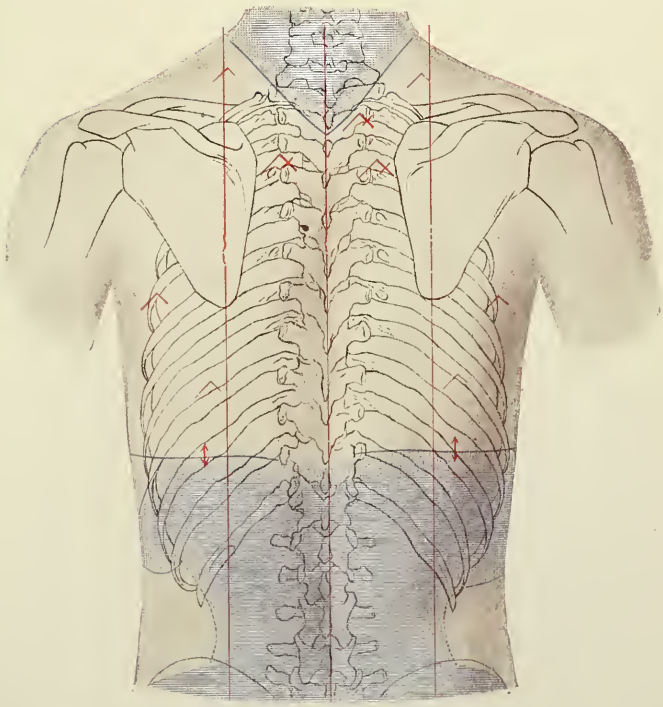


FIG. 2.—Posterior Aspect.



Physical Signs in Health.

Normal percussion outlines of the viscera. Normal heart and breath sounds. Vertical lines for localization.

together of these phenomena according to regions of the body, instead of by methods of examination, is extremely difficult. He is taught to examine the thorax, first, by inspection, then by palpation and percussion, and, finally, by auscultation; and in following this routine the results of the examination naturally divide themselves into the signs obtained by this method or that. In making the diagnosis, however, the grouping must be rearranged, for in order to determine the condition of a certain organ or part of an organ, *all* the local phenomena, by whatever method recognized, must be considered in their relation to one another and not merely as isolated facts. By weighing all the evidence obtained by the various methods of examination, and by balancing the relative importance of this sign or that, a verdict is finally reached in regard to the condition of the part in question. Only after the status of each organ has been thus separately determined can a complete diagnosis of the case be made with certainty.

In describing in the text the physical signs of the various diseases of the internal organs, it is necessary, in order to avoid endless confusion, to consider data in the order in which they are elicited—*i. e.*, grouped according to the method of their recognition. To redescribe them grouped according to regions would involve constant repetition, and would still fail to give a clear picture of the sign-complex of the part. And yet it is essential that this picture should be so clear and well defined that the physician, in summing up the examination, has but to glance at the part in order to call up to his mind all the various data obtained by its examination. Experience adds daily to the facility with which this piece of mental gymnastics is performed, and it finally becomes half-automatic, but for the beginner it is most discouragingly difficult. He may, however, obtain great assistance in acquiring the right habit of thought by systematically writing down each sign *as it is perceived*, and by grouping with it the other signs belonging to the same region. This he may do by means of short descriptions, or, better still, he may employ symbols to represent the various sounds, etc., and may mark them directly on the patient's body, or may fill them in on blank diagrams of the thorax and abdomen, and thus obtain a complete and vivid picture of the results of the examination of each separate region The practical value of this method, both as an aid to the beginner and as an easy and accurate means for preserving records, has been widely recognized, and numerous symbols have been devised, to represent graphically the various physical signs. Those suggested by Wyllie, of Edinburgh, and by Sahli, of Bern, are among the best. Many of the symbols used in the following plates will be recognized as borrowed from the above authors.

Explanation of the Symbols Used in the Plates Illustrating Special Diseases.

Percussion Sounds. Superficial dulness (also called absolute dulness) is alone indicated in the following plates. As has already been stated, the personal equation enters so largely into the determination of the extent of deep (relative) dulness that it is scarcely possible to make any positive statements in regard to the areas over which it is obtained in health and in disease. Absolute dulness is, on the other hand, easily recognized, and it is, therefore, far better that the student first become thoroughly familiar with this, about which there can be little or no question, before being taught what, in the case of relative dulness, is after all merely the expression of the individual skill and acuteness

of ear of the instructor. With a clear picture of the areas of superficial dulness once firmly fixed in the mind, the student should for himself determine just how far he individually is able to rely upon his perception of deep dulness. As his skill in percussion increases, and as his ear becomes better trained, he will find himself progressively better able to make use of deep dulness as an aid in diagnosis. He should, however, remember that many skilled diagnosticians are content to rely almost exclusively upon superficial dulness.

Blue shading = Areas of superficial dulness; the intensity of the color expresses the intensity of the dulness.

HR = Hyper-resonance.

T = Tympany; the pitch is indicated by a dot above or below the letter.

Breath-sounds. An ascending line indicates inspiration; a descending line expiration. The length of the line shows the length of the sound, the thickness, its intensity. A dot above or below the line indicates high or low pitch. Two cross lines are used to designate bronchial breathing; a single cross line indicates bronchovesicular breathing. An interrupted line stands for cog-wheel or interrupted breath-sounds.

= Normal vesicular breath-sounds.

= Weak vesicular breath-sounds.

= Harsh vesicular breath-sounds (puerile breathing).

= Harsh vesicular inspiration, prolonged vesicular expiration.

= Sharp vesicular inspiration, slightly prolonged vesicular expiration.

= Interrupted (cog-wheel) breath-sounds.

= Bronchial breath-sounds (bronchial breathing), inspiratory and expiratory.

= Bronchovesicular inspiration, low-pitched bronchial expiration.

Rales. *Dry râles* are represented by undulating lines, the length corresponding to the duration, while a dot above or below the line indicates the pitch.

= Sonorous râles.

= Sibilant râles.

Moist râles are represented by circles the diameter of which indicates the size of the râles. An ascending line drawn through the circle shows that the râle is heard during inspiration, a descending line that it is heard during expiration. The clear, sharp, moist râles heard over consolidated areas, râles with over-tones, are indicated by large or small dots, according to their size.

= Small, moist (subcrepitant) râles.

= Medium-sized moist râles.

= Large moist râles heard during both inspiration and expiration.

= Large and small moist râles.

= Small moist râles heard over consolidated areas.

= Medium-sized moist râles heard over consolidated areas.

= Large moist râles heard over consolidated areas.

= Large and small moist râles heard over consolidated areas.

Crepitation.

= Crepitant râles, to be heard only during inspiration.

Friction Rub.

= Friction rub, as heard over any serous surface.

Heart-sounds. The symbols used to indicate the feet in Latin poetry are made to represent the heart-sounds. The straight line indicates the longer, the curved line the shorter sound. The thickness of the lines shows the relative as well as the absolute loudness.

– ∪ = Normal heart-sounds as heard over the mitral and tricuspid regions.

∪ – = Normal heart-sounds as heard over the aortic and pulmonic regions.

∪ – = Normal first sound, accentuated second.

– ∪ = Loud first sound, reduplicated second.

– – = Loud first and second sounds of equal intensity.

Murmurs. Murmurs are represented by short parallel lines either increasing or diminishing in length, according as the murmur increases or diminishes in intensity. The thickness of the lines shows the loudness of the murmur, the number of lines shows its duration.

= A soft murmur, commencing distinctly and gradually fading away.

 = A loud murmur of the same character.

= A short loud murmur, increasing in intensity (type of presystolic murmur).

= Loud first sound, slightly accentuated second sound; short loud presystolic murmur, increasing in intensity to end with the first sound; long, soft, systolic murmur.

Fremitus.

F + = Increased fremitus.

F – = Diminished fremitus.

No **F** = Absent fremitus.

Other Symbols.

X = Impulse.

M = Margin (of an organ).

R = Retraction.

B = Bulging.

v = Visible.

p = Palpable.

X vp = Visible and palpable impulse.

M vp = Visible and palpable margin.

The Neuroses.

The neuroses are affections of the lungs unattended by structural change. To this class belong the varieties of rapid breathing, of slow breathing, of cough and of dyspnœa which appear to arise without structural change, and which are discussed exhaustively in the section devoted to the subjective symptoms. Among other neuroses, *asthma* is fully treated of, and other forms of dyspnœa and cough are considered. Reference need not be made further to the respiratory neuroses other than to bear in mind that their presence may or may not be unattended by organic change in the lungs. On the other hand, we are likely to find the general phenomena or stigmata which are associated with neuroses of other organs, as well as the lungs. Hence, the condition of neurasthenia is likely to be present on the one hand, or the numerous stigmata of hysteria may be found on the other.

The Congestions.

Congestion of the Lungs. Active Congestion. In *active congestion* there is an increased amount of blood, which diminishes the air-space by encroachment and causes more or less consolidation. The signs of that physical condition are present—increased fremitus, impaired resonance or dulness, and bronchial breathing. They are observed on both sides, usually at the bases. Dyspnœa, cough, and frothy, bloody expectoration attend the fluxion. No cases have yet been reported in which bacteriological examination of the sputum was made. Of course, the *micrococcus lanceolatus* is not found.

If the above signs and symptoms develop suddenly—within twenty-four hours—a fluxion to the lung has in all probability taken place. If the patient is subject to heart disease, or if he has been exposed to and has inhaled hot vapors or irritants, the probability of fluxion is increased. The occurrence of fever would point to *pneumonia* as the cause of the objective and subjective symptoms.

Passive Congestion. The physical condition that results is consolidation, manifesting itself by slight dulness and feeble or bronchial breathing; the bronchial mucous membrane is also congested, giving rise to abundant large râles. The affection is bilateral and usually confined to the posterior portions of the bases. It is also secondary. *a.* Mechanical congestion occurs when the flow of blood to the heart is obstructed, as in organic valvular disease or insufficiency. Rarely the pressure of tumors on the pulmonary veins acts in a similar manner. *b.* Hypostatic congestion occurs in fevers, as protracted typhoid, and in prolonged general exhaustion or adynamia. Ascites or other affections below the diaphragm, which lessen the respiratory excursion, cause this form. *Dyspnœa, cough,* and expectoration of blood-stained sputum are common. The sputum contains alveolar cells, often pigmented, but no micro-organisms.

Œdema. The air-cells and alveolar walls are filled with serous exudation, as in œdema of the skin. It is frequently due to the weakness of the heart, which occurs at the end of long-continued diseases

of an exhaustive nature, particulaly if the heart is overtaxed. It occurs, therefore, in the terminal stages of chronic Bright's disease, of organic heart disease, of the anæmias and cachexias. Both congestion and œdema occur in cerebral affections.

Symptoms. They are those of congestion in a more aggravated form. Dyspnœa, cough, and the expectoration of large quantities of a seromucoid fluid are seen. The diagnosis is based upon the result of physical examination and the history of the above causal factors. In cases of myocarditis or acute dilatation of the heart, in valvulitis with failing compensation, œdema of the lungs often takes place suddenly. It may follow some unusual exertion. Its onset is attended with more or less collapse, increased pulse-rate, hurried, oppressed, noisy breathing, cyanosis, and an anxious expression. The *physical signs* are an unusual number of small râles throughout the chest, apparently created in the air sacs, and imperfect resonance, showing that some lobules are collapsed.

Pulmonary Embolism and Thrombosis. Pulmonary embolism consists in plugging of the pulmonary artery or its branches by coagula formed in the right heart or in the veins. The symptoms depend upon the size of the occluded vessel and upon the nature of the embolus—*i. e.*, whether septic or not. If the artery itself is plugged, death takes place suddenly or after a short interval, with symptoms of syncope or asphyxia.

Symptoms. If a large branch is plugged, the first symptom is generally intense dyspnœa, which may amount to an agonizing craving for air. Pain in the chest, which may or may not be acute, is complained of, and may be referred to the seat of the embolus. Cough is not a common symptom, and may be altogether absent. The breathing is considerably altered ; it is usually increased in frequency, and may be much hurried ; it may or may not be shallow, and while the patient can take a deep inspiration, it does not give relief to his dyspnœa. At times it is irregular and gasping.

The face is pale or may be cyanosed, and is apt to be bathed in perspiration. The veins are swollen and prominent. The heart's action is irregular and may be tumultuous. Exophthalmos has been observed. The temperature falls below normal, but a febrile rise may occur later. The intellect is unclouded.

The *physical signs* are indefinite. The respiratory murmur is roughened and exaggerated in most, but not in all cases. Fox states that râles are very rarely heard. Collapse, œdema, and bronchitis are possible results. A systolic blowing murmur may be heard over the heart and pulmonary artery, and in protracted cases albuminuria and œdema may be met with.

When the embolus is septic, a *septic pneumonia* or *metastatic abscesses* are probable results in cases not immediately fatal.

When the emboli produce *hemorrhagic infarcts* the symptoms are milder, and consist principally in dyspnœa, pulmonary hemorrhage, and palpitation. The onset is sudden and accompanied by a fall in temperature. The physical signs indicate consolidation, if the pneumonia or infarcted area is of moderate size. It may be discovered at the root of the lungs in the interscapular region.

Hæmoptysis is a common symptom when the embolus has arisen in the heart. The amount of blood varies from a copious expectoration to the rusty sputum seen in pneumonia; it may persist for weeks. *Pleurisy* and pleural effusion are frequent complications; chills occur sometimes, and *pneumonia*, with corresponding rise of temperature, may develop.

The most important points in diagnosis are the sudden onset of the dyspnœa and other pulmonary symptoms, and the detection of a condition which would give rise to emboli, such as puerperal fever or heart disease.

The Inflammations.

The Bronchi. Inflammations of the bronchi are distinguished from other diseases of the lungs chiefly by the difference in the physical signs. Except in capillary bronchitis, the general and subjective symptoms are not so severe as in other affections.

Signs Peculiar to Inflammations of Bronchi. We are aided in the recognition of bronchial affections, first, by the fact that they are bilateral; second, that the bases are usually affected; third, that there is diminution of fremitus determined by palpation; fourth, that there is absence of dulness on percussion; fifth, that râles are more pronounced in proportion to other physical signs, and more general than in other lung affections.

Bronchitis. Bronchitis is an inflammation of the mucous membrane of the bronchial tubes. It may be acute or chronic, may involve any part of the bronchial tree, the large, the middle-sized, or the most minute branches, and may be primary, or occur secondarily to some general disease, or to disease of the heart or kidneys.

Acute bronchitis occurs most frequently by extension of the catarrhal inflammation from the nose and throat; but in some persons it develops so suddenly that it appears to be primary in the tubes.

When the *larger* or *middle-sized tubes* are involved, the patient complains of soreness or rawness underneath the sternum, especially at its upper part. There are frequently a feeling of tickling in the throat, and a sense of weight or oppression on the chest. Chest pain is due to myalgia or the strain upon the muscles from coughing. The *cough* is at first hard and dry, and often produces pain of a tearing character in the muscles of the chest and abdomen. The cough is apt to be worse when the patient first lies down, and again on rising, especially after a night's rest. *Fever* is usually slight and of short duration. The respirations are accelerated, but not markedly, and there is no dyspnœa. The *expectoration* is at first a white, frothy, viscid mucus, subsequently becoming more abundant and mucopurulent.

Physical Signs. In uncomplicated cases there are no changes in the physical structure of the lungs. On examination of the chest the percussion-note is found to be clear; the respiratory murmur more roughened and harsher than normal, but not bronchovesicular or bronchial; accompanying breathing there are heard sibilant and sonorous râles, and, in the later stages, some large and medium-sized mucous râles. The râles vary in position from time to time, and especially after

coughing. Vocal resonance and fremitus are unaltered. A fremitus may be produced by sonorous râles.

The cough and expectoration usually last for some time after fever has subsided. The duration of the disease is from a few days to several weeks. It is never fatal except in the very old and very young, or in those who are much debilitated.

The *diagnosis* of acute bronchitis is easily made by noting the fact that the disease runs an acute course, marked by fever, cough, and expectoration; and that the physical signs are negative, except as to roughening of the respiratory murmur and the existence of bronchial râles, heard on both sides of the chest.

From *croupous pneumonia* and *local tuberculosis* of the lungs it is distinguished by the absence of dulness on percussion, bronchial breathing, and increase of vocal resonance and fremitus; by the absence, in other words, of the ordinary signs of consolidation. From pneumonia it is further distinguished by the milder character of the subjective symptoms, and by the fact that in bronchitis the physical signs are almost always bilateral, in pneumonia generally unilateral. It is further distinguished from *tuberculosis* by the slow progress of the latter, which involves the apices preferably, whereas bronchitis is more marked at the bases; and by the occurrence, sooner or later, of hectic fever and emaciation, which are absent in bronchitis. Doubt will exist only at first; the progress of the case will in time make everything clear. Systematic examination of the *sputum* is an important diagnostic aid, and will lead to the differentiation of many cases of bronchitis from tuberculosis and from pneumonia. In infants and children especially, bronchitis is at times so rebellious to treatment that tuberculosis is suspected.

In *bronchopneumonia* (catarrhal pneumonia) there is a diffuse bronchitis associated with small areas of pneumonic consolidation. It is distinguished by having graver general symptoms and by the presence of small areas over which there are dulness on percussion and bronchial breathing, associated with physical signs of bronchitis already described.

Acute miliary tuberculosis of the lungs is very easily mistaken for bronchitis, because dulness, if present, amounts to nothing more than tympanitic dulness, because the signs are diffused through both lungs, and because the respiratory murmur is fainter than normal, but only slightly roughened. Close inspection of the patient will, however, make it evident that his condition is worse than could be accounted for by bronchitis alone. The fever is higher, the respirations more frequent, pallor, with a dusky or faintly cyanotic hue intermingled, is common, perspiration is more pronounced. A primary focus or a source of infection may be discovered.

Acute bronchitis may be mistaken for *spasmodic laryngitis* (croup). It is distinguished by the fact that the spasms are less pronounced in bronchitis, and there is fever in addition to the physical signs. In bronchitis the breathing is rarely so stridulous as in laryngeal spasm.

Whooping-cough cannot be distinguished positively from bronchitis before the characteristic whoop appears; but it may be suspected when

the child has been exposed to contagion, and when the coryza and redness of the fauces persist in spite of treatment.

In the diagnosis of bronchitis it is more often difficult to determine the primary cause than it is to distinguish it from other pulmonary affections. Yet the former is more important; it must be borne in mind that bronchitis is a frequent accompaniment of many febrile diseases, such as typhoid fever, measles, and whooping-cough; of diseases of the heart and kidneys, and of septic diseases and blood disorders. The primary will not be likely to be mistaken for the seconday disorder if one is upon his guard and insists upon finding a cause for each case that presents itself.

Measles can usually be diagnosticated from the first by the coryza, but especially by the red spots upon the anterior half-arches of the soft palate, which appear usually several days before the eruption upon the body.

Bronchitis is a common and important early symptom of *typhoid fever*. The latter disease may be suspected when the fever, prostration, and headache are greater, and, especially if these symptoms coexist with a loose condition of the bowels, chilliness, and occasional nose-bleed.

Chronic bronchitis occurs most frequently in middle or later life. Its special feature is long duration, without fever, and with comparatively little impairment of the general health. Cough is not constant; there are periods when it is entirely absent; the disease then returns, perhaps with increased severity, and lingers indefinitely.

Chronic bronchitis in its milder form consists in what is often called "winter cough." It attacks especially persons past middle life who have emphysema. It appears with the cold weather, and lasts until the following summer. The cough is not severe, though sometimes paroxysmal, and expectoration is scanty, non-purulent, and may be confined to the morning. Dyspnœa is not marked unless there is considerable emphysema. Acute exacerbations occur from time to time, and the tendency of the disease is to become worse from year to year, and to be more continuous, even persisting all summer.

In the *dry* catarrh, or *catarrhe sec* of Laennec, paroxysms of cough occur on the slightest provocation, with the expectoration of small, hard pellets, or without any expectoration. The patients are emphysematous.

The *diagnosis* is made by noting the long duration of the disease without impairment of the general health, its relation to season, and the absence of physical signs of involvement of lung tissue.

The *physical signs* of chronic bronchitis are those of bronchitis of the larger and middle-sized tubes. Large moist râles are more or less abundant, depending upon the degree of swelling of the mucous membrane, and the quantity and fluidity of the secretions. The respiratory murmur is roughened and less intense than normal.

W. Fox says that in chronic bronchitis there is commonly hyperresonance from coexisting emphysema, but under acute exacerbations the bases may be dull from congestion or œdema. Respiration is harsh, and in some cases of senile bronchitis expiration may be both

prolonged and high pitched, when other signs of dilatation of bronchial tubes are absent. The percussion-note is clear.

The *sputa* of the severe forms of chronic bronchitis are usually copious and mucopurulent, the latter predominating. They vary in color from yellowish-white to ashy, greenish, or black when the lungs are anthracotic or collapsed.

The *subjective symptoms* of the patient consist, in ordinary cases, of a moderate amount of dyspnœa, and tightness across the chest. At the onset of a fresh attack the symptoms may be those of acute bronchitis. The cough is paroxysmal, somewhat resembling that of whooping-cough, but without the characteristic whoop. It is usually severest on lying down and when rising in the morning.

The quantity and character of the sputa vary more than in acute bronchitis. Sometimes they are very copious, consisting of serum mixed with mucus, constituting *bronchorrhœa*. More commonly they are scanty, glairy, and tenacious.

Chronic bronchitis may be the result of repeated acute attacks, or, rarely, of only one. It is frequently found in association with gout, chronic heart disease, chronic endarteritis, Bright's disease, emphysema, asthma, and chronic alcoholism. It may alternate with other gouty affections, as articular inflammation or eczema, being relieved when the other manifestations are more marked. It also accompanies tuberculosis of the lungs. Climate and season have a marked influence; the disease is worse in damp, cold climates, and in the winter months.

Chronic bronchitis can be diagnosticated from the cough of *aneurism* by the absence of the stridulous breathing, due to paralysis of one-half of the vocal cords, and by the local signs of a tumor of the vessel. Other tumors may cause cough by pressure, and the possibility of their existence should, therefore, be borne in mind.

Capillary Bronchitis, or Suffocative Catarrh, is bronchitis of the smaller tubes. It occurs most frequently as an extension of the catarrhal process from the larger tubes, but sometimes seems to attack the smaller tubes from the beginning, or coincidently with the larger tubes. Infants, young children, and the aged are most liable to it. It begins with a succession of chills or chilliness, followed by high fever. The temperature may rise to 104°. The skin is hot, the face flushed. The head and neck and the upper portion of the trunk may be covered with perspiration. The pulse rapidly increases in frequency.

The aspect of the patient from the first shows that the illness is graver than ordinary bronchitis. The face expresses anxiety, and in children the alæ nasi dilate in respiration, which is both accelerated and difficult (*dyspnœa*). The respirations may be as many as 60 or 80 to the minute, the pulse not being correspondingly rapid. Dyspnœa is more or less constant, but becomes urgent in paroxysms, and the patient may have to be propped up in bed to enable him to breathe (*orthopnœa*). It is *expiratory:* inspiration may be free and easy, or difficult; but *expiration* is always difficult and prolonged. In children the pause in the act of breathing takes place at the end of inspiration, instead of expiration.

Cough is more frequent and violent than in ordinary bronchitis, and the expectoration is viscid and difficult to raise. As the disease progresses, dyspnœa becomes more intense, and signs of insufficient aëration of the blood make their appearance (*cyanosis*). The lips and finger-nails become bluish, and the extremities cool and clammy. If the patient is unable to expel the tenacious secretions from his bronchial tubes, the further progress of the case is that of rapidly developing cyanosis; the breathing continues frequent, but is shallow and more labored. Children often have convulsions, followed by coma and death, while old persons sink into coma without preceding convulsions.

The *physical signs* (Plate XVII.) are those of bronchitis of the larger and smaller tubes; sibilant and sonorous râles, if present at first, give way to fine subcrepitant and crepitant râles, which speedily become moist and very abundant. As an ordinary bronchitis, the bases of the lungs posteriorly are the parts most involved. The percussion-note of both lungs remains clear, but there is apt to be increased resistance. The fremitus may be lessened in some areas, increased in others. If an area of dulness appears, it may be due to pneumonia or to collapse of the lung; if the former, there is usually an access of fever.

The *sputum* contains mucus, pus, occasionally blood-cells, granular matter, and sometimes fibrinous casts of the tubes. The micro-organisms found are the *micrococcus lanceolatus*, streptococcus pyogenes, and staphylococcus aureus et albus. Mixed infections are usually present.

Plastic bronchitis is a form of bronchitis, usually chronic, the characteristic feature of which is the expectoration of fibrinous casts, which, when unravelled under water, are found to be solid casts of the smaller bronchial tubes. The casts are often tree-like in shape, showing that a bronchial tube and its smaller subdivisions have been occluded by the casts.

Persons of all ages are liable to it, but it affects males about twice as often as females.

The subjective symptoms are cough and dyspnœa; hæmoptysis occurs in about one-third of the cases (Biermer).[1] The cough occurs in paroxysms, which are frequent and severe; relief follows expectoration of the casts.

Hemorrhage may appear only as streaks of blood upon the casts, or may be considerable, and follow their dislodgement. The casts themselves when ejected are usually coated with mucus, so that they appear as solid masses of sputum; their arrangement into cylinders may not be suspected until they are agitated in water. The size of the cylinder varies from that of the little finger to that of a bodkin, but they do not often exceed the size of a goose-quill. The larger casts may be hollow, but the smaller ones are solid, and are arranged in layers. They are whitish or gray in color, and firm in consistence, but become softer as the disease improves. Microscopically, the casts are nearly structureless, consisting of a fibrillated base, with pus and mucous corpuscles, a few gland-cells, and, occasionally, blood-cells in the outer layers. Charcot-Leyden crystals and Curschmann's spirals are found.

[1] Virchow: Handbuch der spec. Path. u. Ther., Bd. v., Abth. 1.

PLATE XVII.

FIG. 1.—Anterior Aspect.

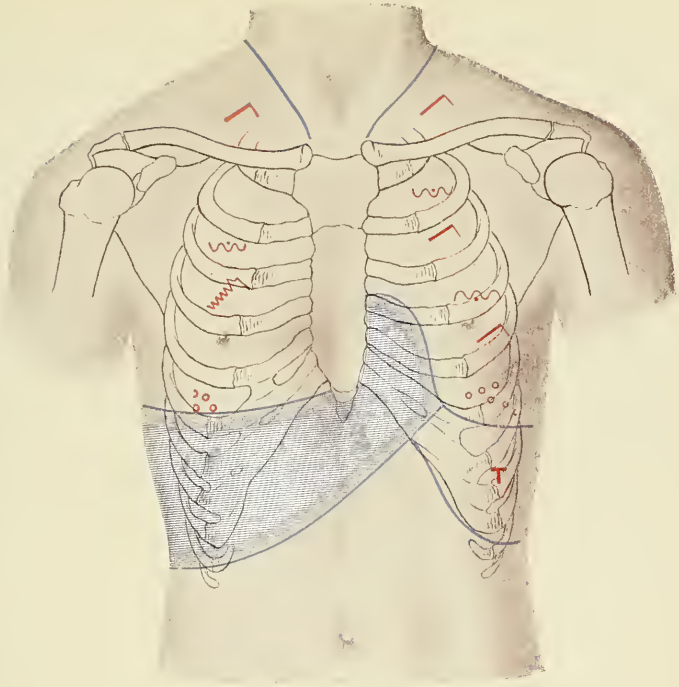
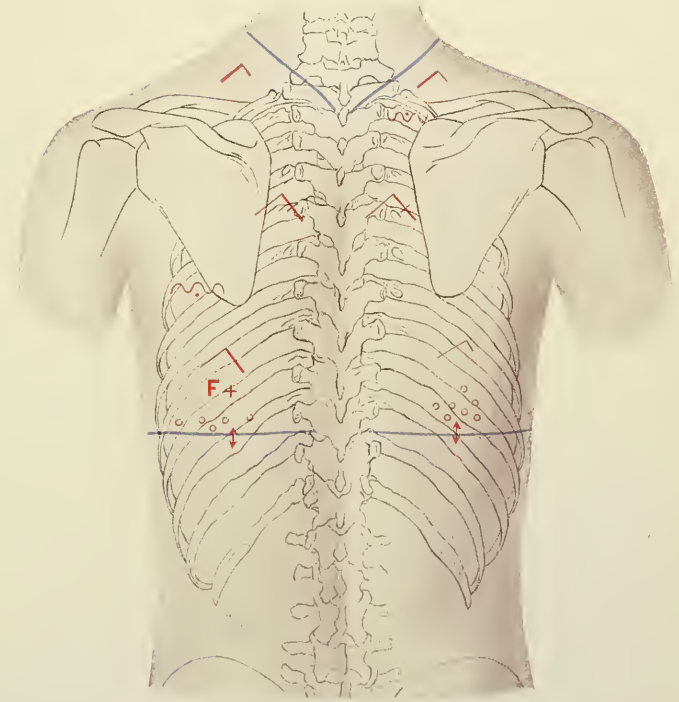


FIG. 2.—Posterior Aspect.



Capillary Bronchitis (early stage).

Rough or sharp breath sounds–expiration in places prolonged. Sonorous, sibilant and small moist rales. Local increase of fremitus.

The *acute* form is rare, and out of ten cases accepted by Biermer six proved fatal. The disease begins with fever, dyspnœa appears early, severe paroxysms of cough occur, sometimes hemorrhage. Death results from asphyxia. Grave symptoms are excessive dyspnœa, scanty expectoration, and drowsiness. Copious expectoration is a favorable sign.

The Physical Signs. The casts obstruct the bronchial tubes. There is less air entering the part, hence there are diminished fremitus and respiratory murmur over the portions of lung supplied by the obstructed tubes. If collapse ensues, there is dulness on percussion ; if the casts are dislodged, the murmur becomes normal, or but slightly roughened. In unaffected portions of the lung resonance is clear or exaggerated, and the respiratory murmur remains unaltered.

Fuller says (quoted by Peacock in *Diseases of Chest*) that the upper portions of the lungs are oftener affected than the lower portions.

Fetid or Putrid Bronchitis is the name applied to the condition in which the sputa have a highly offensive odor and are copious and semi-putrid. The odor is said by some to be due to microscopic sloughs, and by others to a special bacillus.

Putrid bronchitis may accompany (1) dilatation of the bronchial tubes ; (2) chronic pneumonia ; (3) phthisis or (4) empyema with a fistulous communication with a bronchus ; or (5) it may occur independently. The subjective symptoms are cough, irregular fever, and occasional chills. The *physical signs* are those of chronic bronchitis, or of bronchitis and the conditions with which it may be associated (*q. v.*). It is diagnosticated from *gangrene* by the absence of physical signs of disintegration of lung-tissue and by the absence from the sputum of fragments of lung-tissue and elastic fibres. Nevertheless, gangrene of the lung may be the final result of putrid bronchitis.

The sputa of fetid bronchitis have an odor of gangrene or fæces. On standing they separate into three layers. The upper one consists of a greenish, fluid, or frothy layer ; the second is sero-albuminous ; and the third a thick granular deposit in which are small masses, the size of peas (Dittrich's plugs), and flakes sconsisting of granular detritus, and containing fat-crystals and bacteria, the oïdium albicans, and crystals of leucin and tyrosin. (See Sputum.)

Infectious Bronchitis. In addition to the bronchitis that attends the infectious disorders mentioned above, three forms are seen of an infectious nature which are properly classified among the infectious diseases. It is proper to refer to them now, as bronchitis is usually the most pronounced local manifestation. They are *influenza*, *whooping-cough*, and *hay-fever*. The last only will be spoken of at present.

Hay-fever. Hay-fever is a specific catarrh of the respiratory passages, caused by the pollen of certain plants, principally the grasses. The attack begins with itching, burning, and lacrymation of the eyes, and pain in the brow or eyeballs. Subsequently there is itching or pricking of the nasal mucous membrane, frequent sneezing, and an irritating watery discharge. The mucous membrane of the nose is red and swollen. A similar condition obtains in the throat when that is affected. If the disease attacks the bronchial mucous membrane a

bronchitis is set up, which, if it differs at all from ordinary bronchitis, is more persistent and attended by greater dyspnœa, with asthmatic attacks.

Collapse of the Lung. Collapse of the lung is a condition produced by exhaustion of air from the air-vesicles. It may affect alveoli here and there, or a large section of the lung. Formerly such collapse was invariably looked upon as pneumonia, until Legendre and Bailly proved by forcible inflation that the air-vesicles had simply collapsed from absence of air. Collapse occurs most frequently in the course of bronchitis and in cases with feeble respiratory power. The bronchial twigs supplying certain air-vesicles, or tubes supplying sections of lung, become occluded to such a degree that no air can enter. The air already contained in the vesicles then becomes exhausted gradually until the vesicles are completely airless. The vesicles or sections of lung involved then return to the fœtal condition. When the collapse is congenital the term *atelectasis* is preferable. Anything which induces great muscular weakness predisposes to collapse of the lung; hence, in the aged and feeble, in wasting diseases, and in low febrile diseases of long standing, collapse is very apt to occur. But bronchitis is the most frequent and direct cause. The secretions which are poured out, and the swelling of the mucous membrane, occlude the tubes, and if the patient have not strength enough to expel the secretions, and by forced inspiration expand the collapsing vesicles, collapse ensues.

Diagnosis. The diagnosis of the condition in life is difficult. The site of collapse, being airless, is, of course, dull on percussion. The respiratory murmur is more likely to be faint or absent than to be increased in intensity or approach the bronchial. Nevertheless, there is sometimes heard a faint bronchovesicular expiration.

When œdema is superadded to collapse, moist crepitant râles are heard, difficult if not impossible to distinguish from those of pneumonia. Respiration is embarrassed, and is accompanied by sucking-in of the lower part of the chest in inspiration. Sometimes the plug of mucus which occludes the tubes becomes dislodged while the physician is auscultating, and then the respiratory murmur will be heard, accompanied by a succession of crepitant râles, which disappear after a few inspirations. The dull areas, as a rule, are less persistent than those of pneumonia; thus it may be found at successive examinations that one area has cleared up and another has become dull. Stress is laid by some writers upon the signs of emphysema surrounding collapsed areas. But this does not give assistance in the cases in which most help is required—cases in which there is diffuse bronchitis with more or less œdema.

Subjective symptoms are those of dyspnœa and insufficient oxygenation of the blood. If these are developed suddenly, and are accompanied by the appearance of dull areas in the lung without bronchial breathing, the diagnosis is tolerably certain; but when scattered lobules only are involved, the physical signs of collapse are absent, and its existence must be a matter of inference.

From *lobar pneumonia* the diagnosis is easily made by the difference

PLATE XVIII.

FIG. 1.

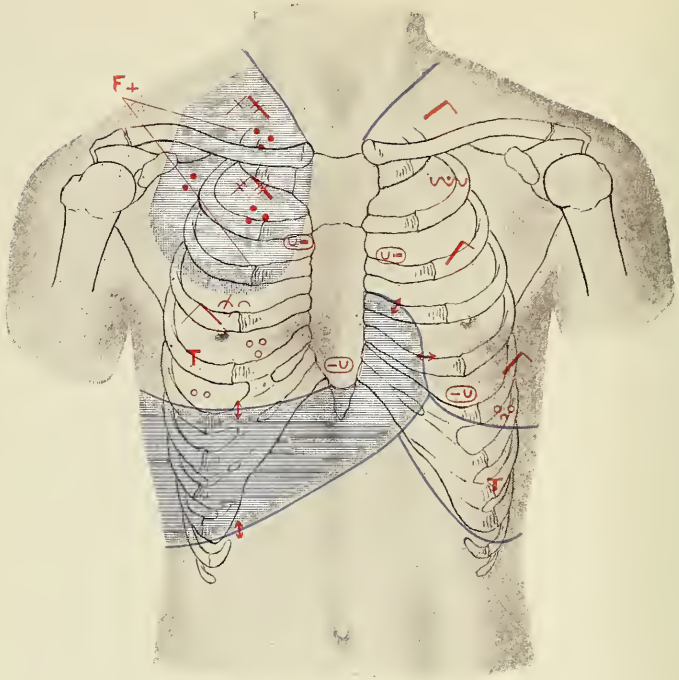
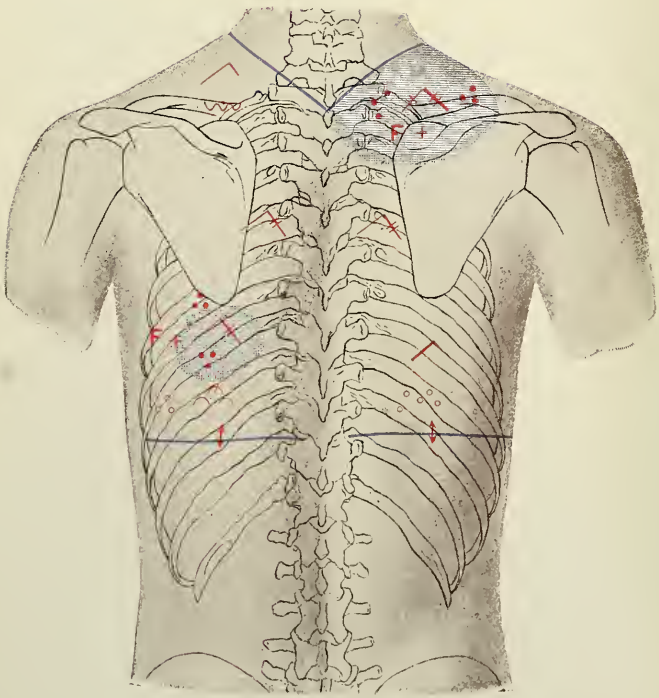


FIG. 2.



Broncho-pneumonia.

Consolidation in the right upper and the left lower lobes. Physical signs of bronchitis over both lungs.

in the physical signs, and by the absence in pulmonary collapse of inflammatory symptoms, by the lower temperature, and the difference in onset.

The diagnosis from *bronchopneumonia*, or *catarrhal pneumonia*, is beset with greater difficulties. But here also the low temperature, and the fact that the physical signs and the location of the dull areas are subject to rapid changes, are of aid in diagnosis.

The Bronchi, the Alveoli, and Connective Tissue.

Bronchopneumonia, or Catarrhal Pneumonia, is a pneumonia occurring secondarily to bronchitis, and is characterized by the development of areas of consolidation in both lungs and the persistence of a bronchitis of the middle-sized or smaller tubes. In proportion as the areas of consolidation are large, the symptoms and physical signs approach those of lobar pneumonia. It is more common in children and in debilitated persons. It is the chief form in infants. 1. It is frequently secondary to measles, diphtheria, scarlet fever, and pertussis. 2. As aspiration pneumonia, it occurs when food, septic particles, blood, or tissue enter the lungs during the loss of sensibility of the larynx in apoplectic, uræmic, or other forms of coma, and in operations about the upper air-passages and mouth. It is a fatal complication of tracheotomy. 3. It is frequently of tuberculous origin.

Catarrhal pneumonia, except the aspiration-form, develops gradually, and it may not always be easy to mark the point at which the bronchitis which precedes merges into pneumonia; but as a rule there are more or less chilliness (rarely a decided chill) and an access of fever. There is usually greater prostration than in the lobar form, in proportion to the amount of pneumonia present. The pulse is more frequent and more likely to be feeble. Cough and expectoration are marked symptoms. The sputum is tenacious and glairy, not rusty. It contains streptococci and staphylococci in much greater numbers than are found in ordinary bronchitis; fatty epithelial cells, epithelium, fat-globules, and diplococci.

Dyspnœa is more extreme than in lobar pneumonia. The respirations are excessively rapid—60 to 80 per minute; cyanosis rapidly ensues. The finger-tips become blue, the face dusky. The fever does not rise as high as in the lobar form. At first the skin is hot and dry; later it becomes cold and clammy, and in the tuberculous form sweats are common. The duration of the disease is usually much longer than in lobar pneumonia.

The *physical signs* (Plate XVIII.) are those of bronchitis, with here and there larger or smaller areas of consolidation, over which the râles are finer and closer set; the percussion-note is dull, and the respiratory murmur bronchial or bronchovesicular. An entire lobe may be consolidated. Areas of collapse and portions more or less œdematous combine to make the more complex physical signs. While both lungs are affected, they are not usually so to the same extent. It is said that the apices are more prone to involvement in this than in the lobar form; and some writers (Osler) look upon it almost, if not always, of tubercular origin.

In the common form seen in infants the symptoms of asphyxia set in at variable periods in the course of the disease. General cyanosis supervenes. Stupor sets in, the hurried respirations grow shorter and more gasping, the pulse becomes excessively rapid and feeble, the extremities cool and clammy; with the stupor the cough abates and the breathing becomes more shallow. The lungs fill up with fluid mucus, and the child drowns in its own secretions, or cardiac paralysis sets in after dilatation of the right heart.

Diagnosis. The affection is distinguished (1) by its pathological antecedents and causal relations; (2) its gradual onset; (3) its distribution in both lungs; (4) the preponderance of physical signs of bronchitis over those of consolidation; (5) the extreme dyspnœa and cyanosis with a lower temperature than in lobar pneumonia; (6) the onset of carbondioxide-poisoning; (7) the long duration and gradual decline. The *tuberculous* form is distinguished by (1) the history of exposure to infection or of a focus of infection in the body, glands, or joints; (2) the longer course; (3) delayed asphyxia; (4) rapid emaciation; (5) diffused sweats; (6) physical signs of consolidation and subsequently of cavity at the apex; and (7) absolutely by tubercle bacilli in the expectoration coughed up or vomited. I have seen a child aged fifteen months, of a tuberculous mother, completely recover. The tuberculous form is common in colored infants.

Bacteriological Diagnosis. Examination of the sputum shows an abundance of the streptococci and staphylococci and the special microorganism which belongs to the primary infection, as that of influenza, diphtheria, and tuberculosis.

Lobar Pneumonia, or Croupous Pneumonia. (Plate XIX.) This inflammatory affection of the lung may be due to one of many microorganisms (single infection), or it may be a mixed infection. For its consideration, the reader is referred to the Infectious Diseases, Chapter XX., Part I.

Chronic Interstitial Pneumonia. Cirrhosis, fibroid phthisis, and chronic interstitial pneumonia are names given to a condition of chronic induration of the lung, caused by interstitial overgrowth of fibrous tissue. Obliteration of the air-vesicles and contraction of the lung result from the overgrowth. The bronchi are frequently dilated, and cavities and gangrene may occur. The disease is rare except as the result of tuberculosis, but it may follow pneumonia and pleurisy, and it is said to be caused by inhalation of fine particles of steel or cotton. *Pneumonokoniosis* is the term, first employed by Zenker, for the chronic interstitial pneumonia from the inhalation of dust.

Physical Signs. (See Plates, Bronchiectasis.) *Inspection.* The disease is unilateral. The chest-wall is *retracted.* The ribs are drawn together, so that the interspaces are obliterated. The shoulder is drawn over the sunken thorax. The spinal column is curved. The heart is displaced. It is drawn toward the affected side. If the right lung is the seat of disease, an impulse is seen to the right of the sternum; if the left, the præcordial area of impulse is increased and extends upward. There is no expansion whatever (immobility) of the affected apex or base. The healthy lung is the seat of compensatory emphysema. (See Fig. 147.)

PLATE XIX.

FIG. 1.

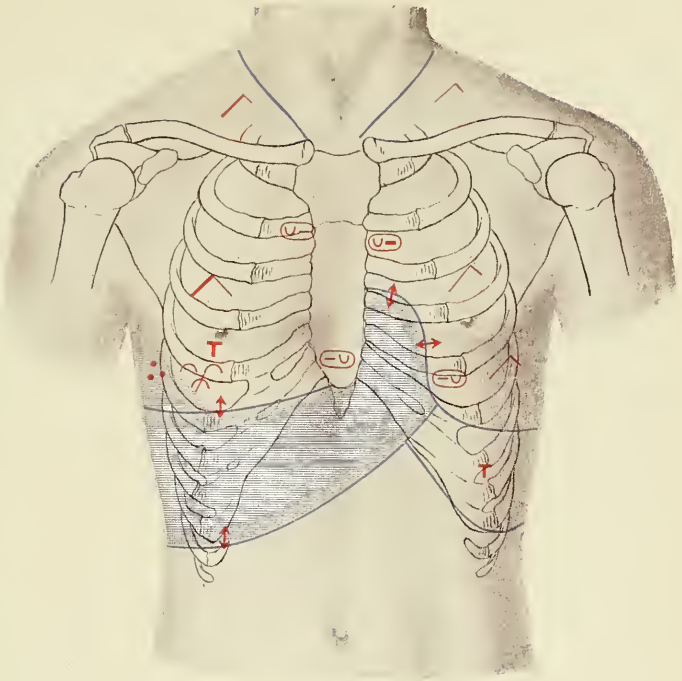


FIG. 2.

Lobar Pneumonia.

Consolidation of the right lower lobe. Transmitted bronchial breathing and signs of bronchitis over the left lung posteriorly.

FIG. 147.

Fibroid (tuberculous) phthisis; right apex. Heart displaced as indicated by oval.

Palpation. Inspection is confirmed. Fremitus is increased, especially at the apex. At the base, pleural thickening lessens the fremitus.

Percussion. The physical signs show increased density of lung tissue, with dulness on percussion, or, over a dilated bronchus, a tympanitic or amphoric note.

Auscultation. The respiratory murmur is bronchial, or, over a dilated bronchus, has a hollow sound. At the base breath-sounds are feeble, distant, or absent. Râles are also heard.

The disease runs a very chronic course, attended by cough, and mucopurulent and sometimes bloody expectoration, even hemorrhage; but there is no fever and not much loss of flesh. Dyspnœa occurs on ascending heights only. Dilatation of the right heart is likely to ensue, with cardiac murmurs and increased lateral dulness and increase of dyspnœa. Death is hastened by the disease, and is often brought on by acute pneumonia.

In *pneumonokoniosis* (also known as *anthracosis*, coal-miner's disease; *siderosis*, from metallic dust; *chalicosis*, from mineral dust, as in stone-cutter's phthisis) there is a history of exposure to the irritating particles for a considerable period, during which time cough

develops, gradually increases, and the general health fails. Emphysema simultaneously arises, causing dyspnœa. The patients wheeze, cough in paroxysms, and expectorate sputum which contains the dust-particles. In anthracosis it is black. On microscopical examination the special dust-particles are often found. The symptoms of emphysema and chronic bronchitis predominate. Tubercular infection may take place late in the disease.

Pulmonary Tuberculosis. For convenience of diagnosis the specific inflammation of the lungs caused by the bacillus tuberculosis will be considered in this section. If a strict etiological classification were followed, it would be considered among the infectious diseases.

Clinically, we see tuberculosis in the lungs manifesting itself in one of the forms of acute pneumonic phthisis, acute miliary tuberculosis, and chronic ulcerative phthisis. (See Chapter XX., Part I.)

Definition. Tuberculosis of the lungs, pulmonary phthisis, and consumption are names applied to an infectious and mildly contagious disease of the lungs, caused by the tubercle bacillus, appearing in an acute and chronic form, and characterized by cough, fever, sweats, more or less rapid emaciation, purulent expectoration containing elastic fibres, and tubercle bacilli, and by peculiar physical signs.

Acute Pulmonary Tuberculosis, Acute Phthisis, Acute Pneumonic Phthisis, Galloping Consumption, may be primary, or be secondary to a localized area in the lung, causing rapid infection, or to tubercular pleurisy, tubercular peritonitis, or to tuberculosis of some other organ. Its onset is usually marked by cough, fever with or without chills, dyspnœa, and sometimes hæmoptysis. The fever rises to 103° or 104°, and is of a continued type (lobar-pneumonic form), or rapidly assumes a hectic type, accompanied by restlessness and exhausting night-sweats, anorexia, and rapid emaciation. Prostration is extreme, but the mind is at first clear and the spirits cheerful. Cough increases, the expectoration, at first mucoid and scanty, but often tinged with blood, becomes more copious and mucopurulent. The bowels may be loosened or constipated. The urine may show the diazo-reaction.

When death takes place without more decided pulmonary symptoms the tuberculosis has been secondary to tuberculosis elsewhere, or death is the result of a general miliary tuberculosis.

When the acute pulmonary tuberculosis is primary, the character of the disease is soon made clear by the early development of consolidation of the lungs, usually of an apex first, rapidly followed by softening and the formation of cavities. The sputum becomes mucopurulent, is frequently streaked with blood, and pure blood is often coughed up. The sputum contains yellow elastic tissue and abundant tubercle bacilli. The patient often presents a cachectic appearance; emaciation has been very rapid, and has reached an extreme degree; there is frequently a red flush about the cheek-bones, which, with the bright eyes, contrasts strongly with the hollow cheeks and temples, and the white wasted hands and clubbed fingers with bluish nails.

The patient's mental attitude is often peculiarly and characteristically hopeful. He expresses himself as better each day, though he is

PLATE XX.

FIG. 1.—Anterior Aspect.

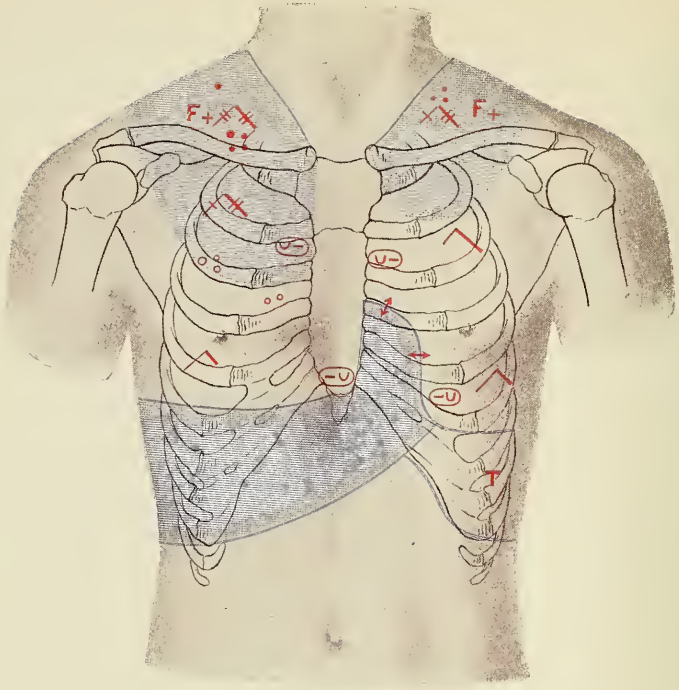
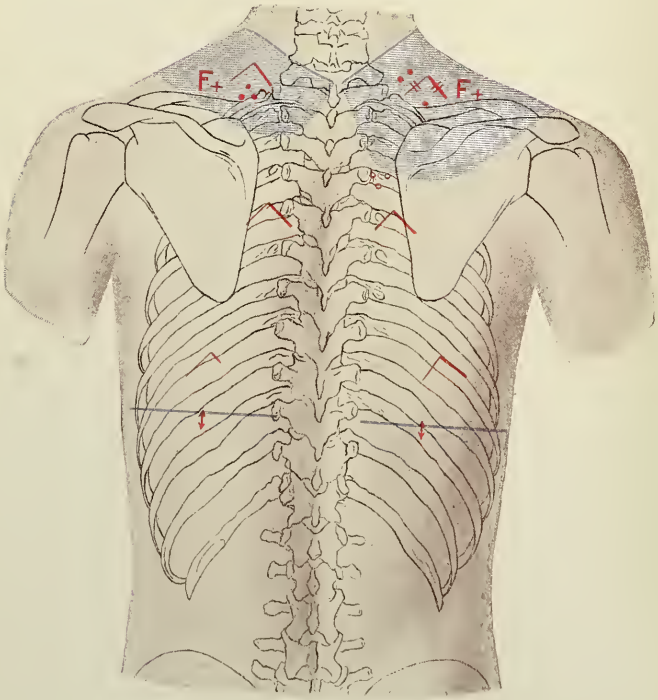


FIG. 2.—Posterior Aspect.



Acute Pulmonary Tuberculosis.

Consolidation of the entire right upper lobe and of the left apex.

occasionally subject to despondency, and is sure that if he could only gain a little strength he would soon be well.

Sometimes, especially in children, the disease is latent. The patient suffers from weariness, the cheeks flush easily, the pulse is readily disturbed, there are nocturnal fever and occasional sweats. Emaciation proceeds very gradually, and a long time may elapse before any disease is demonstrable.

In a few cases the cerebral symptoms are so pronounced as to mask the pulmonary, and in other cases there is actual coincident involvement of the cerebral meninges.

The *physical signs* (Plate XX.) are those of consolidation, often without conjoint pleurisy. The apex is usually first invaded. There are diminished movement, increased fremitus, and dulness on percussion. At first the breathing is bronchovesicular. It rapidly becomes bronchial. At first small moist râles are detected. Later they become large and gurgling. A pleural friction may be heard. It may be first heard above the spine of the scapula behind, above the clavicle in front, or high up in the axilla. The upper lobe of the right lung may be affected first, or the anterior portion of the middle lobe. The physical signs may be observed first in the axillary region of either side. The consolidation extends to the remainder of the lung, being preceded by physical signs indicating gradual encroachment upon the air-containing structure. The respiratory murmur is harsh, but soon becomes bronchovesicular and then bronchial. (*Lobar-pneumonic form.*) As consolidation progresses in the middle and lower portions of the affected lung, signs of cavity or multiple cavities appear in the upper. (The whole of a lobe may be the seat of small cavities filled with mucopurulent or purulent fluid.) Cavernous breathing and pectoriloquy, or the bronchial sniff of consolidation, become more pronounced. The dull note of consolidation is relieved by a dull tympanitic or full tympanitic note. Now moist râles of all degrees are heard. (*Bronchopneumonic form.*) Above they are gurgling; below, small and large moist râles. If the progress is not too rapid throughout the lung first affected, signs of invasion are found in the remaining lung, usually at a point corresponding to the primary focus in the original lung. The apex, therefore, is first invaded in most cases. Infection of the second may begin earlier than the signs in the first lung would lead one to anticipate. The rapid invasion of one lung compels compensatory emphysema of the other. The increased movement, with harsh or puerile breathing, without change in fremitus or in pitch and tone on percussion, masks any small consolidations.

The expectoration becomes more purulent as the disease progresses, and may be blood-tinged. It is copious and possesses some fetor. It is found to swarm with bacilli and to contain yellow elastic tissue. Hemorrhage may take place. The general symptoms become more alarming. The fever becomes of a hectic type. The patient rapidly emaciates. Cyanosis is shown in the dusky countenance and blue finger-tips. The exhaustion becomes extreme. Pallor, with flushed cheeks and an anxious countenance, is seen. The sweats are profuse. The appetite is lost. Diarrhœa may set in. Remissions may take

place, even in acute cases; for a time the fever and more aggravated pulmonary symptoms are in abeyance. The typhoid state ensues in some cases. Death takes place from exhaustion and heart-clot or from meningeal tuberculosis. The duration is from two to six weeks.

Diagnosis. In the earliest stages, before the invasion of new territory is pronounced, the cases are involved in doubt. It may be confounded with pneumonia until the sputum is secured and bacilli are found.

In pneumonia we have the pronounced rigor, the rapid rise of temperature, the altered pulse-respiration ratio, the hot, dry skin, the sticky, viscid sputum, containing the pneumococcus, the peculiar changes in the urine, leucocytosis, the occurrence of herpes, the termination by crisis, to point to the nature of the process. Emaciation is not marked; there are no such profuse sweats as the repeated drenchings we see in pneumonic phthisis; anæmia is not so pronounced. Then cavity-formation does not take place, or at least rarely. In pneumonia the fever is of a continued type; in phthisis it is often intermittent or remittent. The sputum is more purulent in acute pneumonic phthisis. Finally, the history of exposure to infection, the primary occurrence of tuberculosis elsewhere, the secondary occurrence of tuberculosis in other organs after the lung-invasion, the longer duration—aid in determining the true affection. Inoculation of animals may be resorted to in doubtful cases.

Acute miliary tuberculosis (pulmonary type) is attended by high fever, rapid emaciation, hurried breathing, rapid pulse, duskiness of face and extremities, more or less stupor, delirium, and the development of the typhoid state, with prostration and the occurrence of profuse sweats. Intestinal symptoms, as flatulency and distention, may be pronounced, and diarrhœa may form a prominent feature. *Physical signs* are negative or are those of bronchitis. There is resonance or hyper-resonance on percussion. The latter is not uncommon. The onset is abrupt or may follow a period of malaise. In some instances the tuberculous process is more advanced in some situations than in others, giving rise to special local symptoms. Thus, recently, a patient was admitted to the Presbyterian Hospital with stupor and moderate delirium. He had fever, rapid pulse and breathing, and a peculiar dry, harsh skin. There were albuminuria, casts and blood in the urine, and it was thought he had uræmia. The temperature-range was irregularly intermittent. The diagnosis was established later because of the development of undoubted secondary tuberculosis in other organs. At the autopsy general tuberculosis was found, with primary tuberculous ulceration in the bladder, the ureters, and renal pelves.

Diagnosis. Hurried breathing and cyanosis are distinctive features, out of all proportion to the physical signs, and, on this account, of diagnostic significance. It must be distinguished from typhoid fever, septicæmia or pyæmia, and malignant endocarditis. It is distinguished from typhoid fever by the absence of successive stages in the course of the disease; in typhoid fever the evolution of the disease is more characteristic than its symptoms. The headache of the first

week finally disappearing, is noteworthy. The special range of temperature, the onset, the fastigium, and the defervescence at definite periods in the evolution of the disease, are of diagnostic value. Cyanosis is more constant and marked in tuberculosis. The skin and capillaries have more tone in typhoid fever than in tuberculosis, at least in the first two weeks. Hyperæmia follows irritation in typhoid; pallor, with duskiness, in tuberculosis. The eruption, with its specific mode of development, belongs to typhoid fever alone. The stools, the enlarged spleen, the vascular tone are suggestive of typhoid fever. The spleen enlarges earlier in the disease in typhoid fever. Bacteriological examination may be of service. The occurrence of intestinal hemorrhage, pointing as it does to typhoid fever, is a welcome sign in cases in which the diagnosis is obscure. I have never seen it in tuberculosis. In typhoid fever the reflexes (knee-jerk) are never absent; in tuberculosis, if the meninges are involved, they are variable, present one day, absent the next. The diazo-reaction in typhoid is of some service, although it also occurs in tuberculosis. (See Urine.) It does not come on until later than the fifth day in typhoid fever. It disappears at a certain time in the involution of typhoid; it continues indefinitely in tuberculosis. (See Chapter XIX., Part I.)

The distinction of tuberculosis from septicæmia or pyæmia and malignant endocarditis is often difficult. We must search for local areas of septic or pyæmic infection. The ears, the teeth, the bones, the veins, the heart, the pelvic organs in females, the rectum, the genito-urinary tract—must be carefully examined. Hemorrhagic infarcts, or metastatic abscesses, may be found which point to the original conditions. The eye-ground may show hemorrhages. The skin and mucous membranes may exhibit minute capillary hemorrhages or infarcts. They are the size of a pin-head, do not disappear on pressure, and are not elevated. The spleen is more likely to be enlarged in the septic affections. The respirations are not so rapid as in tuberculosis. Cyanosis is a distinctive feature of tuberculosis. The physical signs of endocarditis may be determined, and subsequently embolism or thrombosis prove the nature of the process.

Chronic Tuberculosis, Chronic Ulcerative Phthisis. Chronic tuberculosis or phthisis is much more common than acute tuberculosis, from which it is distinguished by its slow progress and by periods of remission, during which the disease may be arrested temporarily or permanently.

It may begin in a variety of ways. The most common mode of origin is in an ordinary bronchitis with which pleurisy is occasionally associated. Previous to this the patient may have been in good health, but generally the health has been impaired for some time. The bronchitis may be simple or part of influenza, measles, whooping-cough, or some other specific disease.

The bronchitis usually proves obstinate, and by and by there is found at the apex of the lung a small area over which, on percussion, there is increased resistance, with slight impairment of resonance, as compared with the other side; the respiratory murmur is broncho-vesicular, sometimes jerky in rhythm, and the vocal resonance and

fremitus slightly increased or unaltered. Such physical signs are met with more frequently at the right apex than at the left, and oftener in the suprascapular fossa than anteriorly. The next most frequent seat is probably between the clavicle and second rib anteriorly.

The patient will be found to have lost strength, and usually some weight. There is often a slight evening rise of temperature, and occasionally nocturnal perspirations. The appetite is impaired, and anorexia may exist. Cough is rarely absent, especially during the night or on waking in the morning; it may, however, be so slight as apparently to have escaped the notice of the patient. When characteristic it is dry and hacking. Expectoration is scanty and mucoid, but occasionally it may be tinged with blood. It should be remembered that children and old persons sometimes do not expectorate, and that, as a rule, women are more inclined to suppress expectoration than men. No tubercle bacilli may be found in the sputum after repeated examination; but if examinations are continued, they will appear sooner or later.

Instead of developing after a bronchitis, as we have just described, it may set in suddenly under the guise of a pneumonia, more frequently of the catarrhal form. The symptoms and physical signs do not differ essentially from those of pneumonia, except that the expectoration is more likely to be profuse, mucopurulent, and blood-streaked, and bacilli are found in it; the fever is more hectic in type, and night-sweats are common. The consolidation is found at the apex. After the patient convalesces from such an attack he continues weak, does not gain flesh readily, still has a cough with expectoration, evening fever with occasional night-sweats, and an area of consolidation usually at an apex of the lung. Over this area, in addition to the usual signs of consolidation (bronchial or feeble breathing, dulness, etc.), moist or dry subcrepitant râles are heard.

In some cases fever, emaciation, and weakness progress for some time before pulmonary symptoms arise.

In still other cases the invasion of the disease is by sudden hæmoptysis, which is oftener copious than not. Several such hemorrhages may occur in rapid succession, or there may be only one. Moreover, its disappearance may not be followed, or at least not immediately, by any further pulmonary symptoms or physical signs; more commonly, however, it is followed by fever, cough, expectoration, and physical signs of incipient consolidation, usually at the apex.

In still other, but rarer cases, the pulmonary disease is latent, being marked by gastric or peritoneal symptoms, or by a general anæmia.

By whatever path invasion comes, the physician should be on the lookout for it, especially in a young adult predisposed by heredity or environment to tuberculosis. The recognition of the disease in its early stage requires the greatest skill, which in turn is recompensed with the highest reward, since the disease is then curable.

The further progress of a case of tuberculosis of the lungs, after consolidation has once become manifest, is very variable. It may be arrested at this point permanently, cure resulting from cicatrization. More frequently there is temporary arrest of the process; fever lessens

PLATE XXI.

FIG. 1.—Anterior Aspect.

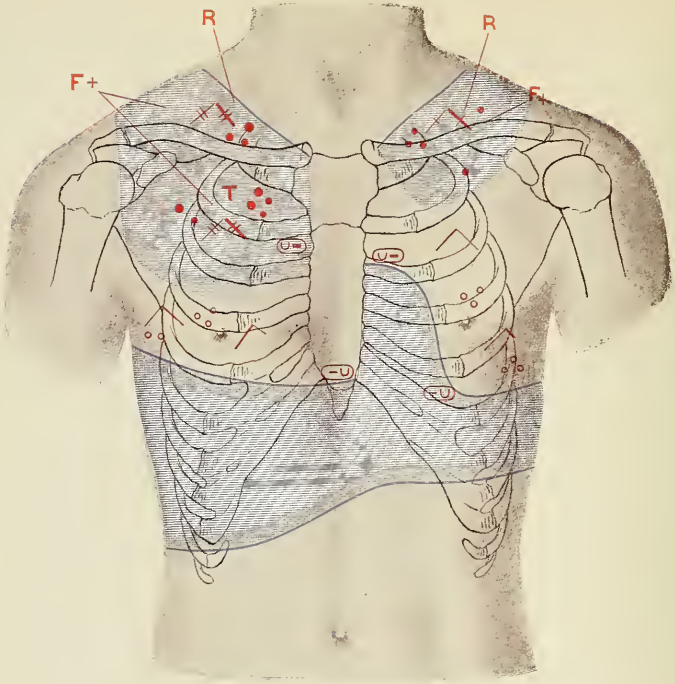
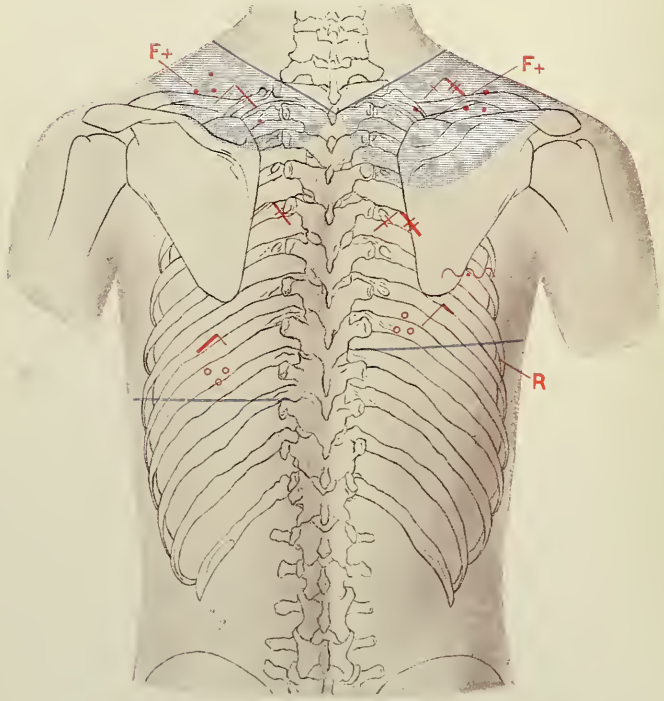


FIG. 2.—Posterior Aspect.



Chronic Pulmonary Tuberculosis.

Consolidation with cavity formation. Chronic pleurisy with loss of respiratory movement of lung margins. Retraction.

or ceases entirely, the pulse resumes its normal rate, appetite improves, and there is a gain in flesh and strength. Cough and expectoration are more likely to persist than the other symptoms, but with the other improvement they diminish in frequency and copiousness. There are fewer râles, but the signs of consolidation are still present, though there is no further extension of the process. Often, after a cavity has been found, the disease is arrested, or progresses very slowly.

After a longer or shorter time, as the result of reinfection from the old focus excited by acute bronchitis or by some depressing influence, the tuberculosis is relighted, so to speak, and runs much the same course, the lung being left more diseased and the general health worse after every such attack. Nevertheless, there may be long intervals between such attacks, the patient in the meantime continuing in fair health. Thus the disease may linger or recur for years, the patient not ill enough to be confined to the house, and not well enough to stand hard work or great exposure. Slowly, by ulceration and suppuration, the lung-tissue is wasted and cavities are formed. Before there are large cavities at an apex the base of the same lung becomes consolidated by the production of tuberculous material, and before one lung is extensively diseased the apex of the opposite lung is attacked, the process being repeated in it if the patient lives long enough. Instead of reinfection from an old focus, new infection may take place, giving rise to the old train of symptoms, or setting up more acute disease. During this time the patient is liable to an attack of acute pneumonia, pleurisy, bronchitis, or general miliary tuberculosis. He is also liable to sudden death by hemorrhage. In a number of cases the intestines and peritoneum become affected, and abdominal pain and diarrhœa are superadded as symptoms.

As a rule, the patient gradually sinks. The later stages are marked by increasing cough and dyspnœa, which are very distressing and prevent sleep. Expectoration is more copious, purulent, and is raised with increasing difficulty.

The appetite is poor and capricious, or anorexia is complete. The heart becomes more and more feeble, the fever is hectic and accompanied by exhausting night-sweats, the feet and limbs swell, and acute cramp-like pains are felt in the legs, probably caused by thrombosis of the veins.

Emaciation is extreme, scarcely anything but skin and bone being left. Death occurs from perforation of an intestinal or gastric ulcer, from hemorrhage, or more commonly from exhaustion, and from asphyxia caused by œdema of the lungs.

The *physical signs* (Plate XXI.) depend upon the lesions. It is often possible to detect all stages of the tubercular process, from early consolidation to large cavity, in the same patient. The signs of consolidation have been sufficiently dwelt upon. When softening begins, the percussion-note continues dull and the breathing bronchial; but it is often difficult to make out the quality of the breath-sounds because they are feeble and obscured by numerous moist crackling râles and moist subcrepitant râles from disintegration of lung-tissue and bronchitis. After the patient has coughed several times and expectorated, and then takes

a long breath, the quality of the breathing becomes perceptible. As the lung-tissue is further softened and removed by expectoration cavities are formed. These, if large enough and superficial, give a tympanitic note on percussion, and, if there is communication with a bronchus, a cracked-pot sound. The breath-sounds are hollow and the râles are bubbling and gurgling, or large and mucous.

The normal vocal resonance is replaced by bronchophony and pectoriloquy. Tactile fremitus may or may not be increased. (See Cavities.)

But if the walls of the cavity are thick from indurated tissue, the percussion-note will be dull and the breathing bronchial. If the tissue composing the wall is less thick and dense, percussion produces a wooden sort of resonance. If much normal lung-tissue intervenes, the percussion-note will be clear.

As tuberculosis of the lungs progresses, the clavicles and ribs become more and more prominent from the loss of fat, and local flattening of the chest, with impaired expansion, marks the seat of the disease.

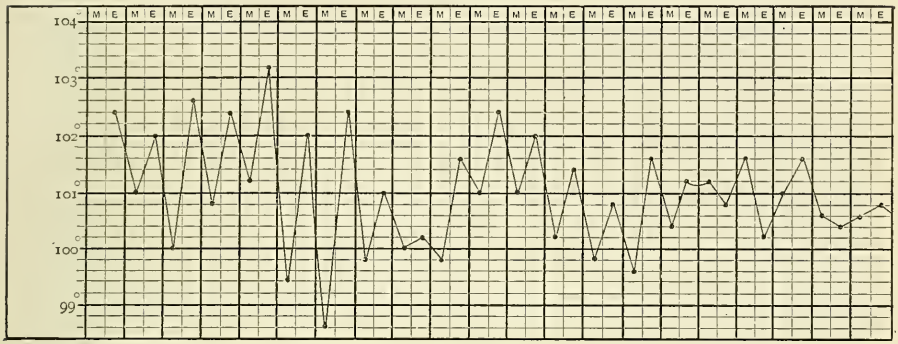
The Diagnostic Features. The striking phenomena of tuberculosis which are considered in the diagnosis are emaciation, anæmia, fever, cough, dyspnœa, chest-pain, hemorrhage, the expectoration, and the objective symptoms. Of less diagnostic value, but important as collateral data, are the aspect, the occurrence of vomiting and diarrhœa, and of symptoms of secondary tuberculosis in other organs. Age and occupation may, to a certain extent, aid in the diagnosis.

Emaciation. This is always seen, even in acute forms of tuberculosis. It is rapid in the acute, slow and progressive in the chronic forms. In the latter there may be a temporary improvement in this respect. It must not be confounded with muscular atrophy, and the emaciation of carcinoma, diabetes, anorexia nervosa, and other exhausting diseases. *Anæmia* is always pronounced. It may be associated with leucocytosis if there is cavity formation. The reduction of red cells and diminution of hæmoglobin are marked. *Fever.* This symptom is always present. The temperature should be taken every two hours for a time, to determine accurately the degree and course. It may be intermitting, remitting, or continuous. It may be intermitting in some acute forms, the morning fall reaching, or going below, normal. The difference between morning and evening temperature may not be more than a degree. In the acute form it is high and continuous, and soon may be attended by the typhoid state. In the more chronic cases it may be intermittent at first, then continuous, and finally intermittent again. In the later stages the intermitting fever is due to a mixed infection, or sapræmia, from the purulent contents (staphylococcus and streptococcus infection) of the lung cavities.[1] (See Fig. 148 and Fig. 149). The intermittent fever of the early stages has frequently been mistaken for malaria. (See Fever.) The occurrence of fever in a

[1] Leyden has pointed out that intermitting fever is part of the tuberculous process, and not a streptococcus or staphylococcus infection, as formerly held, because pus microorganisms are not found in the purulent contents of cavities, and because in other forms of tuberculosis, as empyema or joint-disease, they are notably absent, and yet such form of fever exists.—*Deutsche medicin. Wochenschrift*, Sept. 14, 1894.

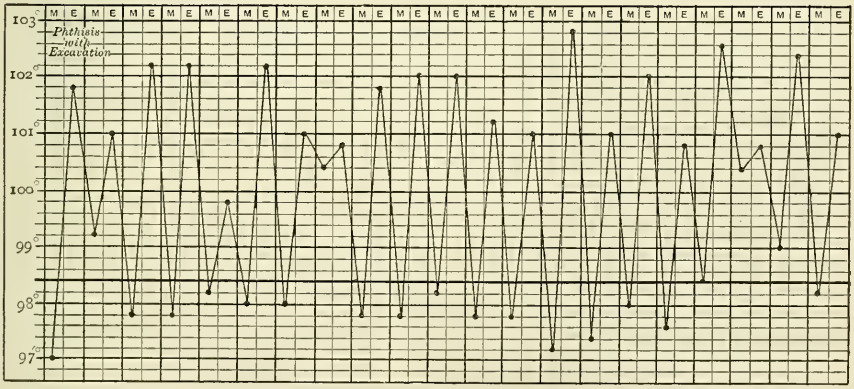
patient who has been losing flesh, and is otherwise in poor health, excludes cancer and diabetes and other afebrile causes, and points strongly to tuberculosis. It must not be forgotten that in chronic tuberculosis in the aged the temperature may not rise above 100°; often, indeed, it is subnormal.

FIG. 148.



Continued fever of tuberculosis.

FIG. 149.



Intermitting fever of tuberculosis.

We must consider, therefore, that fever, the cause of which is not obvious, may be due to tuberculosis; and that if, when such probable causal conditions as gastro-intestinal catarrh or infectious disorders (malaria) and suppurations are eliminated, the fever still persists, then the fever is probably of tuberculous origin.

Sweats. Frequent sweating may be the first symptom complained of by the patient. It may occur with the tripod of symptoms of the intermitting febrile range—chill, fever, and sweat. It would be likely to occur at night under these circumstances. It may occur at any time, however. "Night-sweats" are alarming to the mind of the

laity, and are really of diagnostic significance. The perspiration awakens the patient at night because it is so profuse. It may be only moderate, not rousing the patient until morning. It may be general or local. Local sweats are confined to the head and neck. *Anæmia.* This quite rapidly becomes marked. It is recognized by the color of the surface and by an examination of the blood. When collateral inflammation is present, leucocytosis is seen. *Cough.* Cough is one of the earliest symptoms. It may be the only symptom for some time. It is often dry and hacking at first and may continue so for a long time. Later it is accompanied by mucoid and then mucopurulent sputa, which contain the characteristic elements. (See Sputum.) *Dyspnœa* is almost always present. The degree varies with the association of fever. When the latter is present dyspnœa is more pronounced. It is more pronounced in acute cases. In miliary tuberculosis the frequency of respirations that attends the dyspnœa is out of all proportion to the physical signs. In this form cyanosis is more marked. In chronic localized phthisis the dyspnœa may only occur on exertion, after eating, or upon excitement. The bloodless lips may have a constant bluish hue. The fingers are dusky and become "clubbed." In the later stages the dyspnœa is constant and in proportion to the extent of involvement of the lungs and the degree of fever. Although of diagnostic significance only when associated with other symptoms, it is most distressing, and is the cause of constant demand for relief.

Chest-pain. This is due to localized pleurisy or to myalgia. The latter may be seated in muscles strained by coughing. Pleuritic pains may occur in any situation, and vary in position from time to time. They may be due to extensive inflammation or to tuberculous pleurisy. Constantly recurring and unilateral chest-pains, with or without signs of pleurisy, with cough and emaciation, are significant of the disorder under consideration. (See Pain.)

Hemorrhage. This symptom is alarming, and, in the large majority of cases, is due to pulmonary tuberculosis. It may mark the onset of the acute disease, and continue irregularly throughout its course or recur several times before the advent of more common symptoms of the chronic form. It may occur at intervals of a few months or a year, before emaciation, cough, and characteristic expectoration set in, or before bacilli are found in the sputum. Each attack is attended by fever, usually, and followed by anæmia and prostration. If hemorrhage of the lungs (see Symptoms) occurs in a young adult without cause (as aneurism or cardiac disease, etc.), it must be looked upon with suspicion. The likelihood of tuberculosis is increased if the bleeding occurs in a patient of tuberculous aspect in whom a family history of tuberculosis is found, and who has been exposed to infection. In the aged it may occur from a localized area of disease. Hemorrhage is also common in the late stages of tuberculosis. It is not at this period of diagnostic value as to the primary cause. It is usually due to the erosion of an artery in a cavity. Hemorrhage also occurs in tuberculosis during the quiescent period. The progress of the disease is arrested. The discharge of blood is accompanied by the expectoration of pulmonoliths, calculi formed by the degeneration of caseous areas.

Vomiting (see Gastro-intestinal Disease) is a symptom which is often present in the early stages of tuberculosis of the lungs, and frequently masks the true condition. The vomiting may lead to the belief that a local gastric catarrh or diarrhœa is to blame for the general symptoms. The occurrence of fever with the gastric symptoms should lead to an examination of the lungs.

The occurrence of diarrhœa and symptoms of tuberculosis in other organs may thoroughly establish the diagnosis in tuberculosis of the lungs with otherwise obscure pulmonary symptoms. The intestinal discharges may contain tubercle bacilli, or they may be found in the urine, in joint-suppuration or glandular enlargement.

The Sputum (*q. v.*). The diagnosis is absolute when tubercle bacilli are found in the expectoration. Nummular sputa are more common in phthisical excavation. The sputum is discharged in tough coin-shaped masses, which sink when expectorated into a vessel containing water. Fragments of lung-tissue (yellow elastic) point to tuberculosis, but are possible under other circumstances.

The Physical Signs. The *aspect* of the patient is always suggestive, and is an aid to the recognition of the condition. The tuberculous or phthisical chest, the long neck and arms, the pale face, the occasional hectic flush, the clubbed fingers, the emaciation of the many subjects we see in our infirmaries, fix in our minds a composite picture the recognition of which goes far to diagnosticate the insidious disease.

The objective signs point to an invasion of air-containing structure by solid material, with collapse of lobules, leading to consolidation, followed by cavity-formation, and in both stages by the occurrence of pleurisy. Local contraction (flattening) and impaired movement at an apex, with inspiratory depression above the clavicles, with suppressed breath-sounds and prolonged expiration, with impaired resonance, are the earliest signs of tuberculosis. In the chronic cases, contraction, impaired movement, dulness and increased resistance from thickened pleura may override the signs of consolidation. No one physical sign is of diagnostic significance. The combination of signs, and the orderly procession by which they advance as the physical conditions progress, are the most diagnostic.

The Size of the Lung. In the diagnosis of pulmonary tuberculosis the physical examination must be directed to a determination of the size of the lung, and of the extent of its expansion, by which we judge of the amount of air entering the lung, as well as to the presence of consolidation.

The tuberculosis process is associated with diminution in the bulk of the lung usually. We can estimate the size and the degree of expansion by inspection, palpation, and percussion. The so-called diaphragm-phenomena is studied and the X-rays employed. Any diminution in the excursion in the shadow of the diaphragm is evidence of diminished bulk of the lung or of diminished expansion. By palpation, with mensuration, measurements are taken. By percussion we estimate the lung boundaries. The degree of expansion can be determined by securing the limits of liver dulness and cardiac and splenic dulness in ordinary breathing, and then at the end of full inspiration

and expiration. Valuable information is thus secured. Of course, employing inspection and palpation the two sides of the lung must be compared. Percussion enables one to determine fairly early the presence of consolidation. In thin subjects the change in the note is more readily elicited than in fat or muscular subjects.

On auscultation in the early stage of tuberculosis roughness of respiratory murmur with prolonged expiration, feeble respiratory murmur, and jerking or cog-wheel respiration are common signs. These signs change gradually into bronchovesicular and then bronchial types of breathing. Crackling râles or clicking sounds and consonating râles attending these modifications of breath-sounds are of the greatest diagnostic importance. They must be brought out frequently by cough and then full inspiration.

The Site of the Lesion. The situation of the *physical signs* is diagnostic. Percussion should be directed especially over those parts of the lung in which an infection is liable to occur, as the clavicular and subclavicular spaces, the anterior border of the upper lobe, the tongue-like part of the left upper lobe, which overlaps the heart, the supraspinous space, the upper interscapular region, and the upper borders of the lower lobes posteriorly. The latter is best secured by having the patient place the hand of the arm of the side percussed on the shoulder of the opposite side. The scapula is thus removed from the surface of the lung to be examined.

It is necessary also to consider carefully the general conditions. We inquire the age, adolescence and early adult life being the common periods in which pulmonary tuberculosis develops. The occupation,[1] the history of exposure to the disease, the history of predisposition to tuberculosis in the family, the history of previous, now arrested, tuberculosis, as in joint-disease, or glandular tuberculosis (scrofula), are data deserving special consideration, as they may furnish corroborative evidence of the presence of the disease.

Diagnosis. The presence of tuberculosis is presumed upon in a patient with pulmonary symptoms—as a hereditary predisposition, abnormalities in the form of the chest and imperfect development, or hypoplasia of the circulatory organs. If the patient is under weight and has a poor appetite, and at the same time is undergoing unusual strain or anxiety, the possibility of tuberculosis is increased. Often, before the physical signs of tuberculosis can be established, the shrewd physician will fear recurrence of tuberculosis if there are signs of anæmia, progressive loss of weight, slight fever, disturbed digestion, a frequent pulse, and persistent and localized bronchial catarrh. The examination of the lungs, the examination of the sputa, and the tuberculin test must be employed as soon and as often as practicable. (See Diagnosis of Tuberculosis, Chapter XX., Part I.)

The diagnosis is established by finding tubercle bacilli in the sputum. Their absence, in spite of the most careful search, is against the tuber-

[1] Several undoubted instances are recorded in which hospital residents and young physicians working in laboratories in which tuberculosis is studied, or constantly examining sputum, have been infected in the course of their studies.

culous origin of the disease. (See Diagnosis of Tuberculosis, Chapter XX., Part I.)

In subsequent chapters the differential diagnosis of tuberculosis and other diseases will be pointed out. It must not be forgotten that the disease may set in as the terminal affection in many diseases. Thus, in diabetes, in insanity, in chronic cerebral or spinal disease, and in other affections, tuberculosis may develop insidiously, and finally cause death.

It must be distinguished from chronic gastric disorders, and particularly *anorexia nervosa*. It must not be confounded with malaria. It must be distinguished from simple anæmia, the cause of which may be recognized with difficulty. It must be distinguished from chronic bronchitis with bronchiectasis, from pulmonary gangrene and carcinoma. Finally, it must not be mistaken for cancer of the œsophagus and aneurism of the aorta, two divergent conditions which may have pulmonary symptoms simulating phthisis.

Gangrene of the Lung. Gangrene is a rare disease of the lung, and, like abscess, always secondary. It may be produced by any cause which so obstructs the circulation that a portion of the lung dies in bulk. The gangrene may be circumscribed or diffused; it results most frequently from pneumonia, but may be due to injury, to a general septic condition, or to embolism. It is rather frequently met with in the insane, possibly owing to particles of food which have found their way into the lung. Aspiration bronchopneumonia, bronchiectatic and tuberculous cavities, sometimes lead to gangrene. Gangrene in the lung, as elsewhere, occurs in diabetes.

Symptoms. When it occurs in the insane, or is of embolic origin, it may remain latent, and in septicæmia it may be overlooked, on account of the general symptoms. In well-marked cases, however, the symptoms are characteristic. Symptoms and physical signs of pulmonary disease precede the specific symptoms of gangrene. With the onset of a moderate fever hæmoptysis may occur at once or be preceded by the expectoration of a brownish, purulent sputa having a most intense and persistent gangrenous odor. It contains fragments of lung-tissue, altered blood, and putrid débris. (See Sputum.) It separates into the three characteristic layers in a conical glass. The fetor of the breath and the characteristic sputum is diagnostic.

The disease usually occupies the lower or middle lobe of the lung. The *physical signs* are those of cavity. The disease could with difficulty be distinguished from abscess were it not for the characteristic sputum, though in gangrene there is greater tendency to a general septic condition, with profuse sweats and collapse.

Abscess of the Lung. Abscess of the lung may originate in causes outside the lung, or in causes within the lung. To the former class belong those produced by suppurating bronchial glands, abscess of the mediastinum opening into the lung, cancer of the œsophagus with ulceration, and abscess of the liver, suppurating hydatid cyst, or subdiaphragmatic abscess in general, bursting into the lung. Intra-pulmonary causes are tubercle, septic emboli, in which case the abscesses are multiple and subpleural, and pneumonia. In the aspiration form

of lobular pneumonia abscesses occur. Rarer causes are the presence of tumors and obstruction of the bronchi.

Abscess of the lung is therefore always secondary. Its diagnosis depends upon the demonstration of a consolidation in which a cavity subsequently forms, taken in connection with the history pointing to a definite cause. The sputa are copious, purulent, often odorless, sometimes offensive, but always without the fetor of gangrene. They contain elastic fibre, but no bacilli except in tuberculous cases. (See Sputum.) In embolic abscess the signs of pleural friction can only be detected at times. Of course, the constitutional symptoms of suppuration are present.

The Degenerations.

Emphysema. Emphysema consists in an "excessive, permanent, and unnatural distention of the air-cells," or in "extravasation of air into the interlobular or subpleural cellular tissue." (Lænnec.)

Emphysema may be unilateral or bilateral. Local and unilateral forms are usually compensatory. Bilateral emphysema may be hypertrophic or atrophic.

It is more common in men than in women. Its symptoms are more common in childhood and after middle age. Two factors are essential in its causation. First, defective development of the elastic tissue of the lungs. Second, increased intra-alveolar air-pressure. The latter is due to a number of causes. In childhood, no doubt, nasal and naso-pharyngeal obstructions are operative. In adults occupations which necessitate continuous and severe muscular effort, especially if coupled with forced expiration with closed glottis, act as causes. Such occupations are blacksmithing and playing upon wind instruments. Diseases which cause much coughing or respiratory effort, such as chronic bronchitis and whooping-cough, act in the same manner. Chronic mitral valvular disease and the lessened elasticity of the lung-tissue of advancing age both favor congestion of the lung, and thereby predispose to emphysema. The disease is hereditary; several members of a family are affected. It occurs in many in childhood, is in abeyance in adult life, and reappears in old age.

Symptoms. The prominent symptoms in hypertrophic emphysema are *dyspnœa*, *cyanosis*, and *cough*, with expectoration from associated bronchitis. There is no fever. The dyspnœa is in proportion to the degree of emphysema, and is aggravated by the coexistence of bronchitis, asthma, and eccentric hypertrophy of the right ventricle, which are very frequent complications in cases of long standing. When the degree of emphysema is only moderate, dyspnœa is not complained of except upon climbing or walking briskly, or after a hearty meal. But when the degree of emphysema is great, dyspnœa is constant; it interferes with all exertion, frequently necessitates orthopnœa, and prevents continuous speech, so that patients speak in broken sentences or syllables.

Cyanosis is marked. The livid lip is common in the asylums for old men. The face is of a dingy pale color, but becomes bluish on exertion. The extremities are also dusky, and the blueness is general

PLATE XXII.

FIG. 1.—Anterior Aspect.

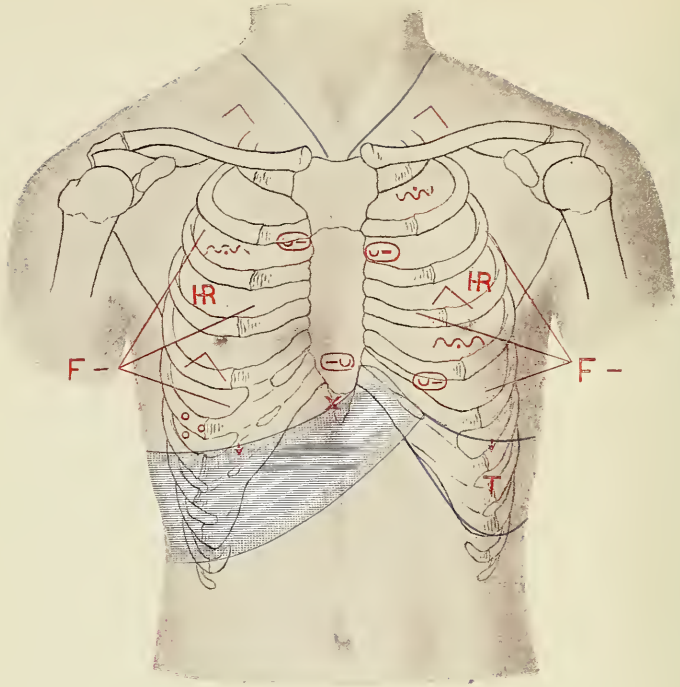
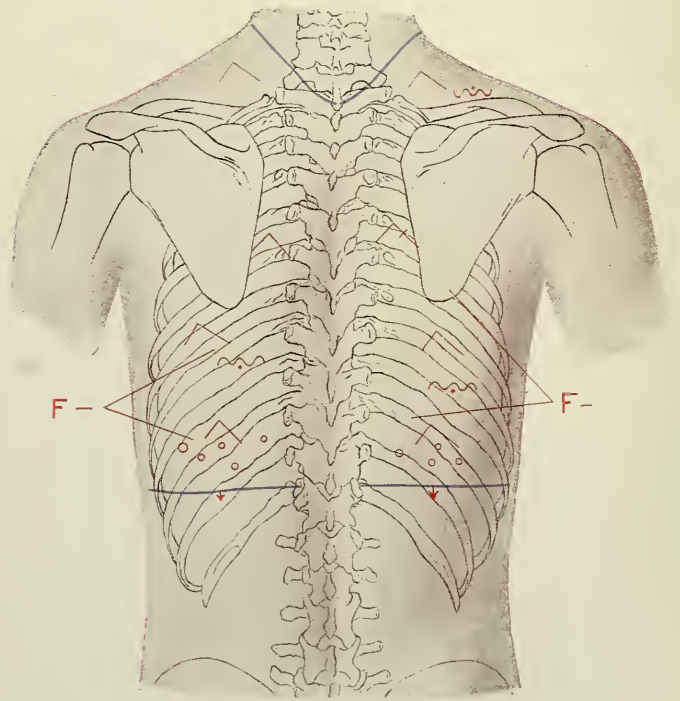


FIG. 2.—Posterior Aspect.



Emphyzema.

Hyperresonance. Enlargement of lungs and diminished respiratory movement of margins. Diminished fremitus. Signs of bronchitis.

in severe cases. This cyanosis, the round shoulders, and the drawn, chronically anxious expression, if I may so term it, make it easy to pick out the emphysematous subjects in a ward of chronic cases.

Respiration is not accelerated, and may be diminished in frequency. It is often accompanied by wheezing when chronic bronchitis coexists.

The cough varies greatly in frequency; it may be altogether absent, since its presence simply indicates an associated bronchitis. This bronchitis may for years be present only in the winter. In children it may be associated with asthma. It may arise on changes of the weather; finally it becomes chronic. The expectoration is that of chronic bronchitis (*q. v.*). It is rarely stained with blood.

Physical Signs. (Plate XXII.). The physical signs of emphysema depend upon its degree and upon whether it is complicated with chronic bronchitis or not.

Inspection: In well-marked cases the chest is barrel-shaped (see under Inspection). There is little movement of the chest in respiration, because the lung is already in a condition of full inspiration (expiratory dyspnœa). *Vocal fremitus* and *resonance* are usually diminished. *Percussion:* The percussion-note is abnormally clear, and may even be tympanitic. Hyper-resonance is typical of the disease. When the distention is extreme the note may be woodeny. The lungs are enlarged. The heart-dulness becomes obliterated by the overlapping lung. The upper margin of the liver falls one or two interspaces below the normal. The resonance extends higher above the clavicles than normal.

On *auscultation* the inspiration is found to be distant and feebler than normal, while the expiration is prolonged, and may become three or four times the length of the inspiration. Grazing or rubbing sounds have been described and attributed to the friction of distended vesicles against the pleura. Other adventitious sounds are due to an associated bronchitis, pleurisy, or tuberculosis. But bronchitis is such a common accompaniment of emphysema that the râles of the former become almost symptomatic of the latter. Their character in emphysema does not differ from that in chronic bronchitis (*q. v.*).

The Heart. The apex-beat is absent. There is epigastric pulsation or systolic shock. The normal area of heart-dulness is encroached upon by the distended lung, and the heart itself is pushed to the right, the apex-beat being frequently at the xiphoid cartilage. If the emphysema attain a very high degree, there may be no perceptible dulness, except on very strong percussion over the cardiac region. The heart-sounds appear feebler and more distant than normal. The right ventricle becomes dilated and hypertrophied, as the result of the pulmonary congestion produced by emphysema. The pulmonary second sound is accentuated. A tricuspid regurgitant murmur may be heard. Venous congestions are common in the later stages. Albuminuria is common. Œdema of the feet and limbs may occur, but general anasarca is rare.

The general health suffers by loss of strength and capacity for physical and mental work, rather than by loss of flesh. The patients are large-chested, stoop-shouldered, and short-breathed, and have an anxious expression of countenance.

Diagnosis. This is based upon the history (heredity, occupation, long duration), the occurrence of dyspnœa and cyanosis, and of winter cough or chronic bronchitis, and upon the physical signs.

Emphysema can be distinguished from *pleural effusion* and from *aneurism*, which may cause dyspnœa, by the universal hyper-resonance on percussion. Pleural effusion, which also causes bulging, is usually unilateral, and the percussion-note is flat. The area of dulness of the heart and aorta is diminished in emphysema.

Pneumothorax, which most resembles emphysema in its physical signs, develops suddenly, affects one side, and has a hollow, tympanitic note on percussion. The succussion-splash, metallic tinkling, and coin-test have no counterpart in emphysema; moreover, the antecedent history and mode of development are different.

Atrophic emphysema is due to the degeneration of age. The lung is reduced in size. The diameters of the chest are lessened. The ribs are oblique. There is atrophy of the chest-muscles. The patients have dyspnœa. There are other signs of senility.

In *interlobular emphysema* the physical signs are the same as those of vesicular emphysema, but it develops suddenly and is liable to be followed by emphysema (intercellular) of the neck, which on palpation gives a peculiar crepitation. The friction-sound and crackling which have been described as occasional adventitious sounds in vesicular emphysema are more commonly heard in the interlobular form.

It is caused by rupture of the air-cells, and hence occurs in diseases in which a great strain is put upon them—especially, therefore, in whooping-cough, but also occasionally in pulmonary hemorrhage and pneumonia; violent coughing and laughing, and great straining, as in child-labor, are capable of producing it.

Bronchiectasis. Dilatation of the bronchi occurs secondarily to affections which tend to weaken the walls of the tubes and to lessen their elasticity. Hence, it is found in chronic bronchitis with emphysema, in chronic phthisis, in catarrhal pneumonia in children, in chronic obstruction from external pressure or foreign bodies. (See Obstructions.) It also occurs when the lungs contract in fibroid pneumonia, or in pleural thickening. It occurs in two principal forms: the *simple*, in which the affected tubes are uniformly dilated; and the *saccular*, in which larger or smaller pouches are formed. It is commoner in males than in females, and probably begins most frequently in adult or middle life. One lung only is affected in about one-half the cases, and when both lungs are affected (chronic bronchitis and emphysema) it is not often to the same degree.

The subjective symptoms consist of cough, expectoration, and a variable amount of dyspnœa. Eventually there may be some loss of flesh and strength.

The *cough* is usually paroxysmal. It may occur only in the morning after the dilated tube fills. It may follow change in position. A paroxysm is followed by copious expectoration, sometimes amounting to a pint and a half in twenty-four hours. It is grayish-brown and mucopurulent, faintly or extremely fetid. The *sputa* contain mucus, pus, casts of the tubules, and various salts. Charcot-Leyden and fatty

PLATE XXIII.

FIG. 1.—Anterior Aspect.

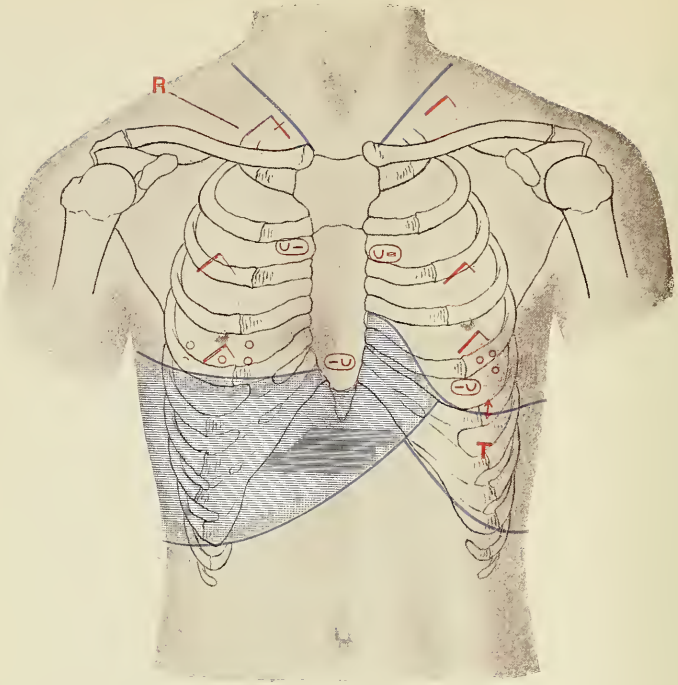
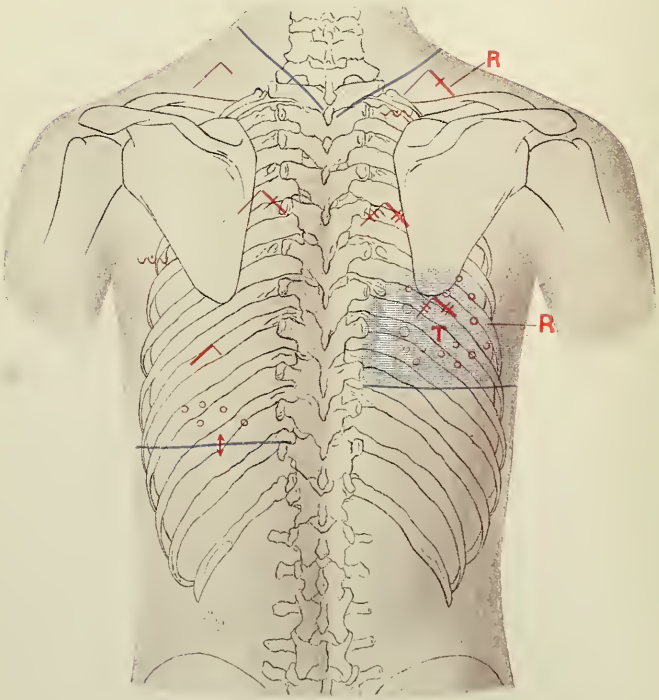


FIG. 2.—Posterior Aspect.



Bronchiectasis.

Chronic pleurisy with induration of the right lower lobe and bronchiectasis. Vicarious emphyzema of the left lung. Bronchitis.

crystals, vibrios, leptothrix, and bacteria (Fox) can be found on microscopical examination. Elastic fibres are found only if the tubes are ulcerated. In a conical glass the sputum separates into three layers—a frothy brown top, a thin mucoid layer in the middle, and a granular layer below. Hemorrhage is rare, but may occur even when tuberculosis is absent.

Dyspnœa is not usually severe, except when the dilatation is complicated by disease of the heart or lungs, or during an acute attack of bronchitis.

Physical Signs. (Plate XXIII.) The physical signs differ according to the extent and variety of the dilatation. In simple dilatation there may be nothing different from the signs found in chronic bronchitis, except a tendency to more bronchial respiration, with râles having a metallic quality. Percussion will vary according to the degree of alteration of the lung-tissue surrounding the affected bronchi, and according to the extent of the dilatation and its proximity to the surface. In the simple forms the percussion-note, if altered, is somewhat less resonant and higher in pitch, whereas in saccular dilatations, favorably situated for percussion, the note is tympanitic if the pouch is empty. On auscultation in simple dilatation the breathing approaches the bronchial, and is accompanied by bronchial râles. In saccular dilatation the sounds are practically those of a cavity, respiration varying from bronchial to amphoric. Vocal resonance and tactile fremitus are usually both increased, but the latter may be diminished.

Diagnosis. The diagnosis of simple dilatation from chronic bronchitis may be impossible, but copious and fetid expectoration indicates the former. The diagnosis of the saccular form from tuberculosis of the lung with cavity is difficult. Wilson Fox says the severe cases are usually associated with consolidation of the lung or with tubercle; but even without the presence of the latter they often present phthisical symptoms—retraction of the chest, with the physical signs of excavation, pains in the side, hæmoptysis, pyrexia, nocturnal perspiration, and diarrhœa—which may all coexist with only an induration of the lung and dilatation of the bronchi. The diagnosis must be made by noting the persistency of the physical signs, which change but little and are not progressive as are those of tuberculosis; the protracted course of the disease; the character of the sputum; and the comparatively slight impairment of the general health.

The Morbid Growths.

Cancer and Other New Growths of the Lung. The new growths may be primary or secondary. The latter are most common. Of primary cancer, the epithelioma is most common; encephaloid and scirrhus come next. Sarcoma is sometimes primary. Secondary new growths succeed disease in the abdominal organs, the genito-urinary tract, the bones, the breast, and the eye.

Symptoms. The general symptoms of malignant growths accompany the thoracic symptoms. Chest-pain, dyspnœa, cough, and a peculiar expectoration belong to the latter. The pain is due to asso-

ciate pleurisy ; the dyspnœa is paroxysmal. (See Dyspnœa from Pressure on Bronchi.) The expectoration is dark, like prune-juice. Signs of intrathoracic pressure are seen. The external thoracic veins are enlarged. The face and arms may be cyanosed, or one arm only may be affected. The heart may be dislocated, the trachea changed in its course ; compression of trachea and bronchus causes dyspnœa.

Physical Signs. In primary cancer the affection is unilateral ; in secondary forms, bilateral. The physical signs are those of pleural effusion or of local consolidation. The consolidation may be massive and not partake of the shape of a lobe. Often signs of effusion and consolidation are combined (enlargement, immobility, absent fremitus, but bronchial breathing). In the secondary forms the disease is bilateral. The signs are mixed. They indicate diminished air in the lung structure. Care must be taken not to overlook the pleural effusion which accompanies the process, the removal of which gives temporary relief. In both forms external lymphatic glands, particularly the cervical, may be enlarged.

Diagnosis. The diagnosis is based upon : (1) The age (after forty) ; (2) the occurrence of emaciation ; (3) the duration of the disease, often rapid, rarely beyond eight months ; (4) the presence of primary disease elsewhere ; (5) the presence of moderate fever ; (6) the signs of intrathoracic pressure ; (7) the involvement of lymphatic glands ; (8) the occurrence of irregular areas of consolidation and of pleural effusion, alone or combined ; (9) the characteristic expectoration ; (10) dyspnœa due to pressure on the bronchus or trachea ; (11) the absence of bacilli from the sputum.

An effusion can often be recognized only after puncture. Hæmothorax is not necessarily present.

Gross Parasites.

Hydatid Disease of the Lungs. The lungs are affected in about 11 per cent. of the cases of hydatid disease. The symptoms, according to Wilson Fox, consist of dyspnœa, pain in the chest, cough, occasional hæmoptysis, and sometimes the expectoration of hydatids, the sputa being otherwise bronchitic, or presenting the characteristics of pneumonia or gangrene when these complications are present. Gradually weakness increases, sometimes with pyrexia, which, when combined with emaciation, may impart to the case a considerable resemblance to phthisis ; pressure-symptoms occasionally occur, and the *physical signs* are either of consolidation of the lung or of pleural effusion, together with certain peculiarities depending on the size and site of the tumor. Graham states that they are more frequent in the right lung and more common at the base, causing marked bulging of the thoracic wall. When the physical signs are those of pleural effusion, localization of the fluid to a definite area takes place, and hence is not related to the shape of the pleural cavity. The breathing may be tubular ; there is condensed lung between the hydatid and the thoracic wall. The symptoms present—cough, dyspnœa, anæmia, emaciation, and clubbing of fingers—too often lead to the diagnosis of phthisis. Hæmoptysis

occurs in many cases. The temperature is normal—an important point in diagnosis. If the cyst ruptures, the sputum is diagnostic. Complications often mask the diagnosis. It must be distinguished from pleurisy, localized empyema, pulmonary abscess, phthisis, actinomycosis, and mediastinal tumors.

Diseases of the Pleura.

The large lymph-structures which cover the lung and line the inside of the thorax are often the seat of disease. It is usually of an inflammatory nature. Hence, pleurisy, or pleuritis, is the most common affection of the pleura. It may be, as to distribution, bilateral or unilateral; as to extent, local or general; as to the nature of the inflammation, plastic, serous, or purulent. The inflammation may be acute or chronic. It is rarely primary. It arises in the course of general disease, or is the result of the extension of inflammation, chiefly of an infectious nature, from neighboring structures.

1. Disease of the ribs or vertebræ, diseases of the mediastinum, of the aorta, œsophagus, and especially of the lung, give rise to various forms of pleurisy, depending upon the nature of the primary affection.

2. Diseases below the diaphragm. Abscess of the liver; perforative inflammation of other viscera adjacent to the diaphragm; abscess of the spleen or pancreas; pus in the pelvis or about the appendix, may give rise to purulent pleurisy by the pus burrowing upward or by infection through the lymph-channels.

3. Disease of the lungs. In the large majority of cases pleurisy in some form occurs in the course of pulmonary disease. In all surface inflammations of the lungs there is associate pleurisy. It is seen in pneumonia, in tuberculosis, in gangrene, and in abscess.

Pleurisy may be simple or purulent. Empyema is always due to infection from the exterior, as the ribs; from the lungs (pneumonia); suppuration below the diaphragm; or to general infective processes, as septicæmia, pyæmia, and tuberculosis.

The general diseases in the course of which pleuritis arises are usually infective, or of such nature as to cause irritating products to circulate in the blood. Of the former, the most common is tuberculosis; the next most common are septicæmia and scarlatina; while to the latter class belong Bright's disease, gout, diabetes, rheumatism, and scurvy. Purulent pleurisy is more common in children than in adults; in males than in females; and more common in tuberculous pleurisy and pyæmia than in rheumatism and Bright's disease.

Acute Pleurisy. Acute pleurisy may be primary, or may be secondary to disease of the lung, or be part of a general infection. Three stages in the morbid processes usually occur, although it may be arrested in the first stage.

Symptoms of the First Stage. Dry Pleurisy. The onset of the disease is usually abrupt, and is marked by *fever*, which may or may not be preceded by chill, and is followed by *pain* in the side, *dyspnœa*, and *cough*. The pain is sharp, stabbing, or tearing in character, and is usually, but not always, referred to the seat of pleurisy. This is

most frequently on a level with the nipple, or a little below this, and more often anteriorly or in the axilla than posteriorly. The pain is caused by the rubbing together of the inflamed surfaces of the pleura, and hence is excited by respiration and cough. For this reason the patient is inclined to restrict the motion of the affected side as much as possible; he does this by leaning over toward that side and by pressing his elbow in against the chest-wall. Pain is usually the first symptom noticed by the patient. The cough is dry and painful. Fever is moderate.

Physical Signs. The physical signs in primary cases are a friction-sound heard on inspiration and expiration. This friction-sound may be a nest of fine, dry, crepitant râles, which are very superficial, and appear to be just under the ear; or a coarse rubbing sound, heard over a larger surface, and resembling a bronchial rhoncus, from which it can be distinguished by its persistence after the patient has coughed. The lungs themselves present nothing abnormal.

If the inflamed surfaces become glued together by plastic lymph, recovery usually occurs very soon, though pain often persists for a long time in lessened degree, and the pleurisy is liable to be re-lighted.

Symptoms of Second Stage, or Stage of Effusion. If *effusion* takes place, the two layers of the pleura become separated; hence, pain and friction-sound cease, and physical exploration shows that a collection of fluid intervenes between the chest-wall and the lung.

The *physical signs* (Plates XXIV. and XXV.) of this stage are (1) enlargement of the affected side, increase in semi-circumference, with fulness of interspaces; (2) diminution of movement; (3) absence of vocal fremitus and resonance; (4) dulness or flatness (deadness) on percussion, with great increase in the resistance to the pleximeter finger; (5) absent or greatly diminished respiratory murmur; (6) displacement of organs.

The dead percussion-note being caused by fluid, it follows that its *upper level* will change with the position of the patient if the fluid is free. If the upper level is at the third interspace when the patient is sitting up, it will fall to the fourth or lower when he is lying down. This change of level cannot be appreciated when the effusion is very large. Moreover, above the line of dulness the percussion-note is *hyper-resonant* or *tympanitic*—Skoda's resonance. Toward the spine on the affected side there may be partial resonance and bronchial breathing, because here the lung is compressed against the vertebræ. In large effusions the tympanitic resonance in the second interspace does not change when the mouth is opened—that is, "Williams' tracheal tone" can often be elicited. The upper limit of dulness in large pleural effusions is higher at the spine and slopes downward, and is lowest in front. This parabolic line is only obtained when the patient is in the erect posture. In moderate effusions the line of dulness is lowest near the spinal column, rises in the middle of the scapula and slopes downward, assuming the shape of the letter S as it passes toward the front (Garland). The patient should take deep breaths before the percussion is performed. At the left base in front the semilunar space is

PLATE XXIV.

FIG. 1.—Anterior Aspect.

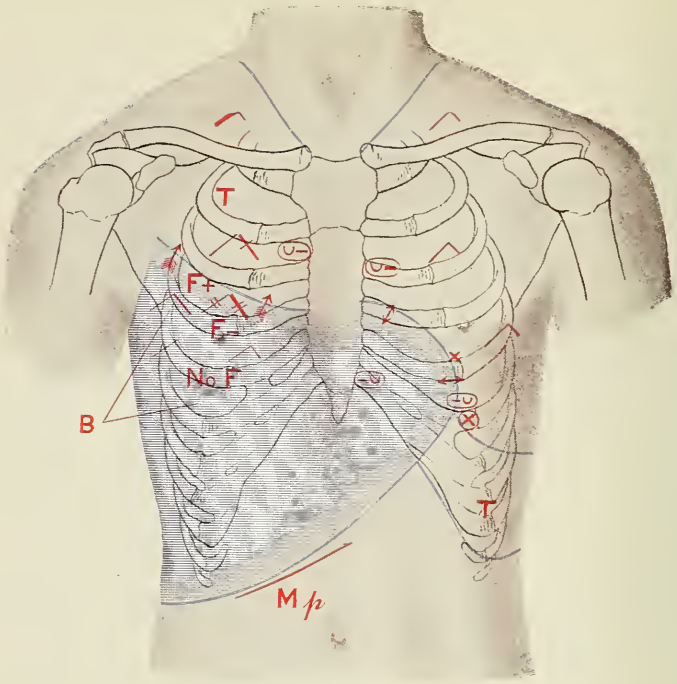
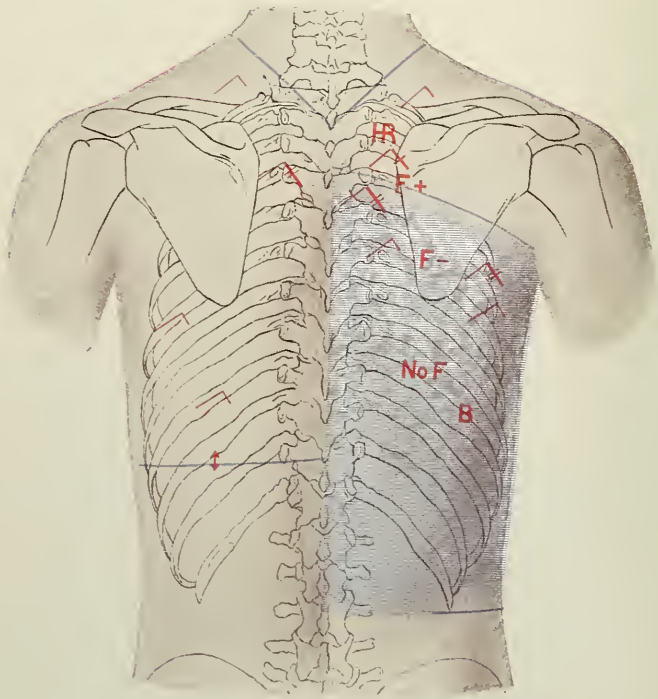


FIG. 2.—Posterior Aspect.



Pleurisy with Effusion (right-sided).

PLATE XXV.

FIG. 1.—Anterior Aspect.

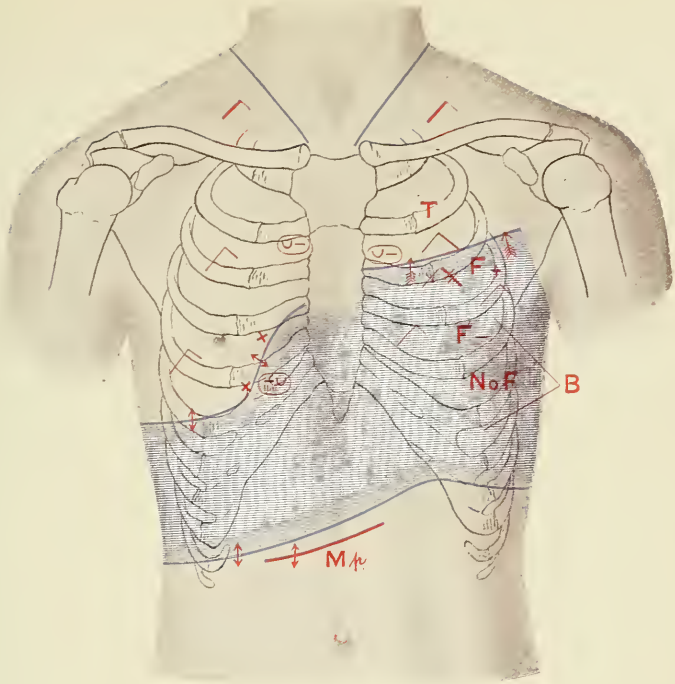
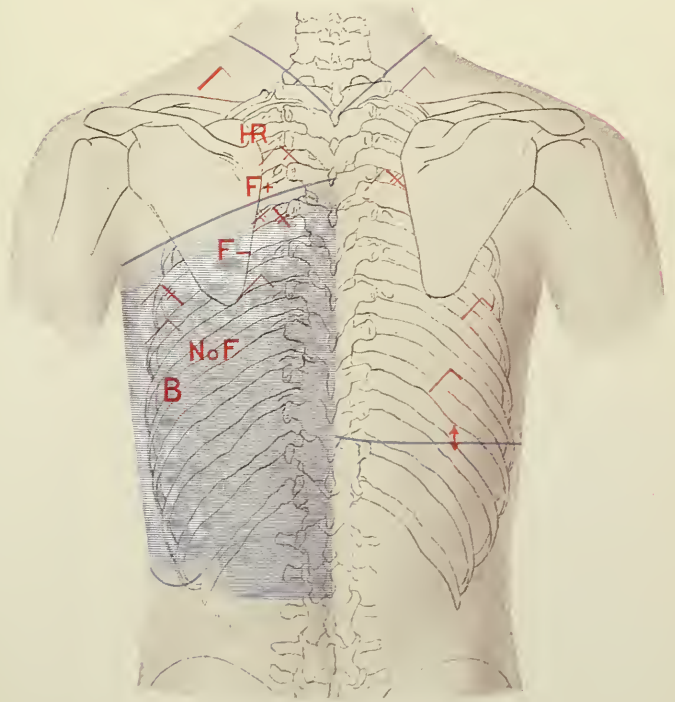


FIG. 2.—Posterior Aspect.



Pleurisy with Effusion (left-sided).

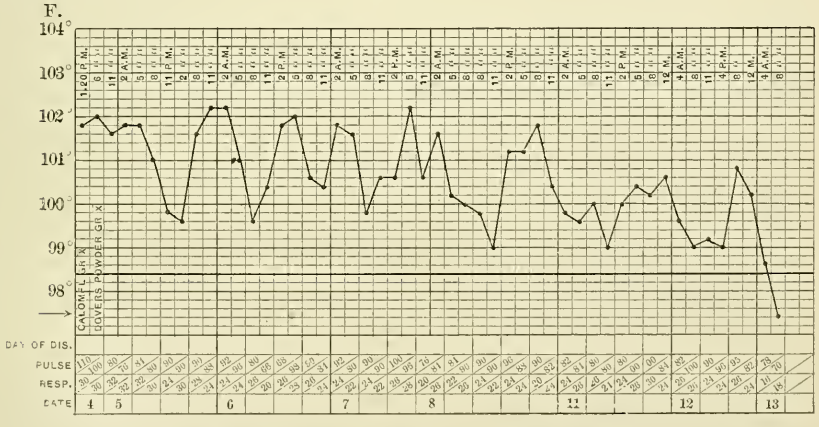
obliterated, dulness continuing to the margin of the ribs. In small effusions the dulness may be limited by the posterior axillary line, resonance being present in the lateral and anterior regions.

On auscultation below the upper level of the effusion posteriorly the voice frequently has a metallic quality resembling the bleating of a goat—*ægophony*. It occurs usually when the effusion is moderate, and may be heard only over a limited area. It is commonly heard at or above the angle of the scapula. Bronchophony may be heard when tubular breathing is present.

While the respiratory murmur is, as a rule, absent, breath-sounds may be heard, and are then weak and distant, or bronchial. In such cases there may or may not be adhesions. Bronchial breathing may be present along the spine in small effusions, and in large effusions in the interscapular region. Bronchial breathing, tubular in character, is said to be almost constant in children. It may also occur when pneumonia coexists. In one of the cases in my ward the signs were like those of a large cavity at the right base, but the immobility, the absent fremitus, the enlargement, and the exploratory puncture disproved its presence.

At the level of the fluid a friction-sound may persist. Above the level of fluid anteriorly the breath-sound may be bronchial or bronchovesicular, associated sometimes with fine râles, due to compression and slight œdema.

Fig. 150.



Pleurisy with effusion. Recovery. (Two days omitted.)

Displacement of Organs. If the effusion is on the left side, the mediastinum and heart become displaced to the right, and the apex-beat may be found in the epigastrium, or even to the right of it. The occurrence of displacement of the heart must also be judged by the position of maximum intensity of the heart-sounds, as the heart may be behind the sternum. At the same time the semilunar space (Traube's line) is lower than usual or entirely effaced. On the left side inaction of the diaphragm may be observed, and the tissues at the costal margin fall in with each inspiration. If the effusion is on the right side, the

diaphragm, and with it the liver, is depressed, and the mediastinal contents are moved to the left.

The *subjective* symptoms during this stage are slight or moderate fever, sometimes intermittent in character, with recurring chills; considerable dyspnœa, occasionally amounting to orthopnœa when the effusion is very extensive; and dry cough, which adds greatly to the dyspnœa. There is frequently some evidence of insufficient oxygenation of the blood; when this amounts to cyanosis, the condition is one of great danger. The urine presents changes in amount. In advancing effusion the amount lessens very much; it increases in amount with the decline of the fluid. Pleurisy may be complicated with bronchitis, pneumonia, and pericarditis.

Empyema. The above-mentioned physical signs apply chiefly to serous effusions. They are also present in effusions of pus. Other physical phenomena, however, and different general symptoms distinguish the two kinds of effusions, although it must be confessed that aspiration must often be resorted to before a positive diagnosis can be made.

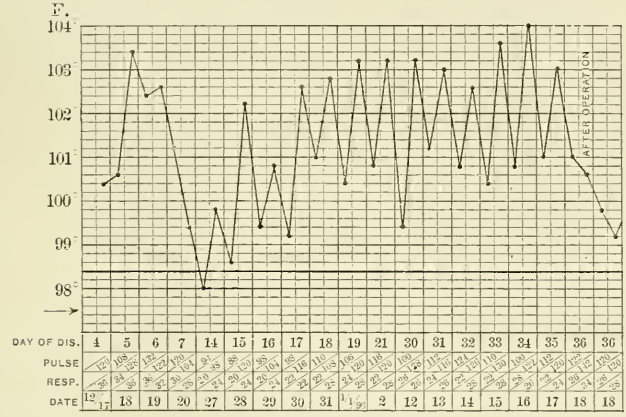
Physical Signs. The physical signs of empyema are the same as those of other effusions within the pleura. In addition, especially in children, local *œdema* of the chest-wall may be found. Another sign was pointed out by Bacelli, and is held by others to be of diagnostic significance. In purulent effusions the fremitus produced by the *whispering* voice is not transmitted to the hand laid over the effusion, whereas in serous effusions such vibrations are transmitted. In loculated empyema the diagnosis is very difficult. In one of my cases dulness continuous with that of the heart extended to the second rib and laterally to the post-axillary line. The dulness occupied three interspaces. Additional physical signs were immobility, prominence of interspaces, localized above the heart, absent fremitus and resonance. There were no breath-sounds, but an abundance of râles, apparently very superficial. The râles complicated the physical signs. Martin operated for me and removed two ounces of pus from a small abscess above the heart and between the lobes.

In empyema a local area may become more prominent and the surface assume an inflammatory appearance. It is an indication of discharge of the abscess through the chest-wall. It is usually found in the fifth interspace in front, or below the angle of the scapula behind —*empyema necessitatis.* (For a microscopical and chemical description of the "Effusion within the Pleural Sac," and of the morphological elements of the purulent effusions, see Chapter XXI., Part I.)

General Symptoms. The general symptoms are more marked in empyema than in simple serous effusion. The temperature is higher from the onset. It soon becomes intermittent or remittent. Chills or chilliness may attend the beginning of each febrile paroxysm, and sweats occur with the daily fall of temperature, or at irregular periods during the twenty-four hours. The heart's action is more rapid and the pulse more feeble, soon becoming dicrotic. Examination of the *urine* may aid in the distinction of the two forms of the effusion. *Albumosuria* occurs in purulent pleurisy. It must be remembered

that albumosuria occurs in suppuration from other causes. Thus, in phthisis with suppuration of a cavity pleural effusion may develop. The albumosuria that attends the primary process must not be mistaken for that which occurs in empyema. *Indican* is also present in excess in the urine in suppurations. Before a decisive conclusion is arrived at two or more examinations of the urine should be made. Examination of the *blood* may aid in arriving at a conclusion. In purulent effusion there is usually leucocytosis.

FIG. 151.



Empyema following pneumonia. (Fever absent from seventh to fourteenth day.)

Notwithstanding the positive physical signs of effusion the character of the effusion may not be recognized until perforation into the bronchus has taken place. The peculiar character of the expectoration that attends this accident is described in the section on Sputum.

Hydrothorax. This is an accumulation resulting from a transudation. (For character of the fluid, see Chapter XXI., Part I.) It occurs in the course of diseases which produce anasarca, as failing organic heart disease, chronic Bright's disease, and debilitating diseases, as scurvy. Locally, it may attend carcinoma of the pleura or obstructive disease of vessels within the mediastinum.

The physical signs of hydrothorax are those of effusion in acute pleurisy. The general symptoms belong to the primary disorder. Dyspnœa may develop gradually and even amount to orthopnœa. It is distinguished from inflammatory effusions by the character of the fluid, by the absence of the general symptoms of inflammation, by its insidious development, and by its bilateral distribution.

Hæmothorax. The transudation of blood into the cavity of the pleura occurs rarely from the rupture of an aneurism into the sac. The fluid is then pure blood. Serous effusions in which a large amount of blood is found point to primary carcinoma of the pleura, or to tuberculous disease. Both specific processes of this serous membrane may occur, however, without the transudation of sero-bloody fluid.

Thickened Pleura. Chronic inflammation, with thickening of the

pleura from excessive development of connective tissue, occurs in tuberculosis and in cases of combined pleuritis and peritonitis. The thickening of the pleura is usually more marked at the base.

The *physical signs* (Plate XXVI.) are pronounced, and are those of effusion, but without enlargement of the chest. There are marked contraction and diminution in movement of the affected side. The fremitus is absent. There is dulness on percussion, or even flatness. The breath-sounds are distant or are absent. Along the vertebræ, especially opposite the angle of the scapula, bronchial breathing may be heard. The subjective symptoms of cough and dyspnœa are present. The degree of cough depends upon the condition of the lung. If there is bronchitis or tuberculosis, the cough is excessive. The amount of dyspnœa depends upon the degree of compression of the lung by the thickened pleura.

Tuberculous Pleurisy.[1] The affection may be acute or chronic. It may occur primarily, be a part of general tuberculous infection, or occur secondarily to disease of the lungs. It may give rise to all forms of the inflammatory process: First, dry pleurisy; second, pleurisy with effusion; third, pleurisy with great thickening. Often the distinction between tuberculous pleurisy and pleurisy due to other causes cannot be determined positively. If it is associated with tuberculosis in other organs, or the patient is of tuberculous habit and exposed to infection, or if there has been a history of previous tuberculosis, the pleuritic infection is probably of tuberculous origin. If the affection is bilateral and associated with peritoneal inflammation, and at the same time no other cause exists for serous membrane inflammation, the probability of its tuberculous origin is very strong.

Pulsating Pleural Effusion. Wilson has made the most recent studies of this rare affection. The effusion within the pleura pulsates synchronously with the ventricular systole; the pulsation is detected usually by inspection and palpation. In some instances its presence is only determined by palpation. It may be confined to two or three interspaces, or occupy the anterior aspect of the thorax and the axillary region on the left side. Rarely the pulsation is behind. It is usually situated on the left side. The original effusion is purulent in the large majority of cases. The physical signs and general symptoms of empyema are present. Nevertheless, the disease simulates aneurism of the aorta. The latter affection, however, is accompanied by vascular symptoms and physical signs in the course of the aorta. Pulsating empyema is distinct in movement from the pulsation of the aorta and occupies a different anatomical site.

Diaphragmatic Pleurisy. In diaphragmatic pleurisy there is intense pain in the epigastrium. Gueneau de Mussy[2] regards a pain along the tenth rib, extending from the anterior extremity to the sternum and xiphoid cartilage, as pathognomonic. Other symptoms are nausea, vomiting, and hiccough. The dyspnœa often amounts to orthopnœa, or the patient sits stooping forward. The anxiety of the

[1] See "Notes on Tuberculous Pleurisy." Musser, American Climatological Association, 1893.

[2] Arch. gén. de Méd., 1853, vol. xi. Quoted by Fox.

PLATE XXVI.

FIG. 1.—Anterior Aspect.

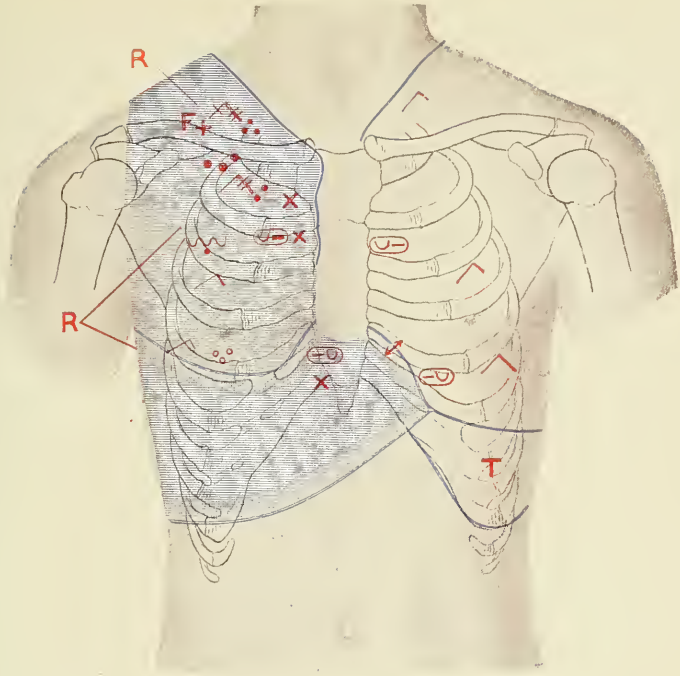
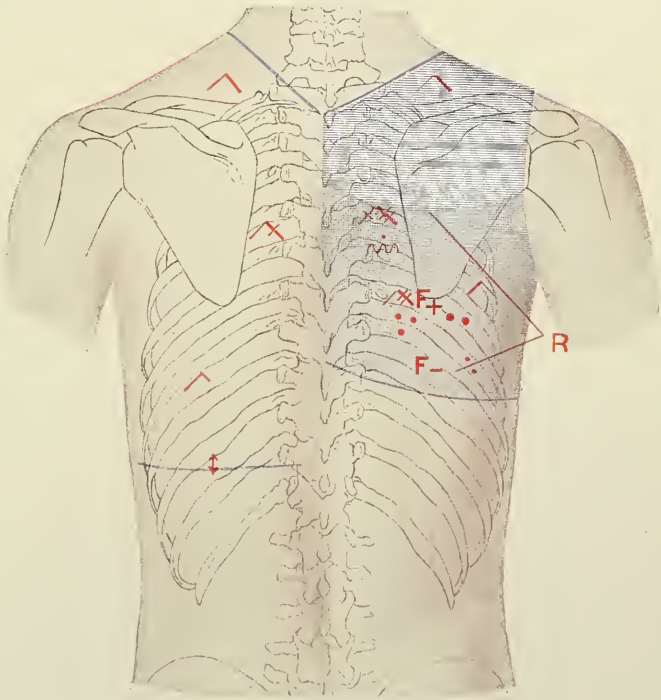


FIG. 2.—Posterior Aspect.



Fibroid Phthisis with Chronic Pleurisy.

Heart drawn toward the right and aorta uncovered by retraction of lung margin. Vicarious emphyzema of left lung.

patient is very great. The fever is usually higher than in ordinary pleurisy, and there may be delirium. Effusion may lessen the pain. Peritonitis may occur at the same time, or be secondary to the pleurisy.

Diagnostic Features. The special features of diagnostic importance that are observed in the course of pleurisy are the pain, the dyspnœa, the cough, the fever, the physical signs of effusion within the pleura, and the results of exploratory puncture. *Pain:* The pain is short, sharp, lancinating, and is usually recognized quite readily by its character and location. It must be distinguished from the pain due to pleurodynia and intercostal neuralgia. The pain of pleurisy is associated with cough and is increased by breathing. It causes diminution of movement of the affected side. The patient is compelled to sit up in bed, or lie on the side which is the seat of pain. *Cough:* In the first stage the cough is short, suppressed, dry, and painful. It is constant. In the second stage it changes in character. There is no pain, there is no expectoration. It is frequent and irritating, and of a peculiar sound which is difficult to describe, and yet, when once heard, is most suggestive in subsequent cases. It is short and lacks resonant quality, as if the fluid in the chest stopped the sound-waves. *Dyspnœa* in the first stage is due to pain, in the second stage to the large effusion which encroaches upon the normal air-space. It is not diagnostic. The *physical signs* of pleural effusion have been frequently reiterated. The most decisive are diminution or absence of movement, enlargement of the affected side, absence of fremitus, flatness on percussion, fulness of intercostal spaces, and the displacement of organs. The latter is of the greatest diagnostic importance in the distinction between consolidation and effusions. The results of *exploratory puncture* lead to decisive conclusions usually, although it must not be forgotten that effusions may be loculated and therefore missed by the aspirating-needle. Or the enormously thickened pleura may intervene between the exudation and the surface of the chest, and prevent withdrawal of the fluid. Finally, effusions may complicate inflammatory processes, as pneumonia, tuberculosis, or abscess of the lung. Securing fluid for diagnosis by aspiration, therefore, does not necessarily exclude these conditions, and hence, before the process is decided to be within the pleura alone, the sputum and other conditions must be taken into consideration.

Differential Diagnosis. *Acute plastic pleurisy* is diagnosticated from *acute pneumonia* by the friction-sound and the maintenance of the clear percussion-note and normal respiratory murmur, with unaltered vocal resonance and fremitus. When *effusion* takes place the chest is enlarged and immobile, especially on the affected side; the interspaces are filled out and the diaphragm is depressed; these changes do not occur in pneumonia. Moreover, the percussion-note in pleural effusion is flat, with greatly increased resistance; the shape of the upper line of dulness is diagnostic; the respiratory murmur is feeble and distant, or entirely absent, except along the spine, where the compressed lung yields bronchial breathing, and also above the line of effusion, where the lung yields exaggerated breathing. In pneumonia, on the other hand, the percussion-note is dull, without greatly increased resistance,

and the breath-sounds are bronchial. In addition, in pleurisy, the vocal resonance and fremitus are usually almost if not entirely absent, and posteriorly at the level of the effusion ægophony may be detected. In pneumonia, on the contrary, vocal resonance and fremitus are increased in intensity. In pleurisy with effusion the movable organs are dislocated and Traube's line is obliterated.

Finally, the fever of pneumonia is much higher and more continuous than that of pleurisy, the respirations more frequent, the cough looser, and in typical cases followed by rusty sputa. (Compare the temperature chart in article on Pneumonia.) A crucial test is *aspiration* with a hypodermic needle; in pleural effusion, serum is withdrawn; in pneumonia, a few drops of thick blood.

In *pleurodynia* there is also severe pain in one side; but the pain is more continuous than that of pleurisy, and consists of a constant aching or a burning sensation. It is made worse by twisting or turning, as well as by breathing. The side is also tender to the touch. The pain is not so sharply localized as that of pleurisy, and may leave one side and affect the other. It is unaccompanied by fever or friction-sound, and is frequently found in rheumatic subjects.

In *intercostal neuralgia* there is the same absence of fever and friction-sound. The pain, however, is sharply localized, as in pleurisy, but is of the darting, neuralgic character, and is associated with tenderness at the points of exit of the intercostal nerves. It is most common in women, especially if they have uterine disturbances. It is more frequent on the left side, and just beneath the mammary gland.

Chronic Pleurisy. *Chronic dry, or plastic, pleurisy* is the result of an acute attack, or develops insidiously if tuberculous. It causes great deformity of the chest from contraction, and compensatory emphysema of the healthy lung. The heart is dislocated or cannot be found on physical examination, because it is overlapped by lung or is drawn behind the sternum. There is considerable spinal curvature, dislocation of the scapula, deformity of the shoulder, and indrawing and overlapping of the ribs at the base of the chest.

Chronic pleurisy with effusion results from an acute attack of pleurisy, in which the fluid remains unabsorbed, or from subsequent attacks. The physical signs are the same as in acute effusion. So far as subjective symptoms go it may remain latent; patients so affected not infrequently go about their work with comparatively little dyspnœa. There may be an evening rise of temperature and acceleration of the pulse. Chronic effusions are more likely to be purulent in children than in adults. When empyema results, the fever becomes hectic; there are chills and sweats, pyæmia develops, and death is likely to occur from some intercurrent suppuration, as cerebral abscess.

After *chronic effusion* the chest is rarely restored to its original shape, even if the effusion is finally absorbed. The affected side becomes motionless and retracted. In process of time the spine may be bent. The opposite lung becomes hypertrophied. The patient is usually in precarious health, liable to acute attacks of pain in the affected side, and liable also to be carried off by phthisis or some intercurrent affection. Rarely the patient may maintain good health; complete cure

PLATE XXVII.

FIG. 1.—Anterior Aspect.

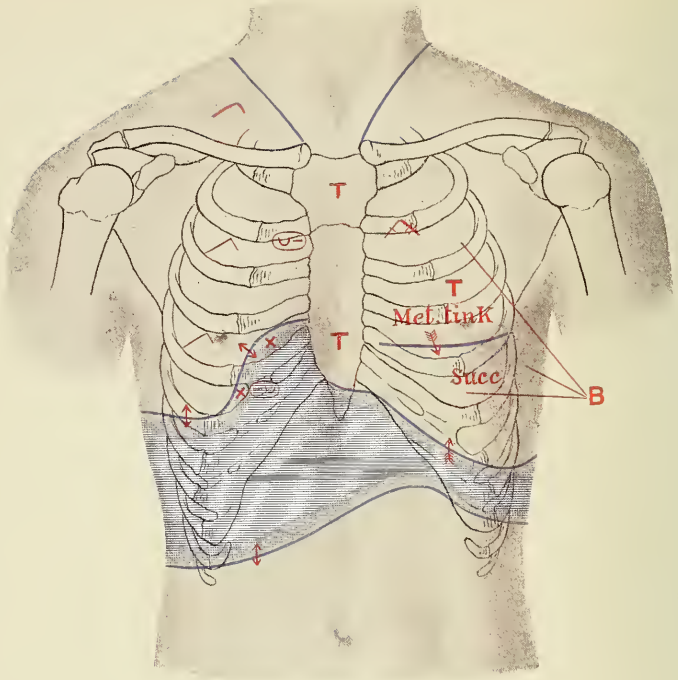
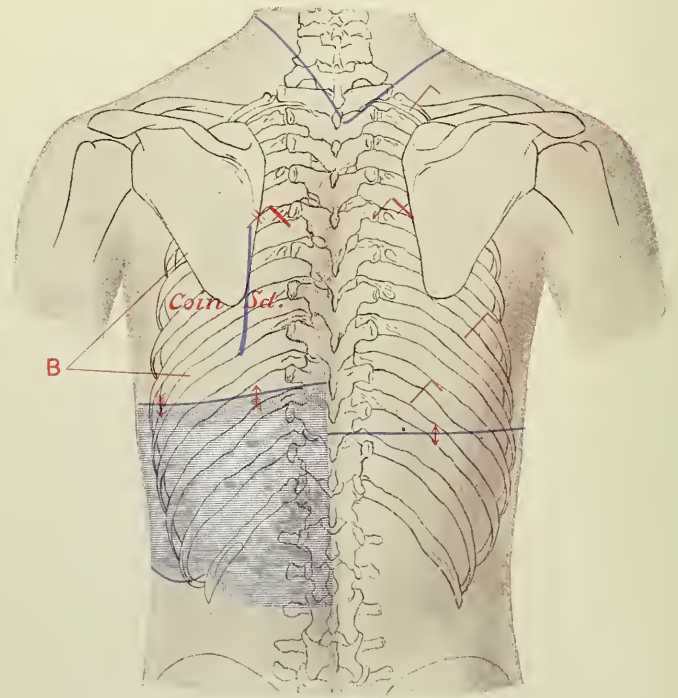


FIG. 2.—Posterior Aspect.



Pneumothorax (left-sided).

is even possible, with restoration of the retracted side to, or almost to, normal dimensions, especially in children.

Pneumothorax. Pneumothorax consists in an accumulation of air in the pleural cavity, accompanied or followed by an outpouring of fluid, which may be serous or purulent, constituting respectively *hydro-pneumothorax* and *pyo-pneumothorax*.

Pneumothorax may originate: 1. In causes external to the chest, by perforation of the chest-wall and pleura. 2. In perforation of the lungs, bronchi, or œsophagus. 3. It may be caused by gases developed from an existing effusion.

The most frequent cause is tuberculous disease of the lung, and next an empyema; out of 121 cases collected by Saussier, 81 were due to phthisis and 29 to empyema. It may occur very early in tuberculosis of the lung, and may even be the first symptom of that disease. (See cases referred to by Fox and recorded by Louis and Chomel). The left side is affected not quite twice as often as the right; the disease is usually unilateral. The onset of the condition is usually sudden. During a paroxysm of coughing or vomiting, or without immediate cause, there is an escape of air into the pleura, and in the majority of cases the patient at once complains of acute pain in the chest and excessive dyspnœa with great dread of impending suffocation. The patient often sinks into collapse from shock, but sudden death is rare. If the escape of air into the pleura is gradual, there will be less pain and dyspnœa.

Physical Signs. (Plate XXVII.) The chest is distended, especially on the affected side; the percussion-note is a bell-like tympany except when the distention is excessive and the air contained is under great tension, when the note is proportionately duller and higher in pitch; the diaphragm is depressed and the heart displaced, unless adhesions prevent it. In left pneumothorax it may beat on the right side, the whole mediastinum being pushed to the right; in right pneumothorax the mediastinum may be pushed to the left nipple; hence there is resonance over the normal cardiac region. The pitch of the percussion-note may be raised when the mouth is closed, and lowered when it is open (Wintrich's change of note), and a cracked-pot sound can be elicited in some cases, but this occurs only when the communication with the pleura remains open.

A valuable sign of pneumothorax is the coin-test, or, as Trousseau named it, the *Bruit d'airain*. A silver coin is laid upon the chest and struck with another, while the auscultator applies the stethoscope opposite to the point struck, or over any part of the side distended by air. The ringing coin-sound is reproduced with great intensity. It is pathognomonic, and the outlines of the cavity can be traced by it.

When fluid is present, as it usually is, there will be the ordinary signs of a pleural effusion, which have been sufficiently dwelt upon. The fluid is more mobile in pneumothorax, however, than in simple pleurisy, so that its level changes more quickly with change of posture of the patient, and *Hippocratic succussion* is readily obtained. This movable dulness is a very valuable sign—indeed, almost pathognomonic.

As the lung is compressed against the spine by the air, as it is by

the fluid in pleurisy, the breath-sounds are feeble or absent, except over the root of the lung, where the breathing is bronchial. But if the lung is not completely collapsed, amphoric breathing may be heard, the air-chamber of the pleura acting as a consonance-box; it may be heard with both inspiration and expiration, or only with expiration.

Metallic tinkling is a sound believed to be due to the vibration of bubbling bronchial râles re-echoed through the air-chamber, or to drops of fluid falling from above upon the surface of the effusion. Re-echoing, with metallic quality, may also accompany the heart-sounds, and in cases in which the respiratory murmur is amphoric the vocal resonance is of the same character. Vocal fremitus is generally absent.

Differential Diagnosis. *Pneumothorax* is most likely to be confounded with (1) emphysema; (2) tuberculosis of the lungs with large cavities; (3) cases of pleural effusion in which above the upper level of the fluid the lung is markedly hyper-resonant; and (4) abscess below the diaphragm containing air (pyo-pneumothorax subphrenicus).

1. *Emphysema* can be distinguished by its slow onset, its relatively slight impairment of the general health, by the fact that it is bilateral, whereas pneumothorax is almost always unilateral, and by the existence of feeble breathing with greatly prolonged expiration. Amphoric breathing and resonance, metallic tinkling, and signs of fluid are all absent in emphysema.

2. When the *pneumothorax* is circumscribed the physical signs resemble those of *pulmonary cavity*. But over a large cavity the chest is usually flattened; cracked-pot sound and alteration in pitch upon opening and closing the mouth are more common in cavity than in pneumothorax. Displacement of viscera does not necessarily occur in phthisical cavity, the coin-test is negative, succussion cannot be produced. Fremitus is absent in pneumothorax and increased over a cavity.

3. The hyper-resonance above a *pleural effusion* develops with a very different clinical history, is accompanied by increase of fremitus with bronchial or, at times, amphoric breathing, and changes when the patient's mouth is open or closed. The percussion-note usually lacks the metallic quality heard in pneumothorax, metallic tinkling is absent, the coin-test is negative.

4. Pneumothorax must be distinguished from abscess below the diaphragm containing air (*pyo-pneumothorax subphrenicus*). Often the distinction is difficult. The constitutional symptoms of suppuration are present. Leyden points out the importance of remembering the sequence of events in the development of the disease. When the abscess is situated below the diaphragm, abdominal symptoms precede its development, and early in the course of the disease there is absence of respiratory symptoms. If the patient has had gastric ulcer, this would point to subphrenic abscess, as most of the cases of subphrenic abscess are secondary to gastric ulcer. Moreover, in subphrenic abscess the heart is not displaced nor the interspaces bulging. Indeed, the viscera below the diaphragm are more likely to be displaced than those above it. In pneumothorax, according to Leyden, the respiration is

normal under the clavicle, and the transitions from the normal to the metallic and amphoric sounds lower down are abrupt. In pyo-pneumothorax on the left side the semilunar space disappears. In subphrenic abscess the amphoric sounds laterally or posteriorly may be above and below the diaphragm, or they may be loudest at the epigastrium. In addition, in pyo-pneumothorax subphrenicus, as Mason points out, adhesions of the lung to the diaphragm and parietes can be made out, particularly if the case has been under observation in its earlier stages and dry pleurisy has been discovered. Abscess in this location and slight fluctuation are likely to develop with associated effusion. The limited extent of the effusion is of diagnostic import in favor of sub-diaphragmatic inflammation.

CHAPTER III.

DISEASES OF THE HEART, THE BLOODVESSELS, AND THE MEDIASTINUM.

The symptoms of disease of the heart are due to the anatomical structure of the organ, to its physiological offices, and to the morbid process. The heart is a hollow muscular structure which hangs in a cavity and encloses cavities separated by valves. Both sets of cavities are lined by serous membrane. The serous membranes are subject to the same diseases, and present the same symptoms as diseased serous membranes elsewhere. In inflammation of the external membrane the surfaces rub together and create a sound of friction. The external serous cavity may also become filled with the products of exudation or transudation. Physical signs are produced. They are the physical signs of a localized increase of contents as determined by inspection, palpation, and percussion, and of physical interference with the heart's action. The heart-muscle is also subject to the same morbid processes as other muscular structures. They are hypertrophy and atrophy; inflammation, acute and chronic, with overgrowth of connective tissue; and degenerations. The symptoms are likewise the same. Increase or diminution in the power of the muscle is associated with corresponding change in size, which is determined by physical signs. Above all, however, such change modifies the heart's action so that strength or weakness of the muscle shows itself in excessive or deficient vascular pressure. The latter is more particularly an object of observation because of the congestions, dropsies, and cyanosis that ensue.

The heart is constantly subjected to internal pressure. Dilatation of the cavities or a portion of cavity (aneurism) follows previous disease of the muscle or increase of internal pressure, and causes physical signs of enlargement. Degeneration of the heart-muscle, nearly always secondary to deficiency of vascular supply, is also attended by symptoms of weakness and physical signs of enlargement (dilatation), or of diminution in size (atrophy). When dilatation occurs the orifices of the cavities enlarge, the valves cannot close them, and symptoms of incompetency and of blood-regurgitation result.

The serous membrane that lines the cavities of the heart and, with the subserous tissues, makes up the structure of the valves, is subject to inflammations, the symptoms of which are common to all serous inflammations. The swellings and outgrowths that attend such inflammation occlude the orifices and prevent closing of the valves. A physical interference with the heart's function is produced, recognized by physical signs. The successful effort of the heart-muscle to overcome such obstruction on the one hand (hypertrophy), or its failure on the other (dilatation), again leads to the production of symptoms and

signs. The serous membranes, and hence the valves, are exposed to causes which excite inflammation. By virtue of the position of the heart at the centre of the circulation, the blood, infectious or irritative, as in rheumatism and Bright's disease, constantly bathes the vulnerable structure. For the same anatomical reason positive symptoms arise, not common to serous membrane inflammation—that is, *embolic* phenomena. (See Symptoms of Morbid Processes.) Hence, the physical signs (objective symptoms) of cardiac disease may be due to primary and secondary morbid anatomical changes. They may be due (1) to valvulitis as indicated by signs of (*a*) obstruction or regurgitation at the valve-orifice, or (*b*) of embolic phenomena ; (2) to secondary changes in the heart-muscle as seen in (*a*) change in the size and strength of the organ (hypertrophy or dilatation), and (*b*) in consequence of the latter, signs of congestion, œdema, cyanosis, etc.

It is the function of the heart to propel the blood. It has been shown how interference with the action of the muscle and with the consequent flow of blood through the cavities and orifices modifies the function. The functional power is increased or diminished by the physical changes. The evidence of increased power is increased force of the heart-beat, and increased pressure in the arteries (pulse).

Diminished power shows itself in symptoms of diminished blood-supply to parts, and in stagnation of the blood that is sent to the periphery. The former is more pronounced in cerebral anæmia, and physiological weakness of organs or the organism as a whole ; the latter, in congestion and dropsies.

The functional activity of the heart is controlled by a nervous mechanism, any alteration of which alters cardiac action and consequently produces symptoms. Just as with the larynx, a break in the cardiac mechanism may be in the centres in the medulla, the centres in the muscle, or in the sympathetic nerves to and from the heart. The rich anastomosis of these nerves exposes the heart to disturbance by reflex influences. We should suppose such extensive innervation would invite frequent cardiac perturbation. In a measure it does, but, fortunately, so perfect is this mechanism that the inhibitory fibres control such perturbation to a large extent, and we do not see such pronounced symptoms as occur in the larynx. The symptoms which point to disturbance of the cardiac mechanism are alterations in the rhythm of the heart. Its action may on this account be increased or diminished in frequency, or it may be irregular or intermittent. Such alterations of rhythm may be due to organic disease of the centres, notably the pneumogastric from apoplexy, softening, or tumor in the medulla, or to stimulation or depression of the centres by toxic substances in the blood, as in uræmia, acetonæmia, or autogenetic or other toxæmias, or by nicotine or other extraneous material. The altered rhythm may be, and most frequently is, of reflex origin. It may be due to disease of the nerves, as the pneumogastric or sympathetic, from pressure upon the nerve-trunk by tumor or inflammatory growth. The most pronounced symptom of altered rhythm of which the patient is cognizant is palpitation. The exciting cause of this, as well as other rhythmical changes, must, in the great majority of cases, be sought for beyond the domain of the heart.

While the symptoms or signs of cardiac disease are often due to morbid processes in the organ or its membrane, it must be remembered that grave and persistent subjective and objective symptoms may be caused by, or at least associated with, disease of contiguous structures outside of the pericardium. The symptoms are not excited through the nervous system, but are produced by mechanical encroachment upon the organ, as in pleurisy with effusion, mediastinal disease and disease of subdiaphragmatic viscera. They will be referred to in the study of objective symptoms. Care must be taken never to overlook the possibility of their presence.

In the study of the symptomatology of cardiac disease the student must bear in mind two things : *first*, that the cause of the morbid processes and of the symptoms (pain and palpitation) may be elsewhere than in the heart ; and, *second*, that the ultimate object of the examination is to determine the muscular power of the heart. He will soon learn that with that power intact the functions can be performed, notwithstanding the presence of marked physical abnormalities.

The recognition of disease of the heart is not usually attended by much difficulty, except in some special lesions. The non-recognition of cardiac disease is due to faults in the examination. The physician is too often satisfied with the recognition of the remote process, as a congestion or functional weakness in some organ. Safety lies, as has often been said, in the examination of all the organs of the body. Often, for instance, indigestion from gastric catarrh is not relieved, for the cause, mitral regurgitation, is not recognized.

The Data Obtained by Inquiry.

The Social History. The incidents in the social history to be considered in the determination of the presence of cardio-vascular disease are those which notably influence by strain, excitement, or wear and tear, the cardio-vascular mechanism—those which alternately increase and diminish cardiac action, open and shut, dilate and contract peripheral vessels. Whether it be symptoms of functional disorder or of organic disease we wish to unravel, we must inquire as to the use of stimulants, of tea, coffee, tobacco, and other narcotics or poisons ; as to mental anxiety or physical strain ; as to excesses of various kinds. Excess in any form induces vascular wear and tear. Tersely put by one of our most distinguished clinicians, the devotee at the shrine of Venus, Bacchus or Mars, is too frequently the victim of vascular disease. Occupations which invoke such vascular excitations are suggestive diagnostic factors.

The *age* in which we are wont to find cardio-vascular affections varies with the character of the lesion. Apart from congenital cardiac affections, acute inflammations are more common at the age when infections are more operative, as in the early decades. On the other hand, and it goes without saying, degenerative lesions are found in later life. But as man is no older than his arteries, and as these degenerative lesions may occur in comparatively early life, from a cardio-vascular stand-point, a man may be senile at *thirty-five* or even earlier. *Sex*

influences diagnosis in so much as the one sex is more exposed to the causal influences of cardiac lesions. Females are more prone to acute infectious processes and to the neuroses from immobile nervous systems. Males to degenerative lesions and the intoxication neuroses.

Family History. Inquiry in this direction yields information of great diagnostic value. The gouty and rheumatic diatheses, with their long train of associated disorders, which predisposes to cardio-vascular affections, are notably inherited. Moreover, the tendency to atheroma of vessels is itself pronouncedly hereditary.

The History of Previous Disease. The occurrence of any one of the numerous infections may have been the initial step in the production of the affections we are considering. The determination of the nature of a cardiac lesion may hinge upon the correct decision of this question. The infection of acute rheumatism is of course to be eagerly sought for. A history of chorea, of various skin affections related to gout and rheumatism, of eye affections, of tonsillitis, of other affections related to the so-called uric-acid diathesis, must be sought for. If found, such history is more than suggestive.

The Subjective Symptoms. *A.* Symptoms Referred to the Heart. Pain. 1. *In Disease Outside of the Heart.* Although pain in the region of the heart may be a symptom of disease of that organ or of the pericardium, in the large majority of instances it is due to other causes. The physician is frequently consulted by the anxious patient on account of pain, other than heart-pain, but referred to this region, or more precisely to the fifth or sixth interspace on the left side. The causes of such pain are various : (1) Neuralgia ; (2) pleurodynia ; (3) myalgia ; (4) local pleurisy ; (5) periostitis. The *neuralgias* may be associated with points of tenderness, which are usually the seat of the greatest intensity of the pain. These points of tenderness correspond with the positions at which the nerves have their exit through the fascia to the surface, and are found along the sternum, in the course of the mid-axilla, and along the vertebræ. The pain is paroxysmal, occurs at variable periods of the day, and in anæmic subjects or in the course of neurasthenia. It may precede the development of herpes zoster. In these cases the exact nature of the pain is not known until the eruption appears. In gout or diabetes we may have local neuritis, which causes neuralgic pain in this situation.

Pleurodynia, which is thought to be an affection of the pleural nerves, is more general. The pain is increased by pressure of the finger-tips, although it is not localized. It is relieved by pressure of the whole hand. In *myalgia*, which is seen so frequently in phthisis, on account of severe coughing, in rheumatism and in debilitated subjects generally, the pain is more or less diffuse, interferes more or less with movements of the chest, is relieved by uniform general pressure, and is usually associated with myalgia in other organs. The pain of *pleurisy* is recognized by the fact that it usually inhibits the act of breathing, and is associated with cough, and because friction-sounds may be detected. *Periostitis.* In disease of the ribs of the præcordia the pain is associated with tenderness and swelling. One or more of the costo-sternal articulations may be extremely tender. The pain and tender-

ness are due to the periostitis of syphilis or to that which follows typhoid fever. In one of my cases the rib had to be resected. It may be due to the internal pressure and erosion of ribs in aneurism. The same affection may cause neuralgic pains in the nerves. *Abscess.* Pain in this region may, in rare instances, be due to localized tuberculous abscess between the pericardium and the walls of the thorax. One such case was under my care. The abscess developed secondarily to empyema and occupied the præcordial region, causing bulging. The pain was intense, and was only relieved after the caseating pus was removed by incision.

Pain in the *epigastrium* is often held to be due to cardiac disease. It is usually due to gastralgia, or, as it is sometimes termed, cardialgia. It is recognized by the location of the pain and its association with gastric symptoms, as flatulency, weight, fulness, and acidity. In gastric ulcer the epigastric pain is localized, accompanied by tenderness on pressure, and is increased by food. However, acute, severe, and excruciating pain in the epigastrium may be due to *rupture* of the heart and also to *pericarditis.*

2. *In Disease of the Pericardium.* Pain in the region of the heart is sometimes due to affections of the pericardium. *Pericarditis* is the most common. While centralized in the heart-region, it may radiate to the left shoulder and extend down the arm. It is paroxysmal and may have some of the characteristics of angina. It is increased by movement, by pressure, and by the action of the diaphragm. The patient is often obliged to sit up in bed and suffers from orthopnœa. It may be referred to the epigastrium. A pericardial friction-sound is usually detected. *Pain due to disease of the aorta. Acute inflammation of the aorta* is also the cause of cardiac pain. The pain extends along the course of the aorta, may be referred to the sternum, and extends along the spine. The pain is severe, causing an anxious countenance and an expression of extreme suffering. In gouty subjects with *atheroma* pain may occur in this situation in paroxysms. There is usually valvular disease at the aortic orifice. Similar pain occurs in syphilis and in alcoholic subjects, and may be due to malaria. It is a visceral neurosis, or a form of neuralgia.

Pain in the region of the heart is frequently due to *aneurism.* The pain is usually due to pressure of the aneurism upon adjacent structures. If it presses on the bone and causes erosion, the pain is of a boring character, localized at one point. It has been previously referred to. In aneurism alone, without pressure, the pain is of a dull aching character, increased by movement, relieved by rest, or by change of position. When nerves are pressed upon, pain may be acute and of a neuralgic nature. It may follow the course of the nerves and be associated with numbness or sensations of tingling. The long duration of the pain, its localization, and its aching character are sufficient to exclude angina pectoris. When the pain is unilateral it may be due to pressure of an aneurism upon the nerves at their exit from the canal; the pain extends along the course of the intercostal nerves. It is severe and burning, but there are no localized points of greater intensity. The pain may extend down the arms, and, when the abdomi-

nal aorta is affected, it may extend down the legs. If rupture of the aneurism takes place, the pain is sudden and sharp. Death, however, ensues quickly, so that the pain will rarely be complained of.

3. *In Disease of the Heart.* Three forms are seen : (1) Pain due to disturbances of the rhythm ; (2) pain due to valvular disease ; (3) pain due to angina pectoris.

Disturbance of the Rhythm. Palpitation, intermission, and irregularity of the heart occur in the large majority of cases without pain. Paroxysms of palpitation are sometimes attended with severe præcordial pain and distress. This occurs in the reflex palpitation, which, as will be seen, is due to disease in other situations ; in the palpitation of Graves' disease and of anæmia. The palpitation of organic disease is induced by exertion. The rapid action of the heart is painful and the throbbing is complained of as causing distress.

While intermission and irregularity may continue without pain at times, the patient is conscious of this disturbance of the rhythm, and complains of the stoppage, which then is attended by distress, sometimes amounting to severe pain. This is particularly the case when the heart-action is tumultuous, as the disturbance of rhythm seen in pericarditis and in valvular disease.

Pain due to Valvular Disease. In disease of the aortic valves pain is of more frequent occurrence than in other valvular lesions. It is usually complained of in the region of the aorta at the base of the heart, and is aggravated by exertion. (See Atheroma.)

Pain due to Angina Pectoris. Heberden was the first to describe the attacks of angina pectoris, which, in its typical form and in association with disease of the heart, is not of common occurrence. The pain of angina is severe and is associated with the most intense anguish. It comes on suddenly, and may occur in paroxysms. The patient realizes that the pain is in the heart, and complains of feeling as if the organ were held in a vise. From the heart it radiates to the neck and down the arms. It extends particularly to the left arm, and may be severe in the wrist or in the ends of the fingers. With the pain there is a sense of impending death with sinking and depression. The pain lasts but a few seconds or minutes, and during that time the face of the patient becomes pale or of an ashy hue, perspiration breaks out on the forehead, the extremities become cold, the breathing is short. Prostration usually follows the attack, but the præcordial distress disappears entirely. The attack may occur in patients who are entirely free from organic disease of the heart. It is most commonly, however, associated with some lesion. The lesions frequently found are disease of the coronary arteries, atheroma of the aorta, aortic valvular disease, and myocarditis with fatty degeneration. It occurs after middle life, and is more frequent in males. It may occur without exciting cause, or follow undue exertion, exposure to cold, mental excitement, or profound emotion.

The points upon which the diagnosis is based are : 1. The seat of the pain. This is usually behind the middle or the lower part of the sternum, and more to the left than to the right. Thence it extends to the posterior portion of the axilla or it may radiate up to the neck.

In some instances it extends to the occiput. Frequently the pain extends to the left arm as far as the elbow or even the fingers. It may extend to the abdomen or to the right arm. I have seen it affect both arms. It is not influenced by external pressure. 2. The sense of constriction with the indescribable torture are most characteristic. 3. The respirations are shallow, or may even cease, but there is no dyspnœa. 4. The patient is terrified and restless. 5. The pale face, extremely anxious countenance, the cold sweat on the forehead, make a striking picture, which when once seen can never be forgotten. 6. Such extreme depression and sensation of impending death occur in no other affection. Particularly characteristic is the immediate relief, without hysterical manifestations or dyspeptic symptoms of any kind, which follows an attack. 7. During the attack the frequency of the pulse is not much influenced, and the action of the heart may be uniform and regular. Rarely its frequency may be lessened. The tension of the pulse is increased during the attack.

Some authors speak of various grades of angina, and call all forms of præcordial pain and oppression, with radiation of the pains to the arms and neck, mild forms of angina. Such attacks have often obvious causes in disturbance of digestion and in emotional excitement. When associated with increased arterial tension and signs of arterio-sclerosis, they may be of an anginoid nature. The greatest difficulty exists in distinguishing them from true angina. *Hysterical* or *pseudo-angina* can be distinguished only with extreme difficulty. It occurs much more frequently than true angina. One attack seems to predispose to others. It occurs in females who present other symptoms of hysteria. It occurs usually before forty years of age. The attacks most frequently come on at night, and may be periodical. They are particularly associated with menstrual disorders. The pain is less severe and the oppression is not so marked in pseudo-angina; coldness of the hands and feet, with the occurrence of syncope, or a general feeling of sinking, are common symptoms. The pain is of long duration and is associated with great agitation. It is preceded by neuralgia, and neuralgic pains persist after the attack. Low tension, feeble second sound, and soft arteries may be present, although the opposite is also seen. The disease is never fatal. In one of my patients attacks of hysterical hæmoptysis alternated with the anginal attacks.

Palpitation. In palpitation the patient is conscious of the action of the heart. Although it may occur in organic disease, it is more frequently due to disease outside of the heart.

Symptoms. The symptoms vary in degree. In mild forms the patients may complain of a fluttering or a sensation of sinking in the præcordial region. In the more severe forms the heart beats violently against the chest. The arteries throb, the action of the heart is increased, and the area of impulse against the chest-wall is enlarged and visible. The patient complains of distress in the præcordial region. The pulse may be increased to 150. In nervous palpitation the face becomes flushed, and after the attacks large quantities of urine are passed. Sometimes, in this form of palpitation, exertion relieves the attack. On examination, the sounds are found to be normal, but they

are clear and metallic in character. The diastolic sounds are greatly accentuated. If anæmia is present, murmurs due to that condition are increased in intensity. The attack may last but a few minutes or continue for hours.

(*a*) It is most common in cases in which the nervous system generally is in a state of increased excitability. Attacks occur at puberty and at the menopause. It is very common in hysteria and neurasthenia. It follows emotional disturbance. It is more frequent in women.

(*b*) It is due to the action of the toxic substances, as tobacco, tea and coffee, and alcohol.

(*c*) From strain and over-exertion, particularly if associated with excitement, palpitation may occur and continue for a long period. This is the form of irritable heart described by Da Costa, common in young soldiers during the war.

(*d*) In valvular disease of the heart when compensation fails, and in myocarditis, attacks of palpitation occur, distinctly from exertion.

Intermission and Irregularity. When the patient feels the alteration in rhythm, it is usually due to nervous disturbance. In organic disease it is not, as a rule, appreciated by the patient. Although not a subjective symptom alone, it may be well to speak of irregularity in this connection.

Arrhythmia is the general term applied to irregularity of the action of the heart. When the heart intermits—that is, when one or two beats are dropped at intervals of half a minute, a minute, or longer; when the beats are unequal in volume and force, or occur at unequal distances in time, the heart's action is irregular. The causes of disturbance of the rhythm have been classified by Baumgarten[1] as follows:

1. Central causes in the medulla either from organic disease, as hemorrhage or concussion, or from physical influences. 2. Reflex influences, as in dyspepsia and diseases of the liver, lungs, and kidneys. 3. Toxic influences—tobacco, coffee, and tea are common causes; various drugs, such as digitalis, belladonna, and aconite. 4. Changes in the heart itself. Mural changes, as in dilatation, fatty degeneration, and myocarditis; changes in the cardiac ganglia; sclerosis of the coronary arteries.

It must not be forgotten that both irregularity and intermittency may occur in persons otherwise in good health, and continue for a long period of time without any evidence of arterial or cardiac disease. (For the varieties of arrhythmia, see The Pulse.)

B. Symptoms Referred to the Circulation. 1. *Pulsation of the Arteries.* Pulsation of the arteries, especially the carotids, the abdominal aorta, and the brachial arteries, occurs in anæmia, and is common in emotional disturbances. Such pulsation, as of the abdominal aorta, may be reflex from organic disease in the vicinity. Similar localized pulsation in the innominate arteries may be mistaken for aneurism. The pulsation that attends organic heart disease may be due to hypertrophy of the heart, but is particularly characteristic of aortic regurgitation.

[1] See Transactions of the Association of American Physicians, vol. iii.

2. *Hemorrhages.* In the description of valvular lesions it will be seen that hemorrhages from the lungs occur quite frequently in disease of the mitral valve. The hemorrhage may be due to congestion, to actual rupture of the vessels, or to hemorrhagic infarct. (See Pulmonary Hemorrhage.) It may simulate hemorrhage due to tuberculosis.

3. *Cyanosis.* Cyanosis is a symptom of common occurrence in the course of organic heart disease.

4. *Dropsy.* The dropsy of heart disease occurs after failure in compensation in the course of valvular disease, and in dilatation of the heart. It may disappear entirely, if the conditions are improved, or become permanent and progressive. In general, it may be said to be distinctly a phenomenon of mitral regurgitation and secondary tricuspid regurgitation. It occurs in a lesser degree in mitral obstruction, and still less in disease at the aortic orifice.

C. Symptoms Referred to the Lungs. The chief subjective symptom is dyspnœa. *Dyspnœa,* due to disease of the heart, is clinically divided into (1) dyspnœa caused or increased by exertion; (2) paroxysmal dyspnœa; (3) orthopnœa; (4) rhythmical dyspnœa, or Cheyne-Stokes respiration. The dyspnœa of effort comes on after the slightest exertion. In paroxysmal dyspnœa the attack comes on without apparent cause. It must be distinguished from the paroxysmal dyspnœa of uræmia, asthma, or emphysema. The physical signs of lung disease usually point to the latter. The paroxysmal dyspnœa of heart disease is attended by more violent efforts in breathing than the physical state of the lungs admits, and the difficulty attends both inspiration and expiration. Wheezing is not so marked as in forms of asthma. There is some obstruction to the outgoing of air; but, on account of air-hunger, all the efforts of the patient are exerted to fill the chest. In paroxysmal dyspnœa the breathing usually becomes quiet if the patient is placed in a comfortable position, provided there is no lung or pleural complication. The position does not modify the severe dyspnœa of asthma or emphysema. Orthopnœa has been described previously.

Cough. Cough is of frequent occurrence in heart disease. The causes are various. It may be due to pressure upon the bronchus or the pneumogastric nerves, as in pericardial effusion. It may be due to the passive congestion of the lungs which occurs in failing compensation. If hemorrhagic infarcts take place, cough may be present. It attends the bronchopneumonia that follows. In cough from pressure of an aneurism a metallic brassy sound is created. (See The Larynx.) It occurs in paroxysms, and may be associated with alterations in the voice. It may result in the expectoration of blood-tinged sputum, which may be due to the gradual rupture of the aneurism.

D. Symptoms Referred to the Nervous System. The symptoms are usually due to disturbance of the cerebral circulation, because either an insufficient quantity of blood or improperly oxygenated blood is supplied to the brain. Vertigo, faintness, and languor are complained of in the first instance. Dulness, stupor, and moderate delirium (car-

bon-dioxide poisoning) may occur in the later stages in the second instance. In the course of organic heart disease *epilepsy* or *epileptiform convulsions* may arise, on account of embolism or thrombosis. *Chorea* is of common occurrence, and apparently of the same cause as the heart disease. *Coma* may be due to hemorrhage into the brain, to embolism, or to thrombosis. Hemorrhage occurs in patients in whom there are usually found hypertrophy of the left ventricle, atheroma of the arteries, and renal disease. Embolism occurs in valvular disease, particularly in aortic regurgitation and mitral obstruction. We may have the occurrence of paralysis for the same reason, with or without coma. The *Stokes-Adams syndrome* of vertigo, syncope, loss of consciousness, and slow pulse—pseudo-apoplexy—is seen in myocarditis and endarteritis.

Thrombosis in the course of heart disease is usually due to disease of the bloodvessels rather than to disease of the heart itself, although a weakening of the heart, as in dilatation, is a factor predisposing to the development of thrombosis.

E. Symptoms Referred to the Alimentary Canal. In the course of organic heart disease dyspepsia and forms of catarrhal gastritis and enteritis are of common occurrence. Patients complain of various forms of indigestion, or of nausea and vomiting. While water-brash and flatulence are caused primarily by the condition of the heart, they may in their turn cause symptoms of palpitation and cardiac distress. These gastric difficulties are more particularly seen in diseases of the auriculo-ventricular valves, and are associated with congestion and secondary cirrhosis of the abdominal viscera.

F. Symptoms Referred to the Throat. The patient may complain of pain in the throat. This may be paroxysmal, and is sometimes said to be due to angina pectoris. Hoarseness or modifications of the voice are occasional symptoms of pericarditis. They are of frequent occurrence in the course of aneurism due to pressure upon the recurrent laryngeal nerves.

G. Symptoms Referred to the Kidneys. The kidneys are intimately related with the heart at a distant point in the circulation, and are frequently the seat of changes due primarily to disease of the central organ of circulation. The changes in the urine will be referred to again; suffice it to say, that in the course of mitral and tricuspid disease and dilatation, scanty urine, of high color, loaded with urates, containing a small amount of albumin, is quite common and indicative of *passive congestion* of the kidney. It may result in cyanotic induration or interstitial nephritis. On the other hand, the urine may be of low specific gravity and pale in color. There may or may not be traces of albumin. The change is due to a granular, *contracted kidney*, which is associated with hypertrophy of the left ventricle and arterial sclerosis. Bloody urine is usually due to *renal embolism* when it occurs suddenly in the course of organic heart disease. It may be due to the emboli that are found in septic endocarditis. Renal disease in all forms may complicate disease of the heart. (See Kidney Disease.)

The Subjective Symptoms of Arterial Disease. The patient may have symptoms of congestion or of anæmia of the brain. Headache,

vertigo, photophobia, tinnitus, and paræsthesia, due to either cause, may prevail. (See also Cerebral Thrombosis.) The diseased vessels prevent the blood from reaching the extremities, hence they are cold. *Pain* is common only when atheroma or aneurism is present (*q. v.*). *Throbbing* or *pulsation* is complained of. It may be a striking feature of hysteria and neurasthenia. The abdominal aorta is frequently thus affected. The pulsation may be constant or intermittent. There may be dyspeptic symptoms. The pulsation of the carotids may cause disagreeable sensations in the head, and the beating transmitted to the ear be a source of extreme annoyance.

The Data Obtained by Observation.

Before describing the methods of observation it is well to review some of the facts of anatomy and physiology essential to the accuracy of any observations.

Topographical Anatomy. (Plate XIII.) Outline of Heart on Chest-wall.[1]

To have a general idea of the form and position of the heart, map its outline on the wall of the chest as follows :

(*a*) To define the base—*i. e.*, the part to which its great vessels are attached—draw a transverse line across the sternum, corresponding with the upper borders of the third costal cartilages ; continue the line half an inch to the right of the sternum and one inch to the left.

(*b*) To find the apex, mark a point about two inches below the left nipple, and one inch to its sternal side. This point will be between the fifth and sixth ribs.

(*c*) To find the lower border (which lies on the central tendon of the diaphragm), draw a line, slightly curved downward, from the apex across the bottom of the sternum (not the ensiform cartilage) as far as its right edge.

(*d*) To define the right border (formed by the right auricle), continue the last line upward with an outward curve, so as to join the right end of the base.

(*e*) To define the left border (formed by the left ventricle), draw a line curving to the left, but not including the nipple, from the left end of the base to the apex.

Such an outline shows that the apex of the heart points downward and toward the left, the base a little upward and toward the right ; that the greater part of it lies in the left half of the chest, and that the only part which lies to the right of the sternum is the right auricle. A needle introduced in the third, fourth, or fifth right intercostal space close to the sternum would penetrate the lung and the right auricle.

A needle passed through the first intercostal space close to the right side of the sternum would pass through the lung and enter the superior vena cava above the pericardium.

[1] From Holden : Landmarks, Medical and Surgical.

The best definition of that part of the præcordial region which is less resonant on percussion was given by Dr. Latham years ago in his "Clinical Lectures." "Make a circle of two inches in diameter round a point midway between the nipple and the end of the sternum. This circle will define sufficiently, or for all practical purposes, that part of the heart which lies immediately behind the wall of the chest and is not covered by lung or pleura."

Valves of the Heart. The aortic valve lies behind the third intercostal space, close to the left side of the sternum.

The pulmonary valve lies in front of the aortic behind the junction of the third costal cartilage with the sternum, on the left side.

The tricuspid valve lies behind the middle of the sternum, about the level of the fourth costal cartilage.

The mitral valve (the deepest of all) lies behind the third intercostal space, about one inch to the left of the sternum.

Thus these valves are so situated that the mouth of an ordinary-sized stethoscope will cover a portion of them all, if placed over the sternal end of the third intercostal space, on the left side. All are covered by a thin layer of lung; therefore we hear their action better when the breathing is for a moment suspended.

Physiology. *Action of the Heart.* The heart beats—that is, alternately contracts and dilates or relaxes—65 to 85 times per minute in an adult. In females, the frequency varies from 75 to 85; in males from 65 to 75. With each beat, blood is propelled throughout the vascular channels of the body, and drawn from them to the heart-chamber. The first effect is produced by the contraction of the heart, or the *systole*; the second by the relaxation, or *diastole*. Other events, as the act of respiration, contribute to the completion of the outflow and inflow of blood, particularly to the latter.

The completion of the act of contraction and the act of dilatation make up one revolution of cardiac action, or, as it is termed, a *cycle*.

Events of the Cardiac Cycle. The following events make up the cardiac cycle. The act of contraction is the systolic period of the cycle; that of relaxation is the diastolic period. During the *systole* (1) the ventricles contract; (2) the auriculo-ventricular valves close; (3) the blood is propelled from the ventricles into the vessels, the columns of blood in the aorta and pulmonary artery receive a shock from the impact of the new volume of blood, and their bulk increases. The movement of the blood-wave from this cause and from the contraction of the large vascular trunks produces pulsation of the peripheral vessels, which is known as the *pulse*. The contraction is immediately followed by *relaxation*—the *diastole*. (1) The blood-columns in the aorta and in the pulmonary artery fall back upon the valves guarding their outlets, the aortic and pulmonary valves. At the same time (2) the auricles are filled by the blood pouring in from the veins. (3) The auricular muscles contract upon the blood in the chamber, driving it into the ventricles.

The systolic and the diastolic periods of a cardiac cycle are nearly equal in the length of time occupied in their occurrence. The systolic period occurs at the same time, or is synchronous with the apex-beat

and carotid pulse, and precedes by a fraction of a second the radial pulse. It is immediately followed by the diastolic period, which, therefore, follows the carotid and radial pulse.

Inspection. The Heart. The Method of Examination. The patient should be stripped, and a good light should fall directly, as well as obliquely, on the surface. The patient can be examined in any position, and indeed for accuracy should be examined both in the upright and recumbent postures. This is particularly true when the pulse-rate is taken and when auscultation is practised. The sounds vary frequently in different positions. Some diagnostic significance is attached to these variations. It is necessary sometimes to have the patient lean forward, to bring the heart into more immediate contact with the chest-wall.

The examination should not be confined to the heart and vessels. The reader will remember that in the account of the exterior and of local areas it was pointed out that various abnormal conditions may be due to disease of the heart. In the examination, therefore, of a case of suspected heart disease, observation is made of the general and of the local color, as of the lips, the fingers, and the conjunctivæ, to determine the presence of *cyanosis, pallor, or jaundice;* of the feet, to discover *dropsy;* the face, to note the appearance of the countenance; the neck, to note the state of the vessels; the eyes, to note their prominence; the thorax, to ascertain the presence of dyspnœa.

The Præcordia. The præcordia is the region of the chest which overlies the heart. In the study of the appearance of the præcordia we observe: 1. The degree of *prominence* or swelling. 2. The *impulse* and other pulsations. 3. The *interspaces.* 4. The *hue* of the surface.

The Prominence. The præcordia may be unduly *prominent* in children who have had rickets and possibly some cardiac hypertrophy in childhood. It persists in later life. The ribs as well as the soft tissues are prominent. The lower end of the sternum may project. Swelling also occurs in hypertrophy or dilated hypertrophy of the heart, in pericardial effusions, localized pleural effusions and pointing empyema, and in aneurisms in the region of the heart. In pericardial effusion ribs and interspaces project. The latter are full or even with the surface. The prominence of cardiac disease is observed between the third and seventh ribs on the left side, and extends from the left nipple to the sternum, and even as far as the right nipple. The distance from the middle of the sternum to the mid-axilla is greater on the left than on the right side. Local bulging may be seen at the apex in cases of aneurism of the heart.

The præcordia may be *sunken.* Old pericarditis, but more frequently old empyema, causes sinking in of the region. It may be a result of *rickets* or of spinal curvature.

The Impulse. The normal impulse is that portion of the heart which strikes the chest-wall, and is improperly known as the *apex-beat.* It is evident in health in the fifth interspace just inside of the mid-clavicular line. It can readily be detected by inspection with a good light, in patients with moderately thick chest-walls. It is due to the

impulse of the right ventricle, three-fourths of an inch above the apex, against the chest-wall when the heart contracts, and hence it is *systolic* in time.

Changes of Position in Health. It is not a fixed point in health. It moves with the movements of the body, and hence, when the trunk is inclined to the left, the impulse falls toward the left axilla as far outward as the mid-clavicular line or even beyond that point. It moves toward the right and downward in full inspiration, or may disappear entirely toward the completion of that act. It may not be observed if there is a large amount of subcutaneous fat, or if the mammary gland intervenes. It becomes more conspicuous at the end of expiration or when the body is inclined forward. In children it is higher (fourth interspace) and more to the left. It is depressed in old people. It must be remembered that in transposition of the viscera the position of the impulse is changed.

Change of Position in Disease. The apex-beat, or the lowest point of impulse, may be *displaced* or may be *absent* entirely. These changes are due either to (*a*) *disease outside of the pericardium*, to (*b*) *disease within the pericardium*, or to (*c*) *disease of the heart itself.*

I. Displaced to the Left. This occurs from (*a*) *Alterations outside of the Pericardium.* When the right lung is the seat of extensive compensatory emphysema, or the right pleura is filled by a large effusion, the impulse is displaced to the left. On the other hand, fibroid phthisis of the apex of the left lung, or pleural adhesions which have become attached to the pericardial sac, with, probably, coincident pericarditis, pull the heart to the left, thereby changing the position of the impulse. In disease of the mediastinum the heart is pushed downward and toward the left. An aneurism, an abscess, or enlarged glands in this situation may invade the normal cardiac territory and cause dislocation of the heart.

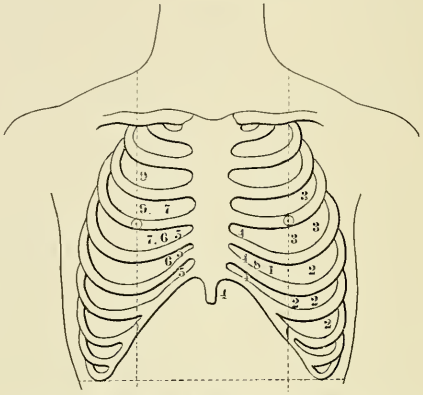
In disease of the abdomen the impulse is displaced. If the liver and spleen are enlarged, or the abdomen distended by ascites, the diaphragm is raised, and, therefore, also the heart. The impulse is then seen to the left of the normal position, and may be one or two interspaces higher than normal. A common physical change in the stomach—dilatation—is a frequent source of displacement of the impulse. The dilatation may be temporary from flatulency or may be due to organic disease.

(*b*) *Alterations within the Pericardium.* In cases of pericardial effusion the impulse is shifted to the left and upward. It is seen in the fourth and even as high as the third interspace, and sometimes only an impulse is noted in the second interspace. This, however, is not the true apex. Instead, we undoubtedly see in pericardial effusions the impulse of the right auricle and the conus arteriosus against the chest-wall.

(*c*) *Diseases of the Heart.* The impulse is diplaced to the left in dilatation and hypertrophy of the heart. In the latter it is also displaced downward. It may be as low as the sixth or seventh interspace and extend as far to the left as the anterior axillary or the mid-axillary line.

II. Displaced to the Right. (*a*) *Alterations outside of the Pericardium.* The heart is dislocated to the right in left pleural effusion, and in emphysema of the left lung. We find, moreover, in pleural contractions and fibroid phthisis of the right lung the heart drawn to that side. Under these circumstances the impulse is noted either in the epigastric region, along the margin of the ribs, or even

Fig. 152.



Normal and abnormal impulses.

1. Normal position of impulse. 2. Displacement to left and downward. 3. Displacement to left and upward. 4. Impulse from enlarged right ventricle. 5. Displacement to right. 6. Dilated right auricle. 7. Displacement in fibroid phthisis. 8. Impulse of conus arteriosus. (Errata: "8" should be in 2d interspace parasternal line.) 9. Fibroid phthisis, right lung.

to the right nipple-line, in any interspace from the third to the sixth, along the right edge of the sternum. The impulse in the epigastric region usually represents the hypertrophied right ventricle, which usually attends the lung-changes that cause displacement of the apex-beat. The impulse along the right edge of the sternum may be the apex-beat, or the right auricle and the right ventricle brought in apposition to the chest-wall by the cardiac dislocation. The apex or the tip of the heart is, in all probability, displaced but little beyond the mid-sternal line. (*b*) The impulse is not displaced to the right in *alterations within the pericardium*, or (*c*) in *disease of the heart.*

III. Absent. Following the same order, we find that the impulse may be absent entirely in (*a*) disease *outside the pericardium*, on account of which something intervenes between the heart and the chest-wall. Hence, in emphysema of the lungs and in compensatory emphysema of the left lung the impulse is entirely effaced; in (*b*) *disease of the pericardium* the impulse is absent when there is large effusion. The absence here succeeds the dislocation to the left, and with its effacement the impulse in the second and third interspaces disappears. In (*c*) *disease of the heart* the impulse is absent when the heart is diminished in size, as in atrophy, or in myocarditis, or when weakened by fatty degeneration or dilatation

The Extent of the Impulse. In *health* the impulse is limited in extent to about one square inch. The area of impulse may be in-

creased when the individual leans forward, and at the end of expiration. It is more evident when the chest-walls are thin, and less when they are thick.

Extent in Disease. The area of impulse may be *increased.* The causes are: (*a*) *Diseases outside of the pericardium.* The area is increased in chronic phthisis with fibrous adhesions, and in pleural adhesions when the lung is drawn away from the surface of the heart. It is increased when the heart is pushed against the chest-wall, as in aneurism or in diseases of the mediastinum, from inflammation or cancer, or other mediastinal growth. The impulse is seen not only in the third and fourth interspaces, but also as high as the second, and is not limited to the spaces between the sternum and parasternal lines, but may extend beyond the mid-clavicular line. It may not be systolic in time only, but diastolic, presystolic, and systolic, and have the appearance of a peristaltic wave from base to apex. The time coincides not only with contraction of the ventricles, but also of the auricles, and of the closure of the semilunar valves. (*b*) *Disease of the pericardium* tends to increase the area of impulse if moderate effusion is present. It will be seen as a diffuse wave occupying the second, third, and fourth interspaces. It is also increased in pericardial adhesions, without increase in strength. (*c*) *Disease of the heart.* The heart must be enlarged, and hence must either be hypertrophied or dilated. The extent of impulse varies. In hypertrophy the impulse may be communicated to the sternum, so that the lower part heaves with each contraction. It falls below the fifth interspace and toward the left, particularly if the left ventricle is the seat of the enlargement. If the right ventricle is hypertrophied, the impulse is very marked in the third, fourth, fifth, sixth, and even the seventh interspaces near the termination of the cartilages, or in the epigastrium along the border of the ribs of the left side. It may be seen in anæmia in this situation, particularly in persons whose respirations are habitually shallow. Sometimes, when associated with and displaced by lung disease, it is seen to the right of the xiphoid cartilage.

New Impulse. New areas of impulse, the heart not dislocated, arise from enlargement of one of the cardiac chambers or from disease of the bloodvessels. A new area of impulse in the second or third interspace on the left is from the conus arteriosus, or is due to hypertrophy and dilatation of the right ventricle; or it may be due to retraction of the lung in that region. It may be due to a dilated right auricle, and is then seen in the fifth right interspace along the sternum. If the impulse is noted in the course of or adjacent to the aorta, it is indicative of aneurism.

The Interspaces. They are retracted possibly from pericardial adhesions; they are full or bulging in effusion. This retraction may be limited to the apex or may occur in each interspace over the præcordial region. It may occur with the systole or with the diastole. It may occur in hypertrophy of the heart, and is then systolic in time. It is of some, although doubtful, diagnostic significance when it is systolic in time, as it is said to indicate adhesions of the pericardium. The traction at the systole of the heart causes the interspaces to be drawn in.

On inspection behind, a systolic retraction of the interspaces is seen in adherent pericardium, known as *Broadbent's sign.*

Color of Surface. Only when purulent pericardial effusion is about to rupture, or an empyema to discharge, do we note redness or other change in hue of the surface of the præcordia, not observed over the remainder of the thoracic surface.

The Arteries. By inspection we may be able to determine pulsation or any undue swelling or other change in the course of the vessels. With the exception of pulsation in the carotids, which may temporarily increase under excitement, pulsation of the vessels is not usualy seen in health. In old people we can see the pulsation of the aorta (rarely) at the episternal notch, and often in others, the temporals, the innominate, the carotids, the subclavians, the brachial and radial arteries, the abdominal aorta in thin subjects, the femoral arteries and the posterior tibials.

The Arteries in the Neck. Temporary pulsation of the carotid arteries from excitement has been mentioned. It is commonly seen in anæmia, and is quite marked in exophthalmic goitre. It is striking in aortic regurgitation. It often attends the vascular changes of old age. It may be due to atheroma or aneurism. It is always suggestive of aortic valvular disease. The innominate artery, as well as the carotids, often pulsates visibly in the neck, and may be so large as to simulate aneurism. The subclavians may pulsate for the same reasons; they may also be seen to pulsate if the lungs are consolidated or shrunken by disease. If the patient is young, the throbbing is more likely to be of neurosal or hæmic origin. In later life, if such pulsation is associated with a more or less defined swelling or tumor, with other physical signs of aneurism, that disease is doubtless present.

The Thoracic Aorta. An impulse of the *thoracic aorta* is usually from aneurism. The pulsation is not always due to disease. The aorta may be pushed against the chest-wall, or the lung-structure which overlaps it normally may be withdrawn.

Tumor. An enlargement or swelling in the course of the aorta may be due to aneurism of that vessel. It must be distinguished from the tumor of mediastinal disease, and of empyema.

The Abdominal Aorta. Pulsation of the abdominal aorta is often the cause of serious distress. The violent throbbing keeps the patient awake at night, and makes him more and more nervous and irritable. The pulsation is usually seen in the epigastrium. It is more frequent when the vessel is not diseased, in neurasthenic subjects. It occurs reflexly in patients with dyspepsia or organic disease in the upper abdominal tract. The shock of the pulsation is transmitted to the hand with considerable violence. The impulse is diffused, but not expansile.

Epigastric pulsation also may be due to the transmission of the impulse of the aorta by enlargement of the pancreas, or tumors of the stomach or the omentum. The transmitted pulsation is distinct. The impulse is a transmitted one when the tumor can be defined and when a sensation of lifting is transmitted to the hand. The physical signs of aneurism are absent. If the patient lies on the abdomen, or in the

knee-chest position, the tumor falls away from the aorta, and the impulse is not readily transmitted. Epigastric pulsation is also caused by aneurism of the abdominal aorta. The pulsation is distensile or expansile, and the aneurismal sac can be defined at times. The other physical signs of aneurism are usually present—namely, thrill, dulness over the tumor, a murmur on auscultation. In these conditions, however, we cannot always rely on the physical signs alone; the history of the subjective symptoms and of disease of other structures must be carefully inquired into. Aneurism rarely occurs without some evidence of arterial sclerosis or some physical effect upon the circulation. Accentuation of the aortic second sound, variations in the femoral pulse, high arterial tension, and the usual evidences of sclerosis favor aneurism. While functional epigastric pulsation usually occurs in neurotic subjects, and, hence, in the earlier periods of life, yet such pulsation is frequently seen at the climacteric and in the neurasthenia of old age. Late in life, with such impulse, fibrous thickening about the pylorus, or contraction of the omentum, may easily be confounded with malignant disease. Cancer of the stomach has been diagnosticated under these circumstances when the pulsation was simply reflex from chronic gastritis. Some time ago a private patient in the Presbyterian Hospital had extreme pulsation of the abdominal aorta, with great local discomfort, on account of the throbbing. She was sixty-five years of age, and had within the past two years nursed her son through tuberculosis. She failed in health, and came to the hospital emaciated, with some chronic gastritis and diarrhœa. On examination, a distinct tumor was felt above the umbilicus, which she had been told was due to carcinoma. It was hard and painless; the physical signs of aneurism were not present; the pulsation was extreme. A second tumor, not so large, was felt in the right hypochondriac region. Both tumors were dull upon percussion and surrounded by tympanitic areas. They were also movable. While it was impossible to be sure of the nature of the tumors, it seemed to me they were tuberculous, or simply fibrous, and would not influence the patient's immediate welfare. Under treatment, the pulsation disappeared; the gastro-intestinal symptoms were relieved entirely; the patient rapidly gained in weight and strength; the tumors continued, but they are not so distinctly outlined because the previously scaphoid abdomen has become distended (two years under observation). The questions arose for decision: Was the epigastric pulsation due to a throbbing aorta or transmitted by an obscurely defined probable tuberculous mass in that region? No doubt it was the vessel alone that caused the impulse. The diagnosis must be made by carefully weighing all concomitant circumstances and phenomena that surround cancer. (See Symptomatology of Morbid Processes.) *Fecal accumulations* in the colon may be made to heave by the beat of the aorta and cause exaggerated epigastric impulse. The bowels must be emptied before definite conclusions are arrived at.

An epigastric impulse due to one of the above-mentioned causes must not be confounded with the impulse of hypertrophy of the right ventricle, or to the shock of the hypertrophied heart transmitted to the left lobe of the liver. In hypertrophy of the right ventricle or

dislocation of the heart from disease within the chest, the impulse may be seen to the right or left of the xiphoid cartilage. The symptoms and signs of right-ventricle hypertrophy explain the pulsation.

The Smaller Arteries. By inspection of the arteries beyond the abdominal aorta we can often recognize more distinctly the condition known as arterio-sclerosis. Examination of the femoral, popliteal, tibial, brachial, and radial arteries reveals dilated, tortuous, hard, often pulsating vessels in endarteritis. Elongation of the artery, so that instead of a straight tube it becomes a sinuous canal, turning and twisting at short intervals, is seen. (See Arterio-sclerosis.) But pulsation of the above-mentioned peripheral arteries may be due to other causes. In hypertrophy of the left ventricle arterial pulsation is prominent, although more marked in the vessels near the heart, as the carotids. In regurgitation at the aortic orifice, pulsation is also frequently seen.

Capillary Pulse. The capillary pulse is seen under the finger-nails or in the skin after hyperæmia is induced by firmly stroking the skin with the nail. It may be seen inside the lips, if a piece of glass is pressed against them. There is rhythmical pulsation of the capillaries, from which the surface becomes alternately white and red. It is a sign of aortic insufficiency.

The Veins. Diseases of the veins are largely surgical and do not frequently come under the notice of the physician. Alterations in the veins from physical causes in the circulation, local or general, are of frequent occurrence, and are of the greatest diagnostic significance. The "venous phenomena" are physiological and pathological evidences of the circulation of the blood in the veins.

Examination is limited largely to the jugular veins in general affections of the circulation; to other subcutaneous veins in addition in local affections. The examination is made by inspection, to determine the size and degree of pulsation of the veins; by palpation, to confirm the results of inspection and to determine the presence of a thrill; by auscultation, to determine the presence of murmurs.

By *inspection* we note the presence of: A. *Enlargement* of the veins. The change in size may be general or local. In both instances there is interference with the venous return of blood.

1. *General enlargements* may be observed in all the veins, but is more readily studied in the *jugular veins* of the neck. Associated with the enlargement, general venous engorgement is observed, and hence œdema (which obscures external veins), cyanosis, effusions in serous cavities, and congestion of internal organs attend the pathological venous phenomena. It must follow that a central disturbing influence upon the circulation is present, and so we find interference with the circulation in the right heart to be the causal factor. This interference is due to dilatation of the right auricle and ventricle, which in turn may have arisen from valvulitis, myocarditis, pericarditis, or, on account of increased pulmonic blood-pressure, from emphysema and other pulmonary obstructions. In rare instances pressure upon the cavæ by a mediastinal tumor may cause general over-fulness of the veins.

The jugular veins, both internal and external, are seen to be distended, even in stout people. The observation can better be made by viewing the head when it is turned to the opposite side from the vein which is under examination. The external jugular can almost always be seen; the internal jugular frequently when engorged. They may also be felt under these circumstances. The position of the veins can be more readily distinguished by observing their relation to the sterno-cleido-mastoid muscle. The internal jugular vein is seen in the inter-sterno-cleido-mastoid fossa, just behind the sterno-clavicular articulation. Here the jugular bulb is seen, and at this point in the veins the bulbar valves are situated. When abnormally full it may project beyond the surface and rise one-fourth or one-half inch above the articulation. The over-fulness is more marked in the dorsal than in the upright posture.

Local Enlargements. Local increase in fulness of the veins is due to narrowing or closure of the venous trunk by pressure or by thrombosis. A mediastinal tumor pressing upon the cava will cause abnormal fulness of the *jugulars*. The veins of the *scalp* become distended and tortuous in thrombosis of the longitudinal sinus. Enlargement of the veins of the *arm* or *leg* points to compression or thrombosis of the axillary or femoral vein respectively. The enlargement is associated with œdema of the respective extremity. Enlargement of the superficial veins of the *thorax* is seen in intrathoracic pressure from tumor or aneurism, rarely in dilatation of the heart. Enlargement of the veins of both *legs* may be due to obstruction of the vena cava or both iliac veins. The latter is liable to occur in pelvic tumors. When there is engorgement of the portal vein collateral circulation is frequently carried on through the *abdominal veins*. The veins are enlarged; and, in some instances, the veins about the navel enormously distended, because of a permanent patulous umbilical vein. The crown of veins—*caput Medusæ*—is significant of cirrhosis of the liver and of pylo-thrombosis. Enlargement of the veins of the extremities, from the causes above mentioned, must not be confounded with the unilateral or bilateral varicosity that occurs during and after pregnancy, after prolonged intra-abdominal pressure from other causes, or in inflammation of the veins in the course of septic diseases, as typhoid fever.

B. *Pulsation* of the veins. The circulation in the veins differs from that in the arteries. The blood-flow is continuous. Two circumstances modify it—respiratory movements and cardiac action.

Pulsation due to Respiratory Movements. The modification is particularly seen in the veins of the neck. During inspiration all of the veins empty rapidly, while in forced expiration, or with strong effort, as seen in coughing, the discharge from the veins is checked and they become full and even over-distended. When the fulness of the veins is normal the respiratory alterations are not observed, except the swelling that occurs in severe coughing, as in whooping-cough. When they are abnormal, as from right-sided cardiac dilatation (*q. v.*), they show a corresponding to-and-fro swelling synchronous with respiratory movements. Upon coughing, the jugular bulb may appear as

a rounded pulsating bunch between the heads of the sterno-mastoid muscle. The internal jugular may also swell and contract. Increased pulsation with fulness of the veins is seen during the labored expiration of asthma and emphysema.

Alteration of the respiratory movements of the veins is observed in cases of pericarditis or of mediastino-pericarditis. Normally the vessels are drawn upon and bent during the act of inspiration—inspiratory collapse. In the above pathological conditions they swell up in inspiration and empty during expiration, directly opposite to the normal state.

Pulsation due to Cardiac Movements. The Venous Pulse. The cardiac movements also modify the movements of the blood in the veins. They cause rhythmical pulsation, or the venous pulse. This may be communicated from the carotids underneath or occur in the veins. The so-called *true* and *false* pulses are thus produced. The true venous pulse is divided into the (1) *negative* and (2) *positive* pulse, the former being the pulse of health, the latter the pathological venous pulse.

1. *The normal or negative venous pulse* is so designated because it is not due to positive action of the heart, causing retrogression of blood. It can be demonstrated by pressure of the finger on the middle of the veins. Pulsation ceases below because the blood does not regurgitate from the heart; it does not pulsate above, or the pulsation lessens materially, indicating non-transmission from the carotid. The negative venous pulse is presystolic in time, and can only be seen in the external jugulars. The vein collapses during the systole and distends or pulsates before the systole, hence is presystolic. This may be observed by inspection, keeping in view also at the same time the apex or carotid pulse. The systolic collapse occurs quickly. The presystolic pulsation follows slowly, with an appreciable interval between the two. The presystolic distention occurs during the time that the auricle is filled with blood; the collapse occurs when the auricle is empty —that is, during the ventricular systole. When the auricle is distended the flow of blood from the veins is impeded, and hence the jugulars are overfilled. When the auricle is empty the flow of blood from the veins is favored, hence the vein collapses (the systole).

Diagnosis It may be distinguished from pulsation in the artery by the time, by the greater size of the surface-pulsation on account of the greater size of the vein, by the impression of undulation rather than shock received by the finger, by the impression of passive force rather than of active power. Sometimes it is extremely difficult to recognize the normal or negative venous pulse on account of undulations in the veins produced by the blood-flow and transmitted carotid impulse.

2. The *positive venous pulse* is systolic in time. It is due to positive action of the heart. It is pathognomonic of tricuspid regurgitation (*q. v.*). When the right ventricle contracts the regurgitant blood-wave is transmitted into the cava through the incompetent valves. It appears first in the internal jugulars or their bulbs, because of the direct course of the innominate and right jugular from the cava. Subsequently the left may become affected. If the valve in the vein is competent, the systolic regurgitant wave is seen there only. The pul-

sation of the enlarged bulb is seen in the inter-sterno-cleido-mastoid fossa. Usually the valve is insufficient, or rapidly becomes so, and the systolic back-wave therefore extends upward. The same wave is transmitted to the veins of the liver, causing systolic swelling and diastolic collapse of the liver. These conditions are produced, as previously mentioned, in right-sided dilatation of the heart, providing there are moderate force and slowness of the heart's action. When the heart becomes very weak and rapid the pulsations disappear.

Diagnosis 1. The negative, true, or normal pulse is distinguished from the pathological or positive pulse, and from the transmitted pulsation, by its time. It is timed by the apex-beat, or the carotid pulse of the opposite side. The negative pulse (normal) is presystolic, the collapse of the vein systolic ; the positive pulse (pathological) is systolic in time. The patient should hold his breath, as increased respiratory movement will modify the venous pulsation. 2. The imparted or *false pulse* is transmitted from the carotids, and can be recognized by stopping the flow of blood by pressing the finger or barrel of the stethoscope on the vein in the middle of the neck, after it has been emptied by pressure upward. If the pulsation is communicated (false pulse), the vein remains empty in the portion nearest the heart, and fills up in the peripheral portion, while the pulsation ceases toward the centre (below) and increases in the periphery (above the finger). If the carotid artery is pressed upon as near the heart as possible, the transmitted pulse will cease. In the positive pulse the portion near the heart slowly fills from below upward.

In congenital heart disease with patulous foramen ovale the positive venous pulse may sometimes be seen, but is extremely rare.

Diastolic collapse is seen in pericarditis, as observed by Friedreich. The collapse occurs at the time of the cardiac diastole. It is distinguished from the true pulse as follows : compress the jugular vein, pulsation ceases above and below the seat of compression.

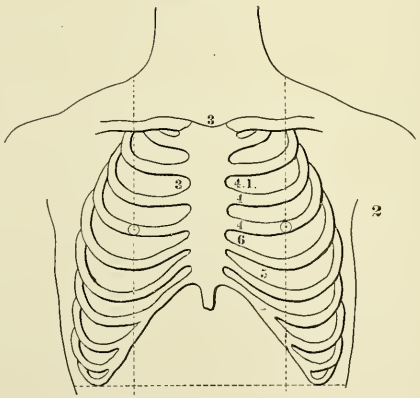
Pulsation of other veins. Quincke has described venous pulse in the hand and back of the foot, with the capillary pulse in aortic regurgitation and in anæmia. It is probably only the arterial pulse propagated through the capillaries. The positive pulse may be seen in the veins of the face, in the cutaneous veins of the arm and hand, and in the superficial mammary veins, and in the veins of the legs.

Palpation. The Heart. Palpation confirms inspection as to the shape of the præcordia, the position and the extent of the impulse, and the condition of the intercostal spaces. In addition, we determine by palpation the character and strength of the *impulse*, and the presence or absence of *valve-shock* and of *thrills* or of *friction*. Palpation also reveals *œdema* of the surface and *fluctuation*.

The Impulse. In a normal chest with moderate walls a slightly prolonged, moderately strong shock is transmitted to the hand when placed over the præcordia. It is synchronous with the cardiac and precedes the radial pulse. It is, therefore, systolic in time. It is stronger when the patient leans forward, exhales freely, removing the lung from the surface, and when the chest-walls are thin ; it is weaker in opposite conditions.

Character and Strength of Impulse. A. Strength *increased.* 1. Overaction. In the violent action of the heart that attends palpitation, and in the increased action in the early stages of fevers or of inflammation, the force of the cardiac impulse is much increased. 2. Disease. (*a*) *Alterations outside of the pericardium.* Increase in the extent of the impulse is attended by increased strength when the heart is hypertrophied or the lung retracted. (*b*) *Alterations within the pericardium.* In pericardial adhesions the heart is held more firmly against the wall and may give the appearance of strength to the impulse. (*c*) *Disease of the heart.* True increase in force of the impulse is seen in disease of the heart. When the organ is *hypertrophied* or the seat of dilated hypertrophy the force of the impulse is increased, sometimes to an almost unbearable degree. Uplifting of the præcordial area or even of the lower half of the anterior part of the chest is seen. The hand or the head laid over the heart is forcibly lifted with each systolic contraction. This great force is most pronounced in the enormous hypertrophy that occurs in cases of aortic obstruction. It is the impulse and force of the so-called *cor bovinum.* In *dilatation* the impulse is diffused and wavy.

Fig. 153.



Abnormal palpable impulse and thrills.

1. Diastolic impulse palpable from closure of pulmonic valve. 2. Presystolic impulse in mitral obstruction in third, fourth, and fifth interspaces. 3. Thrill at aortic orifice; systolic, obstruction; diastolic, regurgitation. 4. Thrill at pulmonary orifice; systolic, obstruction; diastolic, regurgitation. 5. Thrill at mitral orifice; systolic, regurgitation; diastolic, obstruction; presystolic, obstruction. 6. Thrill at tricuspid orifice.

B. Strength *lessened.* This occurs from causes which diminish the extent of the impulse or cause it to be absent entirely, as when material intervenes between the heart and the chest-wall, or the heart is weakened by disease. Hence (following the classification above) (*a*) in emphysema of the lung; (*b*) in pericardial effusions; (*c*) in fatty heart, or myocarditis, in dilatation, and simple weakness of the heart, the strength of impulse is lessened.

Valve-shock. The shock of the closure of the valves can be felt by the hand when placed evenly over the præcordia. The shock from

the pulmonary and aortic valves is best transmitted. It is felt most distinctly in persons with thin chest-walls, and when there is heightened tension either in the aorta or pulmonary artery. The shock follows the impulse. It may be localized more accurately with the finger-tips in the third or fourth interspace along the left edge of the sternum. The shock of the auriculo-ventricular flaps is also transmitted. The shock is synchronous with the first sound. It is felt in the left fourth interspace near the sternum, sometimes over it. It is due to dilatation of the heart, and is more readily felt in thin-chested persons.

Thrills. A thrill is produced when the blood is thrown into vibration by passing over a rough surface. It may be created with the systole or during the diastole. It can only be created at the time blood is passing through the orifices. 1. The most common seat of the thrill is the apex. If the hand is placed in close proximity to the surface of the chest at this point, a vibration or tremor is transmitted to it in most cases of *mitral obstruction.* The blood is passing from the auricle to the ventricle ; as this takes place before the systole, the thrill is felt before the impulse or carotid pulse. It is *presystolic* in time. It is sometimes difficult, however, to distinguish it from the impulse. Its character cannot well be described. The hesitating, jogging manner of the vibrations or the thrill is clearly transmitted to the hand. 2. The next most frequent seat of thrill is the second costal cartilage on the right. Here the thrill or vibration is *systolic* in time and is caused by *obstruction* at the *aortic orifice.* It may be felt away from the heart, in the aorta, or in the carotids. The aortic cusps are thickened, contracted, and stiffened by a sclerotic endocarditis, or the orifice is occluded by valvulitis. 3. Sometimes a thrill is felt at the apex with the systole—*first sound.* This occurs rarely, but must not be confounded with the before-first-sound thrill. It is never so distinct, and is not made up of a series of vibrations. It is due to regurgitation at the mitral orifice. 4. Rarely a thrill is felt at the second costal cartilage on the right, with the *second sound.* It may be felt along the course of the sternum also, and is due to regurgitation through the aortic orifice. The systolic thrill must not be confounded with the thrill elicited over the aorta or at the aortic cartilage, which is due to aneurism. 5. At the second costal cartilage on the left a thrill is sometimes felt. It is *systolic* in time and is not transmitted. It is due to obstruction at the pulmonary orifice. 6. At the lower portion of the sternum a thrill systolic in time is also felt, due to tricuspid regurgitation. Care must be taken not to confound the above-mentioned thrills with those due to aneurism. (See Aneurism.)

Pericardial Friction. In addition to the thrills, a friction or to-and-fro rubbing is transmitted to the hand in cases of pericarditis, in the first stage. The friction may be felt all over the heart region, but is pronounced in the third or fourth interspace. It may be detected on slight pressure or only when the tips of the fingers are pressed firmly against the interspaces.

It is important to remember that the *position* of the patient weakens or modifies the thrill or friction. When the patient is lying down it

may not be felt. The upright posture or leaning forward makes it evident, and hence the patient should be instructed, if possible, to assume this position in the examination.

The Arteries. The results of inspection are confirmed. In addition, the artery is examined, to determine its tension, the character of the coats, and the presence of thrills. *Pulsation of organs.* It is said that in aortic regurgitation an arterial liver-pulse, similar to the venous liver-pulse, can be felt when the hands are placed over that organ. Similar pulsation may be felt in the spleen.

In examining the arteries it is important, as will be detailed in the chapter devoted to the pulse, to compare the arteries of the two sides. Often the pulse-wave is found to be unequal in force, in volume, and in time. This is almost always due to obstruction to the passage of the blood. When not due to *endarteritis* or to *aneurism*, it is due to the pressure of a tumor on the vessel somewhere in its course. A thrombus or emboli in the artery may likewise cause the condition. A difference in the radial and the femoral pulse points to obstruction in the thoracic or abdominal aorta. Anatomical variations must be remembered.

The Pulse. The pulse is an index to the force, frequency, and rhythm of the heart's action and of the pressure, or tension, which is maintained in the arteries.

General Observations. The *frequency* of the pulse before birth is from 120 to 140 beats in the minute. From this time it is diminished in frequency up to adult life, 72 being then accepted as an average; the number of beats, however, is often under 72, and sometimes over that. In old age the pulse-rate is again increased. Sex has some influence. The rate is slightly higher in females than in males of the same age.

The frequency of the pulse is subject to diurnal variations, at times corresponding with the diurnal rise and fall of temperature. The rate will, therefore, be highest in the afternoon and evening and lowest in the early morning hours.

The position of the body has also a modifying influence. The pulse is more frequent when a person is standing than when he is sitting, and more frequent when he is sitting than when he is lying down. Walking, running, bodily and mental exertion, fear, and excitement all tend to accelerate the pulse.

During and for one or two hours after a meal the pulse-rate is higher, especially if an alcoholic or other stimulant, such as coffee, has been taken.

How to Take the Pulse. To make a correct count of the frequency of the pulse, the conditions just mentioned, as normally modifying its rate, should be borne in mind. If the object of the count is to determine the rate which is normal for a particular individual, several counts will be necessary at different times and under different conditions, such as sitting and standing. The best time for the physician to take the pulse will have to be determined by his own judgment in each case. If the patient comes to his office and is excited by the prospect of an examination, it will be well to wait until he becomes

calm. On the other hand, if he is calm at first, a count at that time is to be preferred to one made after the patient has been disturbed by a physical examination. In the same manner, on visiting a patient at his house, the judgment of the physician must decide whether to count the pulse immediately on his arrival or to postpone it until, by general conversation, all apprehension and alarm on the part of the patient have been allayed. In general, it may be said that if the physician finds upon his arrival that the pulse is more frequent than the condition of the patient would lead him to expect, he should wait a while, endeavor to find out whether anything has served temporarily to disturb the circulation, and then make the count when the conditions are most favorable. Some patients are so nervous that the mere act of placing the finger upon the wrist sends the pulse-rate up ten or twenty beats in the minute. In such cases an effort should be made to obtain a count without the patient's knowledge by observing the pulsations of the temporal or carotid. In other cases it may be well to entrust the counting of the pulse to the nurse or to a member of the family. In infants and young children, count while they are asleep. In febrile conditions the count is more likely to be too high than too low.

In hospital practice, or when a nurse is constantly in attendance, the pulse and respiration should be taken at the same time as the temperature. But the nurse must be warned against taking them under dissimilar conditions upon successive days. For example, the pulse should not be taken one day while the patient is lying down, quiet and comfortable, and compared with the count of the next day when the patient is sitting up or has just had some hot liquids, or a spell of coughing, or been subjected to some other disturbing influence.

The preferable position is the recumbent one in the case of patients in bed, and in the sitting position in those not confined to bed. Care should be exercised in all cases to see that the patient's position is comfortable and that nothing obstructs the artery or interferes with the unimpaired flow of the blood.

The wrist is the place usually selected at which to feel the pulse. At this point the radial artery passes over the radius, and can readily be compressed and its character made out. An old-fashioned rule prescribes that three fingers should be applied to the artery, the index-finger of the physician being nearest the heart. In particular cases it may be advisable to count the pulse at the temporal or carotid artery. The fingers should be applied so that the beats can be most distinctly felt. The beats are counted for fifteen seconds by the second hand of a watch when only an approximate count is desired, or when time is a factor, and then multiply by four. It is better to count the pulse for half a minute, and still better for a full minute.

The arteries of the two sides must be compared. Difference in the force, volume, and time may be due to the anomalous distribution of arteries. In disease, it may occur in aneurism and atheroma, in pressure on the trunk from external disease, and in embolism and thrombosis.

Condition of the Walls of the Artery. The condition of the artery is often of more importance than the pulse-rate. A healthy

radial artery, in a person not advanced in years, can be compressed easily against the radius without the finger being able to differentiate the artery from the other tissues. But as age advances, and as the result of certain constitutional diseases—syphilis, gout, chronic endarteritis, alcoholism, and others—the artery tends to become thicker, so that in pronounced cases it cannot be obliterated, but is rolled like a cord or pipe-stem between the compressing fingers and the bone. Small specks or plates of atheroma, feeling like hard particles in the coats of the artery, may be detected. The artery has a beaded feeling. Fatty degeneration of the organs is likely to occur when the arteries are in this condition, and apoplexy is to be feared.

Tension. Tension is the word used to express the degree of blood-pressure—that is, of distention of the arteries. Normally, the pulse nearly or quite subsides between the beats, but little pressure being required to obliterate it. *High tension* may be said to exist when the artery remains continuously full between the beats (Broadbent). It is produced by plethora; increased heart-action; contraction of the arterioles, as by chill; and obstruction in the capillaries. The conditions which bring about obstruction in the capillaries in the order in which they are enumerated by Broadbent are: 1. Age. The liability to high arterial tension increases with the age, especially after middle-life. 2. Heredity. There is in some families a marked tendency to high tension. The younger members show its effects in headaches and bilious attacks, while the older ones develop chronic heart disease and apoplexy. 3. Disease of the kidney. Parenchymatous, but especially interstitial nephritis, is associated with high arterial tension; this, with accentuation of the aortic second sound, is one of the early and, therefore, one of the most valuable indications of chronic Bright's disease. 4. Gout. Gout and lithæmia are almost always accompanied by high arterial tension. 5. Diabetes in old persons associated with gout. 6. Lead-poisoning. 7. Pregnancy. 8. Anæmia. 9. Emphysema and chronic bronchitis. 10. Mitral stenosis.

As regards arterial tension in persons presenting signs of angina pectoris, Sansom asserts that if the tension is increased, even though the signs are not typical, the fear, present or remote, of true angina is justified. On the other hand, if there is persistent low tension, especially during the painful crisis, it is almost certain the affection is a false angina.

Low tension of the pulse is characterized by a softness and a compressibility in excess of the normal. This, like the high tension pulse, may be a family peculiarity. It is met with in conditions of great depression and exhaustion, and wherever there is a marked cardiac weakness. It is most common in fever, particularly in typhoid, in which also an accompaniment of low-tension pulse—namely, dicrotism—is met with in a marked degree. Fat persons are apt to have low-tension pulses, and it may occur in any person temporarily under the influence of external warmth and moisture, such as a hot bath, or after taking hot drinks, or under the influence of depressing emotions, and after diarrhœa, or copious urination.

Volume. The volume of the pulse should be noted. It is usually large in conditions of pyrexia and when the tension is low. A small

pulse is met with in many conditions other than weakness of the heart-muscles. In aortic stenosis the pulse is small, and in mitral stenosis it is small, of high tension, and frequently irregular. In general contraction of the arterioles, as happens under the influence of a chill, the pulse is small. In Bright's disease it is sometimes very small, slow, and hard. Some care will be required to differentiate such a pulse from a weak pulse. In acute peritonitis the pulse is apt to be small and hard.

Rhythm. The rhythm of the pulse is of diagnostic importance. In health one beat succeeds another at equal intervals of time, and the successive beats are of the same force and quality. Here, also, however, as in other conditions, there are variations within physiological limits. In some persons the pulse-rate is somewhat accelerated during respiration and becomes slower in the pauses which follow breathing.

In disease, disturbance of the rhythm occurs as intermission or as irregularity. *Intermission* signifies a dropping of a pulse-beat; several normal pulse-beats succeed each other, and then the pulse is absent during the time occupied by one or two beats. The intermission may occur at regular or at irregular intervals—that is to say, every third, fifth, or sixth beat may be wanting, or the intermission may be irregular—now a second, the next time a fifth or a third beat being absent. Moreover, the intermittent pulse may be constant, or it may, and more frequently is, only occasional. It is not characteristic of any one disease or condition, and it may exist without the patient's knowledge and without producing any perceptible effect upon his health. Sometimes it is met with in a fatty heart, and this disease may be suspected if the intermittent pulse is associated with a weak first sound of the heart without valvular lesion, and evidences of failing circulation, such as œdema of the feet. More frequently, however, the intermittency is a symptom of nervous depression, or is caused by tea, coffee, tobacco, or digitalis. So far as prognosis is concerned, it is much less serious than irregularity. Broadbent says he has met with it at the age of eighty, when it was known to have existed for forty years.

Irregularity is characterized by differences in time, force, or volume of successive beats. A full beat is succeeded by another, which is smaller and weaker, or successive beats occur at irregular intervals of time. Irregularity may or may not be associated with intermission. In advanced cases of mitral stenosis the pulse is both irregular and intermittent. The irregularity may be habitual or occasional; the former is due most frequently to mitral lesions, but sometimes occurs without assignable cause, and is attributed to disturbance of the nerve-supply; the latter is due to digestive disturbances and to the effect of nicotine and digitalis. Irregularity is not incompatible with health, but is much more likely to be of serious import than intermission. It occurs in diseases of the brain, in degeneration of the heart as well as in valvular lesions, and in grave cases of febrile diseases, such as typhus and typhoid, when the heart-muscle is involved. Some cases of Graves' disease are characterized by great irregularity instead of excessive rapidity of the pulse. Irregularity may occur in rheumatoid arthritis also, though increased frequency is the rule.

Frequency. The *frequency* of the pulse is of aid in diagnosis. *Increased frequency.* 1. The pulse is *increased* in frequency in all the febrile diseases, and generally in the proportion of eight to ten beats for each degree of rise in temperature above 98.3°. But there are important exceptions. In *typhoid fever* the pulse is slower in proportion to the temperature and the gravity of the disease than in most of the other acute febrile diseases. It may not beat above 85 in mild cases, and in severe cases frequently does not rise above 100. Consequently a pulse of 120 is of much graver import than it would be in other diseases. It may be more frequent during convalescence than during the febrile stage. This pulse-rate helps to differentiate it from tuberculosis, malignant endocarditis, and septicæmia.

2. The pulse of *scarlet fever* often aids materially in diagnosis. A pulse of 120 to 160 is the rule from the development of the sore-throat to the completion of the eruption. In measles, rubella, diphtheria, and follicular tonsillitis it is much slower during the early stages.

3. In *Graves' disease* great frequency of the pulse is the essential and most constant symptom of the disease. The pulse may be constantly considerably over 100, and in attacks of palpitation 200 or more. In these attacks there may or may not be præcordial distress and mental anxiety. Here belong the cases described as paroxysmal hurry of the heart, etc., the thyroid and ophthalmic symptoms being absent.

4. Cases have been reported of extreme frequency of the pulse (160 to 240) without palpitation, dyspnœa, or any signs of Graves' disease. Some of the patients have been able to perform much bodily and mental labor, notwithstanding that the rate mentioned was maintained persistently for weeks. To this class of cases the name *tachycardia* has been provisionally applied until their pathology is understood.

5. In all forms of valvular disease, except aortic stenosis with failing compensation, the pulse may be increased in frequency. In collapse ; in weakening of the heart ; and in central or peripheral vagus disease, the pulse is increased. *Mitral stenosis* may be latent until great excitement, overexertion, and particularly running or forced marches bring on palpitation, or simply abnormal and persistent frequency of the heart's action, with or without dyspnœa.

6. Attention has been called, especially by Dr. J. Kent Spender, to acceleration of the pulse as an early symptom of *rheumatoid arthritis.* The pulse increases gradually until it reaches a range of 110 to 120, and it persists at that rate with little diurnal variation, even after the arthritic symptoms subside.

7. In *locomotor ataxia* permanent moderate acceleration of the pulse (90 to 100) is a frequent symptom.

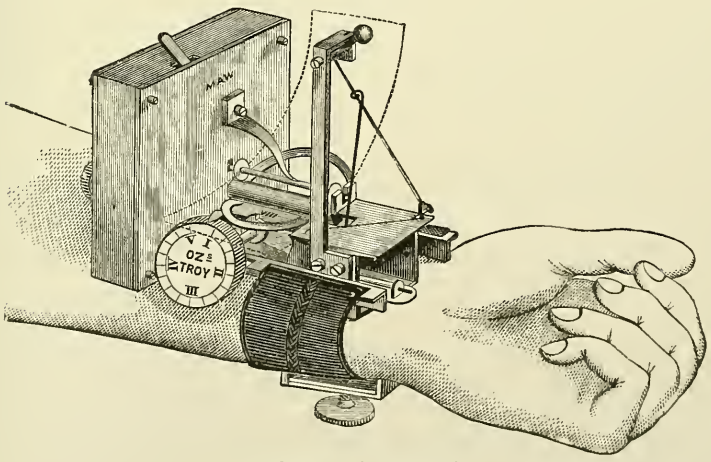
8. *Infections.* In the *puerperium* increased frequency with irregularity of the pulse is a surer indication of intra-uterine mischief than is the temperature. So, too, in all cases of inflammation so situated that the products are absorbed into the circulation and not discharged externally, the pulse shows by its increased frequency that a septic process is going on.

Diminished Frequency. A *slow* pulse (bradycardia), under 60, like a frequent pulse, is sometimes habitual, and sometimes a family characteristic. Pathologically, it is met with in conditions which increase the resistance in the arteries, such as Bright's disease, especially acute glomerulo-nephritis; but it is especially common in jaundice. The bile-acids have the effect of retarding the action of the heart.

A slow pulse is met with in certain forms of *heart disease*, as aortic stenosis, but it is not constant in any of them. It occurs in fatty degeneration, especially when due to obstruction, by atheroma or otherwise, of the coronary arteries. W. J. Pettus has reported a case of bradycardia associated with aneurism of the right sinus of Valsalva, involving the orifice of the right coronary artery. When it appears in the late stages of valvular affections or specific diseases with cerebral symptoms it is usually a sign of danger. It is seen in articular rheumatism (Atkinson). According to Riegel, it is most common in *convalescence from acute disease*, particularly pneumonia, typhoid fever, erysipelas, and rheumatic fever. It is also frequently encountered in diseases of the *digestive organs* and of the urinary organs, particularly *acute nephritis*. Moreover, it is generally slow in *myxœdema*, and both slow and irregular in *epilepsy*. It is slow, not uncommonly, also, in *melancholia* and in the early stages of *cerebral meningitis* and in tumors and cerebral hemorrhage.

The Sphygmograph. The sphygmograph, as its name implies, is an instrument for recording in writing the volume, force, frequency,

Fig. 154.



Dudgeon's sphygmograph.

tension, and general characteristics of the pulse. Many forms of the instruments have been devised since the first one of Marey. The later models have the advantage of simplicity and ease of application. One of the most convenient is Dudgeon's. It has its faults, particularly in exaggerating the vibrations when the pulse is large and the heart is acting violently; nevertheless, with care, trustworthy tracing can be

obtained in all ordinary cases. No matter what instrument is used, the value of the tracing depends very largely upon the personal skill and experience of the one who takes the tracing; hence the sphygmograph occupies a position very different from the thermometer and other instruments of precision. While it is true that a person can learn to detect nearly all the variations of the pulse by palpation alone, yet the tracing has the great advantage of permanency, and many persons are led to palpate the pulse more carefully by seeing in a sphygmographic tracing a dicrotism or irregularity which had escaped their attention.

The expansile pulsation of the artery is communicated by a system of levers to a needle, which graphically records the qualities of the pulse upon smoked paper.

Directions for Using Dudgeon's Sphygmograph. 1. Wind up, by the button, the clockwork contained in the box. The clockwork carries the smoked paper under the writing-needle.

2. See that the patient is in a comfortable position, and have him hold toward you either hand with wrist exposed, fingers gently flexed, and muscles relaxed.

3. Apply the instrument by slipping the band over the hand, the free end of the band being passed through the retaining clamp. The metal box is placed toward the elbow.

4. Now adjust the instrument by placing the bulging button which connects the levers directly over the radial artery at its most accessible point.

5. Keep the instrument accurately in place with the left hand, and draw the band through the clamp with the right until the writing-needle plays freely with each pulsation of the radial artery, then fasten the band by screwing up the clamp.

6. Introduce the smoked paper between the rollers and under the writing-needle.

7. Vary the pressure by means of the thumb-screw, which connects with an eccentric, until the best apparent amplitude of vibration is obtained.

8. Instruct the patient not to move the fingers or hand, and further steady them for him with your own right hand.

9. Start the clockwork by pushing the bar at the top of the clockwork box.

10. Allow the paper to run through, and then stop the clockwork.

The clockwork is so regulated that five inches of smoked paper pass through in ten seconds, so that six times the number of pulsations recorded on the paper represent the pulse-rate per minute. Each instrument, however, should be tested and its time determined. The clockwork should be wound up for every tracing.

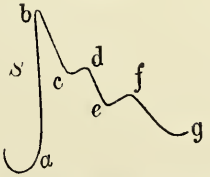
Considerable practice will be required to take a tracing rapidly and accurately, in spite of the simplicity of the mechanism.

Several tracings should be taken at different pressures and compared, or, what is better, as suggested by Sansom, stop the clockwork and alter the pressure two or three times, so as to have the effect of varying pressures on one tracing.

The technique of sphygmography needs a few words. Smoked paper is generally used for the tracings. A paper glazed upon one surface and rough upon the other has some advantages. This paper has to be cut in strips about seven-eighths of an inch wide and six inches or more long. The cutting should be done with care so that the edges are smooth and even, otherwise the paper sticks in the instrument and the tracing is spoiled. The glazed surface is blackened by holding it above the flame of a small piece of burning gum camphor. For convenience a strip of tin, bent upon itself at each end, so as to catch and hold about an inch of the ends of the paper, may be used to prevent the fingers from becoming blackened and to preserve the ends of the paper unblackened for memoranda. The blacking should not be too thick, otherwise the needle will not plough through it easily, and the white line of the tracing will not be distinct. After the tracing has been made, the name of the patient, the diagnosis of his disease, the date of the tracing, and the amount of pressure employed should at once be scratched with a fine-pointed pen upon the blackened surface beneath the tracing, or written in ink upon the unblackened end of the paper. The tracing is then ready for preservation. This is done by dipping it into a solution of shellac or in tincture of benzoin (gum benzoin ℥j, alcohol f℥vj) ; the alcohol evaporates and leaves a smooth, glazed surface. Dr. Dudgeon recommends as a varnish a solution of gum damar ℥j, rectified benzoline f℥vj. When the tracing is likely to be subjected to friction, a second or third coat should be applied subsequently.

Explanation of the Normal Pulse-tracing. With each contraction of the left ventricle a volume of blood is forced into the aorta, which distends it, the distended impulse being transmitted by a wave-like motion to remote arteries. This distending impulse lifts the button of the lever sharply upward, forming the so-called percussion up-stroke,

FIG. 155.



a, b, percussion up-stroke; *a, b, c*, percussion wave; *c, d, e*, tidal wave; *e, f, g*, dicrotic wave; *d, e, f*, aortic notch; *f, g*, diastolic period.

a b ; but the distending impulse is exaggerated by the system of levers, and having been thrown up too high, the lever falls by its own weight too low, so that it is again caught and lifted by the tidal blood, forming the tidal-wave, *c d e*. The gradual descent of the lever is again interrupted at *e f g*, forming a wave, called the dicrotic wave, due to the recoil of the blood from the closure of the aortic valves. (Fig. 155.)

Roy and Adami believe that the apex (*h, b, d*) of the percussion-wave is due to the sudden pulling down of the auriculo-ventricular

valves by the papillary muscles during the first rapid part of their contraction. Hence they call the wave the "papillary wave."

The second wave (*c, d, e*) corresponds in time, they say, with the outflow from the ventricle due to the continued contraction of the heart-wall and papillary muscles after the flaps have been pulled down. Hence, they prefer to call this wave the "outflow remainder," instead of "tidal" wave.

Interpretation of Pulse-tracings. Sphygmographic tracings must be interpreted in accordance with the known peculiarities of the patient, his history, and the associated physical signs.

1. *The Amplitude.* The height of the percussion-stroke varies considerably in health. It is increased in conditions which bring about low tension and rapid systolic contraction of the heart. Hence the febrile pulse is usually one of considerable amplitude. It is increased also very markedly in aortic regurgitation. Suddenness of systole rather than force determines the height of the up-stroke. (See Fig. 156).

Fig. 156.

Tracing from a case of aortic regurgitation.

2. *Obliquity of the Percussion-stroke.* Normally the percussion-stroke ascends vertically from the base-line. A tendency to incline forward indicates a weak and laboring heart or an aneurism interposed between the radial artery and the heart. In the latter case there is also a tendency to rounding of the summit of the percussion-wave, and the up-stroke is generally short. There is usually also irregularity in successive pulsations, some showing the gradual ascent and rounded summit much better than others. Sometimes, however, when aneurism exists, there is no evidence of it in the tracing, and differences upon the two sides are not always significant. (See Fig. 157.)

Fig. 157.

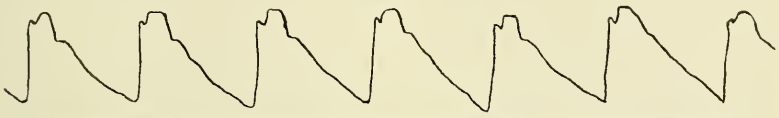
Tracing from a case of aneurism of the aorta.

Disease at the aortic orifice and the intervention of a considerable quantity of subcutaneous fat or of any growth superficial to the vessel may cause a marked obliquity of the percussion-stroke. Sansom asserts that, such causes excluded, as well as aneurism and organic disease of the aorta and its valves, a sloping line of ascent, observed under various gradations of pressure, indicates feebleness of the left ventricle. He considers it of higher diagnostic value than irregularity, which he says is often neurotic.

3. *Increased Breadth of the Apex of the Percussion-wave.* The breadth of the apex of the percussion-wave indicates the time during

which the artery is kept full by the systole of the left ventricle. When the left ventricle acts slowly and forcibly the arteries will be kept distended for a longer time, and this distention will be manifest in broadening of the apex of the tracing. (See Fig. 158.) The degree

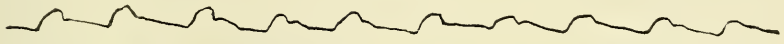
FIG. 158.



From a case of aortic stenosis, showing increased tension and the *pulsus bisferiens.*

of distention of the artery is called tension, hence a broadening of the apex is an evidence of high tension. As the word "high" does not indicate the duration of the tension, Sansom has very properly suggested that we should speak of persistent high tension as "prolonged" tension. This, then, is the significance of the broad top of the tracing. (See Fig. 159.)

FIG. 159.



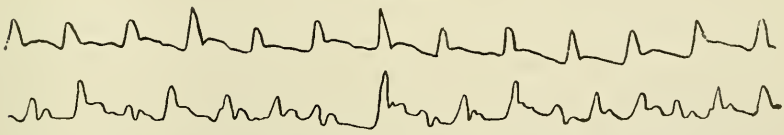
From a case of mitral stenosis, showing increased tension and some irregularity.

Prolonged arterial tension occurs when there is a strong heart acting slowly, a large volume of blood, or obstruction in the capillary circulation. (For specific causes, see under Tension.)

The amount of pressure required to develop the characteristics of a pulse, and still more, the amount required to obliterate it, are good indexes of the degree of tension present. Some pulses, however, appear to the touch to be of prolonged tension, but a sphygmogram does not show it. Such cases are often explained by the fact that the heart has begun to fail under the strain put upon it by prolonged obstruction in the capillaries. There may be regurgitation also from the mitral or aortic orifice.

4. *Acute Angle of the Percussion-wave.* When the heart's action is feeble or sudden, the volume of blood small, or the resistance in the

FIG. 160.



Low tension with irregularity, from cases of mitral regurgitation.

capillaries much diminished, the up-stroke of the tracing is vertical, and the down-stroke forms an acute angle with it. The dicrotic wave is pronounced, and often descends unduly low, sometimes to the base-line. These are the characteristics of low tension. (See Fig. 160.) When the dicrotic wave springs from a lower level than the base-line

of the tracing it is *hyperdicrotic*. When the dicrotic wave is wholly effaced in the succeeding up-stroke it is *monocrotic*.

While dicrotism is commonly associated with low-tension pulses, it is occasionally met with also in high-tension pulses. Sansom says, however, that he has scarcely ever observed the conjunction of broad summit and marked dicrotism without the patient's manifesting the sign of failing heart.

5. *Irregularity of the Base-line.* This occurs normally in some persons as the result of respiration, especially deep breathing. It occurs in respiratory diseases also, and in affections causing dyspnœa. Decided undulation of the base-line, the curves being irregular, occurs in tubercular meningitis.

6. *Differences in the Height of Successive Percussion-waves or in their Distance from Each Other.* These are written evidences of disturbance in the rhythm of the heart. The first expresses irregularity in volume of successive beats, and the second irregularity in time. When this latter amounts to the omission of a beat it is called intermission. All these changes are shown in Fig. 161.

FIG. 161.

From a case of advanced mitral stenosis, showing extreme irregularity and intermission.

THE VEINS. *Thrombosis.* This is usually detected by palpation, and occurs most frequently in the femoral vein. The vein is transformed into a firm, round cord, and is distinguished from the artery by the absence of pulsation. Thrombosis in these veins and in the iliac veins higher up occurs in acute infectious diseases and in the debility of the aged. Dropsy in the area of distribution of the veins is perceived.

Percussion. By means of percussion the shape and size of the heart and changes in the area of cardiac dulness are determined. (See the Lungs for discussion on percussion.) To determine the size of the heart, both superficial or light, and deep, or strong, percussion must be employed. By the former we determine the area of superficial or absolute cardiac dulness; by the latter, the area of deep cardiac dulness.

1. THE AREA OF SUPERFICIAL OR ABSOLUTE CARDIAC DULNESS. (See Plate XVI.) It is the area not covered by the lung at the time of inspiration. The lungs overlap the heart, and, in inspiration, allow a small area to be in contact with the chest-wall. The percussion-force employed must be light, so as not to elicit the resonance of the extreme thin edge of the lung. The area extends from the fourth to the sixth costal cartilages. The right border may be roughly defined by a line drawn along the left edge of the sternum from the upper border of the fourth rib downward; the left border by a line extending from the upper border of the fourth rib at the left edge of the sternum to a point

midway between the parasternal and the mammillary line in the fifth interspace. The lower border is continuous with liver dulness.

Method. The right border is determined by percussing from right to left toward the median line. Always begin to percuss far enough from the heart to get the clear pulmonary note. To insure uniformity, select a definite area from which to start in all cases. Apply the finger vertically at first. The right border may correspond with a line outside of or along the right edge of the sternum, with the median line or the left edge of the sternum, or even beyond the latter. After the edge of modified resonance is reached, percuss with the finger parallel to the ribs, to control the result previously secured, and as each interspace is percussed the upper limit of liver-dulness and the triangle (Ebstein's) between the liver and heart may be determined.

The left edge is determined by percussing in vertical lines from a point near the axilla toward the heart. Opposite the second and third interspaces the aorta on the right side, and the pulmonary artery on the left, will cause impairment of the normal pulmonary resonance. The student should acquire the habit of proceeding from definite fixed positions toward the heart, and to observe the changes during inspiration and expiration. The lower border and rounded apex of an enlarged heart cannot be defined if the stomach contains food or fluid. It is triangular in shape, with the apex pointing downward.

The *cardio-hepatic triangle* is the more or less resonant area in the right fifth interspace which separates the right heart and the liver. The apex of the triangle points to the sternal edge, the base to the axilla. The upper side corresponds to the right border of the heart; the lower is the upper limit of the liver.

Changes in Size. The superficial area of dulness or absolute dulness is *increased* in pericardial effusion in enlargement of the heart and when the heart is pushed against the chest-wall. It is replaced by resonance in emphysema, and hence *absent* entirely, as the lung overlaps or completely covers the heart. It is absent when the heart is drawn under the lungs by adhesions and when there is air in the pleural or pericardial sac.

Absolute Dulness Increased. The increase in the area of absolute dulness in all directions occurs in hypertrophy of the heart and in pericardial effusions. The increase in width at the base of the heart occurs in dilatation, pericardial effusion, and aneurism of the aorta. Change in the position of the heart, a general idea of which is obtained by inspection and palpation, always changes the shape and extent of the dulness. The heart should be accurately delimited when displacements have taken place.

Increase of Dulness Upward. In addition to general increase in cardiac dulness, one of the boundaries or a portion of the boundary may be increased or extended beyond the normal line. Thus the area of dulness may extend *upward*. It may be followed by extension of the right and left boundaries. The relative area of dulness is abolished. The change from pulmonary resonance to dulness is abrupt and decided. The area of dulness becomes pyramidal or pyriform in shape. It is due to effusion in the pericardium. Upward increase of dulness

may be due to disease of the vessels. Increase in the area of dulness over the bloodvessels is usually due to aneurism. It may be general, as in dilatation of the aorta, or local, as in aneurism. Extension of the dulness outward or upward from the normal line may be found at the right of the sternum (aneurism of the ascending aorta), or over the first bone of the sternum (aneurism of the transverse aorta), or to the left just above the cardiac area. In the last case the dulness is an extension upward of the normal area of cardiac dulness with rounding of the area affected ; the aneurism is situated at the beginning of the aorta.

Increase to the Left. Increase in dulness to the *left* occurs in enlargement of the heart from hypertrophy or dilatation. If the dulness extends outward to the left and retains the triangular shape, with the apex pointed, it is due to hypertrophy of the left ventricle. If, on the other hand, it becomes quadrilateral in shape, with the apex rounded, it is due to dilatation of the left ventricle. The results of palpation and inspection aid in detecting the presence of one or the other of the two conditions.

Increase to the Right. The area of dulness extends to the *right*. It is due to hypertrophy and dilatation of the right auricle and ventricle. If the auricle is dilated, the right edge is extended beyond the normal in the third and fourth, or as high as the second interspace. With this increase in dulness there are also seen an epigastric impulse, venous turgescence, and pulsation of the veins of the neck or of the liver.

Deep Cardiac Dulness. Many authorities consider the deep or *relative area* of cardiac dulness of importance in diagnosis. The percussion must be strong. The best method is that advised by Gibson and Russell. Their directions are as follows: "Begin in the upper left interspaces sufficiently far out from the sternum to secure pulmonary resonance. For instance, in the second interspace begin in the mid-clavicular line and percuss strongly. As soon as a slight alteration in that sound is noted, the point is indicated by a mark. The second or third and succeeding interspaces are percussed in like manner, bearing in mind that the percussion must begin further out in each interspace, in order to get pure resonance. As dulness is secured in each space a mark is made. This is continued to the apex if that is visible, or to the base of the chest. By joining the marks in each interspace with the line at the base of the heart, the left border of the cardiac dulness can be fixed." The authors correctly point out that in this way the true apex of the heart is found, enabling auscultation to be conducted more accurately.

The right edge of the vessels and of the heart is defined in the same way. The difference in the sound, in passing from the lung to the heart, is not so distinct along the right border as along the left. The authors include the dulness which is due to the vessels at the base of the heart, and hence begin percussion in the higher interspaces. This they deem is proper, because it is impossible to delimit the two. The dulness of the vessels is not so marked, however, and may be indicated by simple change in pitch in the percussion-note. The lower border

of cardiac dulness is ascertained with difficulty, because of its close apposition with the liver. At times there is a difference in the character of the dulness between the two organs. It can be well made out by stethoscopic percussion. This may not be so pronounced as we pass from the heart to the liver in the median and parasternal lines. Toward the apex the difference is more apparent.

Pleximetric Percussion. For more accurate cardiac percussion, Sansom recommends the use of a pleximeter designed by himself, by which delicate shades in dulness can be readily heard. The pleximeter is a thin, flat, oblong plate one inch by half an inch, which has on its upper surface a column rising from the middle, one and a half inches in height, which is surmounted by a second plate three-eighths to three-fourths of an inch, set parallel with the lower plate. The instrument is held between the forefinger and middle finger of the left hand, the sensitive tips of the fingers resting on the upper surface of the larger horizontal plate. The lower surface of this latter is held close to the wall of the chest, and percussion with one or two fingers of the right hand with an even and not too forcible stroke from the wrist is made upon the upper plate. The resulting vibrations are transmitted to the ear and are also appreciated by the digital sense of touch, so that both senses aid in the determination of the nature of the sound produced.

Method. The pleximeter is placed with its long diameter parallel with the sternum, about midway between the axilla and the right sternal border. Percussion is made upon the summit of the column by one or two fingers, and the pleximeter is moved, always in parallel lines, nearer and nearer to the sternum. A line is reached where the vibrations are modified. Incline the pleximeter so that the vibrations come from its left edge. This edge, or line, is practically the line of demarcation of the dulness, and should be indicated with an aniline pencil. It corresponds to the outline of the right border of the heart. The process must be repeated at higher and lower levels until the entire right border of cardiac or aortic dulness is ascertained. In passing, it may be stated that percussing from above downward with the long diameter of the pleximeter horizontal instead of vertical leads to the upper limit of the liver as indicated by modified vibrations. At about the fifth right intercostal space a short curved line is thus made out along the right edge of the sternum, which indicates the outline of the right auricle at the point where it joins the liver-dulness. Above this, as far as the second rib, the line indicates the outline of the right border of the auricle and the aorta. The outline of the auricle may be in the mid-sternum ; of the aorta, at the right edge. In percussing the left side of the chest the same method is adopted. Begin at the level of the second rib, two or three inches beyond the left edge of the sternum, and move to the right. Join the lines of modified vibrations, and in this manner the left border of cardiac and aortic dulness is secured. The outline of the apex of the heart is readily mapped out. Over the tympanitic stomach light percussion is necessary. To narrow the area of percussion about the apex, the percussion may be performed on the larger plate, while the smaller

is applied to the chest. The vibrations over the liver and over the right ventricle are difficult to distinguish, although sometimes so different that demarcation of the border of the ventricle presents no difficulty. Between the apex of the left ventricle and the left lobe of the liver the space is easily marked out.

A correct outline of the heart and of the vessels is thus obtained. The upper limit of dulness is formed by the right auricle, the aorta, and the pulmonary artery. Any bulging or undue expansion is due to aneurism or aneurismal dilatation of the aorta. The space between the apex and the left lobe of the liver defines the lower border. Sansom points out that by this method of percussion the following absolute data can be obtained: "A projection to the right of the area of the upper part over the second and third interspaces points to aneurism of the aorta or of the innominate artery. It may be traced to the left side of the sternum, on account of saccular dilatation of the aorta. If the dulness at the upper part extend greatly to the left, an increase in size of the pulmonary artery may be suspected. Along the mid-sternal region, extension beyond the right side joining the line indicating the upper border of the liver indicates distended inferior cava. This distention occurs in right-sided dilatation of the heart, and the dulness may also be due to dilatation of the adjoining auricle. The outline of dulness obtained over the apex of the heart, if pointed, indicates hypertrophy; a more rounded outline shows dilatation. In uncomplicated hypertrophy the line of the right ventricle forms a much less obtuse angle with the liver-dulness than in dilatation. Of great diagnostic value is the diminution of the area of dulness from atrophy of the heart as observed in wasting, as in cancer, and in tuberculosis; it may also be observed in typhoid fever. In the above-mentioned conditions it is a bad prognostic sign."

Adjacent Dulness. Care must be taken not to confound the dulness of pleural effusion or consolidated lung with the cardiac dulness.

Repercussion. Modification of the vibrations felt by the fingers on the pleximeter, as pointed out by Sansom, may indicate an abnormal change in physical condition impossible to detect in any other way. It is to be remembered that over the lungs the vibrations are excessive; over solid structures they are modified or lessened. Now, the change from vibrations to absence of vibrations may be *gradual* or *abrupt*. Sansom determines this by percussion, after the heart has been outlined in the above-mentioned manner. In percussing from the lung to the heart area, if the modified vibrations occur abruptly, it is very probable that there is pericarditis with effusion or thickened pericardium; or if, on percussing from above downward, there is pericardial effusion, no vibrations are to be elicited over the area delimited—that is, the absence of vibrations is noted over the whole area—whereas, in ordinary conditions, when the pericardium is unaffected, in percussing from above downward over the area which had been delimited on the right and left sides respectively, a line will be reached where the vibrations become modified. This line commences a little above the ensiform cartilage and inclines toward the

left border of the cardiac dulness at the level of the fourth rib and third interspace. Vibrations are more marked above than below the line. The line at which the lessened vibrations begin points out the commencement of the thick wall of the ventricles ; the portion above (more vibratory) indicates the position of the right auricle and vessels. If the pleximetric percussion is employed, areas of superficial and deep dulness need not be estimated.

The Apex Impulse. Whichever method of percussion is employed, it will be often observed that the spot marked by inspection and palpation as the apex impulse is far outside of the left border of cardiac dulness. In hypertrophy of the left ventricle it may be a considerable distance to the left. In dilatation the difference is not so marked. The percussion-lines are made when the heart is away from the chest, and hence are within the systolic apex-beat.

Method of Graphic Record. (See also page 536.) We are indebted to Sansom and Ewart for a method of recording the outlines of the areas of dulness and the position of the apex-beat and other pulsations, which is of great value for class-demonstration, and for permanent records to compare with other records taken from time to time. The points of pulsation and border-lines of dulness are marked by a dermatographic pencil. Various colors may be used in order to indicate the different data. The landmarks, etc., are outlined by a camel's-hair pencil dipped in olive oil. The episternal notch, the clavicles, the intercostal spaces, the ensiform cartilage and nipples, etc., the percussion-outlines, and other recorded marks are passed over with the oiled pencil. A sheet of tissue-paper, or of copying-paper, is then gently placed over the whole, so that the oil-marks are imprinted. After the paper is removed the oil-outline is colored with the dermatographic pencil, and a permanent record is preserved. By this plan of recording a maximum of precision is attained. Outlines can be measured and positions defined by mathematical data. The name of the patient, the date of observation, with a brief history of the case, should be attached to the chart. If the colored pencil-marks on the patient's chest are objectionable, the outline may be made with the colorless oil-pencil at the various steps of the examination. After they are transmitted to the paper they may be made more distinct with the colored pencils. Packard fits to the chest a square of coarsely woven muslin and outlines the ribs and sternum, etc., which are seen through the meshes. With colored pencils, dull areas, etc., the site of organs, the position of murmurs, are then designated.

Ewart has shown that after long intervals the size of the chest and abdomen is apt to alter from various circumstances—growth, muscular development, habit of sitting, etc. He therefore points out the advisability of using the *sternum*, which is immovable, for the sake of future comparison.

Sense of Resistance. Ebstein delimits the heart by the sense of resistance, change in size being noted by increase or diminution of the area, which in health gives a sense of resistance to the percussing finger.

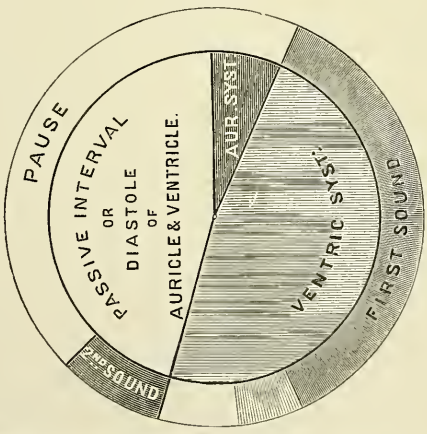
Auscultation. *Method.* Either method of auscultation may be employed. By the immediate method we may form a general notion

as to the condition of the heart-sounds. The mediate, however, is preferable, because it is essential to localize the sounds that are heard, and because, if the double stethoscope is used, we can percuss the cardiac area. The patient should be in a comfortable position. The muscles should not be strained. The general directions for performing auscultation must be followed. Before he begins the observer has, if possible, determined the presence of the impulse, or found the radial or carotid pulse. By this means the time of the heart is taken and the relation of the events of the cardiac cycle to each other is ascertained. With each normal impulse or carotid pulse a systole takes place; hence they are synchronous. The systole occurs just before the radial pulse.

By auscultation we determine (1) the normal sounds of the heart, including their rhythm, their character and the seat of maximum intensity; (2) modifications of the normal sounds as regards (*a*) loudness and (*b*) rhythm; (3) the presence of abnormal sounds or murmurs.

I. The Normal Sounds. The stethoscope is placed over the heart and the finger on the impulse or the radial pulse; a sound will be noted at the time of the impulse or the systole, followed almost immediately by another sound and then a period of silence. The sounds that attend the systole are known as the systolic, or *first sounds*. The sounds that follow are known as the diastolic, or *second sounds*. The sounds and

FIG. 162.



Diagrammatic representation of the movements and sounds of the heart. (After SHARPEY.) This diagram shows merely the general relations of the several events, and does not represent exact measurements.

In a heart beating seventy-two times a minute, Foster estimates each entire cardiac cycle as occupying about 0.8 sec., of which 0.3 sec. represents the duration of the systole of the ventricle, 0.4 sec. the diastole of both auricle and ventricle, or the "passive interval," and 0.1 sec the systole of the auricle.

Only one "pause" is marked here—sometimes called the "long pause"; some writers describe a "short pause" also—indicated in the diagram by the small space between the first and the second sound.

silence mark the completion of a cardiac cycle as far as the ear is concerned. (Fig. 162.) A definite relationship in time exists in the cardiac cycle. *Cause.* Four sounds are created during a cycle, one at

each valve. The sounds created with the systole (systolic sounds) are due to contraction of the right ventricle and closure of the tricuspid valve; and on the opposite side, of the left ventricle and the mitral valve. The rush of blood along the course of the vessels and the shock of the heart may contribute somewhat to the systolic sound. The sounds heard in the beginning of the diastole (diastolic sounds) are due to closure of the aortic and pulmonary valves. They are due to the tension produced on the valves as the respective arteries contract upon the columns of blood. The closures of the valves make up most, if not all, of the sounds. To review: two sounds occur with the systole, one from closure of the *mitral*, another from closure of the *tricuspid* valve; two with the diastole from closure of the *aortic* and *pulmonary* valves, respectively. In health the sounds of the systole blend because synchronous, giving the impression at a common point of one sound. Analysis of the sound in the respective valve areas will show that the systolic sound is made of two sounds. The sounds of the diastole may or may not blend. Often there is an appreciable difference between the two.

Recognition of the Respective Sounds. To distinguish the sounds we study their *rhythm* or *time*, their *character*, their position of *maximum intensity*, and their *direction* of *transmission*. We distinguish the first from the second sounds by their rhythm and character, and then differentiate the sounds respectively of the systole and of the diastole by their point of maximum intensity.

(*a*) The *Rhythm* or *Time*. The sounds that are heard at the time of the normal impulse or just before the radial pulse are the systolic or first sounds; the sounds that follow the impulse are the second sounds. The sounds that follow the long silence are the systolic or first sounds; those that precede the long silence are diastolic or second sounds.

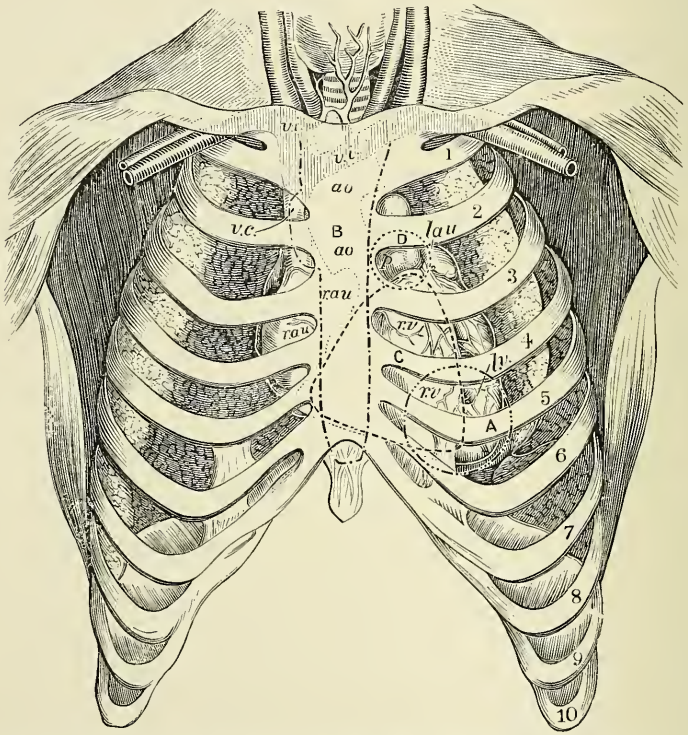
(*b*) Character of the Sounds. The systolic sounds are prolonged, somewhat dull in character, low in pitch, and resemble the sound produced by the pronunciation of the syllable "*ubb*." The diastolic sounds are short, sharp, and quick, and resemble the sound produced by the pronunciation of the syllable "*dupp*." The syllables *ubb*, *dupp* indicate the character of the sounds in health. Modifications in the intensity of the sound are due to changes in the tension of the valve-curtains, and are dependent upon the force of muscular contraction, which, if strong, renders the valves more tense. Experiment and the results of disease have aided in proving these points.

(*c*) Position of Maximum Intensity. In general the first sounds are loudest at the lower part of the præcordia, the second at the upper. But we especially distinguish the independent valve elements which make up the systolic and the diastolic sounds in the following manner. The sounds produced by the closure of the valves are created, as the topography of the heart shows, quite near to each other, but by conduction of the sound they are transmitted away from the respective valves in particular directions, and heard loudest in definite areas on the chest.

The Systolic or First Sounds. Two sounds are created. The valves which cause the sound are near to each other. Because of their

anatomical relations the sounds are conducted into different areas, by virtue of which they are differentiated. The *Mitral Valve Sound.* The sound produced by the closure of the mitral valve is created opposite the fourth interspace near the sternum. It is transmitted to the surface of the chest by the thickened left ventricle, and hence is heard

Fig. 163.



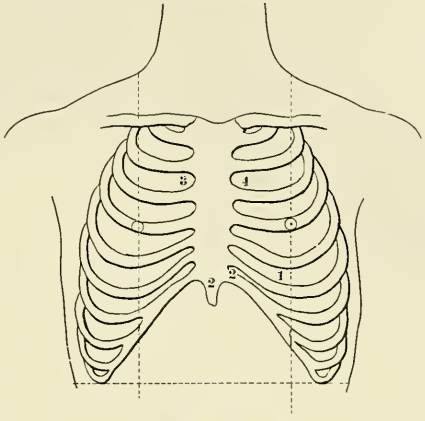
Areas of cardiac murmurs (Gairdner for the areas; and Luschka for the anatomy). The outlines of organs, which are partially invisible in the dissection, are indicated by very fine dotted lines; while the areas of propagation of valvular murmurs, as described in the text, have been roughly marked by additional much coarser and more visible dotted lines—the character of the dots being different in each of the four areas A capital letter marks each area—viz., A, the circle of mitral murmurs corresponding with the left apex; B, the irregular space indicating the ordinary limits of diffusion of aortic murmurs, corresponding mainly with the whole sternum, and extending into the neck along the course of the arteries; C, the broad and somewhat diffused area occupied by tricuspid murmurs, and corresponding generally with the right ventricle; D, the circumscribed circular area over which pulmonic murmurs are commonly heard loudest.

Reference letters: r. au. = right auricle: a. o. = arch of aorta; v. i. = the two innominate veins; v. c. = vena cava descendens; p. = pulmonary artery; l. au. = left auricle; l. v. = left ventricle; r. v. = right ventricle. (Finlayson.)

loudest where that is nearest the chest, namely, at the *apex*—the *mitral area.* The *Tricuspid Valve Sound.* The sound produced by the closure of the tricuspid valve is transmitted by the right ventricle, and is heard loudest over the lower portion of the sternum—the *tricuspid area.*

The Diastolic or Second Sounds. Two sounds are created. The valves at which they are produced are also in close proximity. To distinguish the two sounds it is necessary to auscult over areas into which they are transmitted. They may often be distinguished by

Fig. 164.



The valve areas.

1. Mitral area. 2. Tricuspid area. 3. Aortic area. 4. Pulmonary area.

their slight difference in time, the aortic preceding the pulmonic by a fraction of a second. *The Aortic Valve Sound.* The sound produced by the closure of the aortic valve is heard loudest at the second costal cartilage on the right, because the aorta which conducts the sound is nearest the surface of the chest at this point—the *aortic area.* This cartilage is known as the *aortic cartilage. The Pulmonary Valve Sound.* The sound produced by the closure of the pulmonary valve is conducted to the left and heard loudest in the second interspace near the left edge of the sternum—the *pulmonary area.*

(*d*) The Direction of Transmission. The first sounds are transmitted toward the axillæ. They may be heard all over the cardiac area, but the position of maximum intensity is in the lower portion and toward the left. The second sounds are loudest at the base of the heart. They may be propagated beyond the præcordia toward the neck, and be heard loudest in the vessels of the neck.

Precise Location and Differentiation of Each Sound. This may be determined by listening with the bell of the stethoscope over each area. Then move the bell of the stethoscope gradually from one area into the other. As the sound of the original area lessens the sound of the approached area is observed. The change from one to the other is often very marked. 1. *Mitral* first or systolic sound, heard loudest at the apex, inward to the parasternal line, upward to the third interspace. 2. *Tricuspid* first or systolic sound, heard loudest at the lower part of the sternum and toward the left to the parasternal line as high as the third rib. 3. *Aortic* second or diastolic sound, heard loudest at the aortic cartilage, propagated into the vessels of the neck, and also

heard at and outside of the apex-beat. It is louder than the pulmonary second sound in health. 4. *Pulmonary* second or diastolic sound, localized to the second interspace and the third rib.

II. Modifications of the Sounds. The sounds, singly or combined, may be increased or diminished in intensity or accentuation. They may be altered in rhythm.

Sounds Increased. *a. Causes outside of the pericardium.* 1. Anything which brings the heart closer to the ear of the observer. Thus, in patients with thin chest-walls, when the heart is pushed to the surface of the chest (mediastinal tumor) or the lung removed (pleural contraction). 2. Anything which conducts the sounds, as consolidated lung in the vicinity, or a pneumothorax, or pulmonary cavities. *b. Affections of the pericardium,* as pericardial adhesions. *c. Conditions of the heart.* 1. Hypertrophy. 2. Overaction, as in palpitation, fevers, anæmia, exophthalmic goitre.

Sounds Weakened. *a. Causes outside of the pericardium.* 1. General exhaustion. 2. Thick chest-walls, large mammary gland. 3. Emphysema of the lungs overlapping the heart. *b. Affections of the pericardium,* as fluid or air in the pericardial sac. *c. Conditions of the heart.* Atrophy; myocarditis; some cases of dilatation.

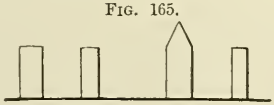
In short, *loudness* of all the sounds occurs from (*a*) conditions outside of the heart; heart nearer chest-wall, consolidation of lungs, cavities; (*b*) conditions of the heart itself; hypertrophy; overaction. *Weakness* of the sound occurs from: (*a*) Conditions outside of the heart: thick chest-walls, emphysema, general exhaustion; (*b*) affections of the pericardium: effusions; (*c*) affections of the heart: atrophy; dilatation; myocarditis.

Modifications of Individual Sounds. The above applies to all the sounds. Increase or diminution of the systolic or of the diastolic sounds, or of any one of the four sounds, may be present.

Increase in Loudness of the Systolic Sound. Increased loudness of the first sound is noted when the muscle is hypertrophied, and the tension on the valves thereby increased. In hypertrophy of the left ventricle the increase is most marked. The sound is duller and has a prolongation which is very characteristic. In hypertrophy of the right ventricle the sound is dull and prolonged over the sternum, but not to the same degree as when the left is hypertrophied.

Increase in Loudness of the Diastolic Sound. Either of the second or diastolic sounds may be increased in loudness or accentuated.

Fig. 165.



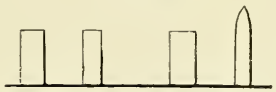
Normal first and second sounds. Accentuated first sound.

1. *The Aortic Diastolic Sound.* Anything which causes increased tension in the aortic circulation, and hence increased contractile force of the aorta, will increase the intensity or accentuation of the second sound. In hypertrophy of the heart the aortic sound is accentuated

because there is corresponding increased contraction of the aorta, following the forcible expulsion of the blood from the ventricle. Increase in arterial tension is also due to increased contraction of the aorta when there is peripheral resistance to the outflow of blood. It is associated with the following conditions which cause accentuation of the second sound: Atheroma of the aorta, or of the arteries in general; aneurism of the aorta; disease of the kidneys, and particularly in that form in which there are also general arterial changes—namely, chronic interstitial nephritis. It is true that the accentuation may be partly due to the hypertrophy of the heart which coexists.

Accentuation of the aortic second sound occurs independently of permanent change in the arteries. If for any reason there is spasm of the peripheral capillaries, as from a chill, from epilepsy, from nervousness due to hysteria, tension in the arteries is heightened, and hence the second sound accentuated. It is seen that accentuation of the second sound is, therefore, a marked index of the state of the vascular system in general; it is not an evidence of disease of the heart alone. In certain fevers and in states of the blood in which the vasomotor nerves are irritated, causing peripheral contraction, as in scarlatina, accentu-

FIG. 166.



Normal first and second sounds. Accentuated second sound.

ation of the second sound is observed, often before the development of local inflammatory diseases due to the same cause, as nephritis in scarlatina. The occurrence of this complication may be suspected when accentuation of the aortic second sound is heard.

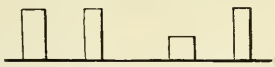
2. *The Pulmonary Diastolic Sound.* This is due to the same physical condition which causes accentuation of the aortic second sound. Anything which heightens the tension in the pulmonary artery will cause increased loudness. In health the pulmonary second is not so loud as the corresponding aortic sound. If, therefore, we find in the second or third left interspace the sound as loud as an aortic sound, or louder, it can be said that the pulmonary second sound is accentuated.

It is due: 1. To any condition which causes congestion within the lungs, the right ventricle being at the same time of normal or increased strength. It is heard in the early stages of pneumonia, and, if the course of the disease continues favorable, may remain accentuated to the end. If, on the other hand, the circulation is embarrassed, and the right heart is failing, it will become fainter, and may be scarcely recognizable. Such change in the sound accompanies increase of respiratory distress, and indicates that the right heart is becoming exhausted. It is, therefore, an ominous sign in acute pulmonary disease. If the case is unfavorable, the signs of right-sided dilatation will subsequently occur. 2. It occurs in emphysema of the lungs. Notwithstanding the covering of the heart by the lung, the sound can be heard,

and may be the only one of the four sounds which can be distinguished. 3. In valvular disease of the heart seated at the mitral orifice accentuation of the pulmonary second sound is heard, due to increased tension in the pulmonary artery. In mitral obstruction the blood is retained in the auricle and pulmonary veins, causing a resistance to the force of the right ventricle. Increased tension in the pulmonary artery is the result, with exaggerated strain upon the valves. In mitral regurgitation, with the systole the blood is thrown back into the auricle, and consequently meets with blood coming from the lungs. This in time increases the amount of blood and of blood-pressure in the pulmonary artery. A heightened tension results. Skoda pointed out the significance of this association. Sometimes in doubtful cases, either in the presence or absence of a murmur at the mitral orifice, the occurrence of this sign makes it more than probable that there is mitral valvulitis.

Diminished Accentuation of Feebleness of the Sounds. 1. *Feebleness of the Mitral Sound.* Feebleness of the mitral sound observed at the apex of the heart may be an indication of weakness of the muscle from dilatation, atrophy, or myocarditis. It must be remembered, however, that weakness of the ventricle is not attended by enfeeblement of sound alone, but that when the right or left ventricle is weakened the duration of the sound is lessened. The loudness remains the same, or may be increased. Note, then, that a short systolic sound,

Fig. 167.



Normal first and second sounds. Diminished first sound.

loud, sharp, flapping, sometimes reverberating, heard at the apex, indicates dilatation or feebleness. The tension of the ventricles and valves creating the sound is increased by internal pressure. The systolic sounds become like the diastolic, and may be distinguished by the ear with difficulty; but if the time is taken with the finger on the apex-beat or carotid artery, if the heart's action is slow the distinction can readily be made.

Diminished Accentuation of the Aortic Sound. This is an indication of cardiac weakness, and is apt to ensue in the course of fevers when exhaustion takes place. It is a sign of myocarditis and of degeneration of the muscular walls of the heart. Under these circumstances the systole of the ventricle is also weakened.

Feebleness of the aortic second sound, with hypertrophy and hence strong contraction of the ventricle, occurs when the aortic leaflets are swollen or enlarged and thickened. This condition of the valves is due to atheroma, and is in all probability associated with atheroma of adjacent vessels, as the coronary arteries. It is, therefore, a sign of serious importance.

Diminished Accentuation of the Pulmonary Sound. This is of importance in the course of valvular disease of the heart, providing previous

accentuation has been observed. If the marked loudness gives way to feebleness, there is strong probability that the right heart is undergoing dilatation with regurgitation at the tricuspid orifice. While accentuation of the pulmonary second sound in valvular disease is of good omen, enfeeblement of the sound is of bad prognostic omen, indicating weakness of the right ventricle.

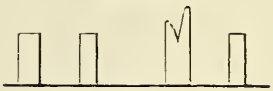
Alterations in the Rhythm. *Fœtal rhythm* of the heart: Embryocardia—a term first used by Huchard to designate a condition in which the pauses between the heart-sounds are of equal length. The first and second sounds are exactly alike, resembling the beat of the fœtal heart. The sign is of importance in prognosis. In acute disease and in fever it indicates enfeeblement of the heart and reduction of arterial tension. In the later stages of Graves' disease it is a forerunner of death. It is distinguished from the rapid beat of the heart in tachycardia by the fact that in the latter condition the normal rhythm is preserved.

Cantering Rhythm of the Heart. The ear recognizes three sounds. The usual sounds may or may not be attended by murmur, and the interpolated sound may be dull, or short and sudden. It may occur at various periods in the cardiac cycle, either just before the systolic sound, just after the diastolic sound, or during the diastolic pause. The rhythm recalls the sound of a horse cantering. It was termed by Bouillaud the *bruit de galop*. When the interpolated sound resembles the first or second it is similar to reduplication of the sounds. It has been observed in hypertrophy of the heart, especially of the left ventricle; dilatation of the heart; in adherent pericardium with dilated hypertrophy; in myocarditis, in the course of fevers; and in excessive anæmia. It is heard loudest over the right and left ventricles. Potain thinks it is due to tension communicated to the wall of the ventricle by the entrance of blood into its cavity, and is more marked when the wall is least extensible, as in hypertrophy on the one hand or exhaustion of the muscle; in either of the two the walls vibrate more readily. The triple rhythm is of bad prognostic omen in chronic Bright's disease.

Reduplication of the Sounds. Reduplication, or apparent doubling of the heart-sounds, occurs in various forms. In health the systolic sounds are created synchronously; a fraction of a second, not appreciated by the ear, separates the diastolic sounds. In so-called reduplication one systolic sound may follow the other, or the aortic and pulmonary diastolic sounds may be created at distinct intervals. As has been stated, in galloping rhythm the idea of reduplication is sometimes transmitted to the ear. Reduplication may take place in *health* under the influence of respiratory movements. The systolic sounds may be doubled at the end of expiration and the commencement of inspiration, while the diastolic sounds are doubled at the end of inspiration and the commencement of expiration. In mitral disease reduplication, or want of synchronous closure of the two valves, is of frequent occurrence. The heart-sounds are doubled and heard over the base of the heart. Reduplication of the systolic sounds occurs in chronic Bright's disease.

Reduplication, or Doubling of the Systolic Sounds, is heard over the apex or the right ventricle. Several explanations have been given for the cause of the reduplication. At first it was thought to be due to want of synchronism in the action of the ventricles—that one ventricle contracted before the other, due to the fact, of course, that the presence of blood stimulates one but not the other. By Hayden it was thought that reduplication of the first sound was due to the two major elements of the sound acting asynchronously, the muscular sound

FIG. 168.



a. Normal first and second sounds. *b* Reduplicated first sound.

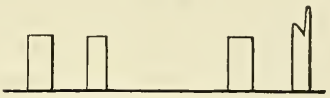
taking place before the sound produced by the tension of the valves. Dr. George Johnson took the view that the reduplication was due to the contraction of the auricle and ventricle; that the sound produced by the former was heard on account of hypertrophy of the auricle, and heard first because of the natural order of precedence. Thus far the reasons for each view have not been fully established.

Sansom believes that reduplication of the first sound is due to the shock communicated to the contents of the ventricle just before systole —that is, during the auricular-systolic period—in other words, it is due to the indirect effect of the auricular systole. The contraction of the auricle makes tense the auriculo-ventricular valve of the left side. If it occurs late in the diastole, or just before the systole, reduplication of the first sound is caused; if early in the diastole, reduplication of the second sound is created.

Reduplication of the Diastolic Sounds. While held by some authorities to occur in a large proportion of healthy individuals at the end of inspiration and the commencement of expiration, other observers, equally careful, think that it is extremely rare. It is of frequent occurrence in the patients of the Philadelphia Hospital. This is no doubt due to the fact that so many of the inmates are the subjects of all forms of lung disease, or disease of the vascular system, with muscular degeneration of the heart, that the equability of the pulmonic circulation is disturbed. There is no doubt that it can be modified or induced by inspiration. It is usually heard at the end of inspiration and commencement of expiration. Actual reduplication of the second sound occurs when the normal asynchronism of the closure of the aortic and pulmonary valves is exaggerated. It has been found that the valve of the pulmonary artery closes a fraction of a second after the aortic valve. The ear usually fails to appreciate the difference unless there are differences of blood-pressure; when doubled, and therefore appreciated, it is indicative of a difference in blood-pressure between the two sides of the circulation. Increased resistance in either will lead to increased tension, quickened recoil, and hence quickened closure of the valve. The conditions that are associated with the

doubling of the second sound are (1) and most frequently, mitral stenosis ; (2) obstruction of the circulation in the lung—tuberculosis, emphysema, and bronchopneumonia ; (3) dilatation of the right ventricle ; (4) myocarditis. The sound is heard at the second and third costal cartilages along the left edge of the sternum. It is frequently heard at the fourth and fifth cartilages on the left side. In cases of mitral stenosis it is heard near the apex.

FIG. 169.



a. Normal first and second sounds. *b.* Reduplicated and accentuated second sound.

Illustrating diagrammatically modifications of the heart-sounds. (GIBSON and RUSSELL.)

Simulated doubling, or false reduplication, is a sound produced at the mitral orifice. It is difficult to tell it from true doubling or reduplication. It is most distinct at the base of the heart along the left edge of the sternum. Occasionally it is more distinct near the apex than elsewhere. It occurs with the conditions found in true doubling and in mitral obstruction. *Cause.* Sansom, Cheadle, and others distinctly point out that this double second sound is of frequent occurrence, and that it is heard most frequently at the apex. Sansom thinks that the cause for simulated doubling of the second sound is the same as for doubling of the first. There is, first, the normal second sound ; second, a tension of the mitral curtain producing the second simulated sound. This tension is due to the shock of the blood coming from the auricle to the ventricle.

III. Abnormal Sounds or Murmurs. Abnormal sounds may be heard over the heart in addition to or replacing the normal sounds. These sounds are produced in the *pericardium*, in the *heart*, or in the *bloodvessels*. They are divided into friction-sounds and murmurs. They are recognized because they are a departure from the normal sounds or because they are superadded sounds.

ABNORMAL SOUNDS IN THE PERICARDIUM. They are known as *friction-sounds* and *splashing* or *bubbling sounds*. The former occur in the first stage of pericarditis, and are due to the rubbing together of the inflamed surfaces, either the congested, vascular pericardium, or the membrane bathed in exudation, or covered by lymph. The friction-sound is recognized by (1) its character, (2) time, (3) position, (4) transmission, (5) movability, (6) modification by position of patient, pressure, course of disease, etc. 1. The pericardial friction is usually of a to-and-fro character, and can be recognized as distinct from the heart-sounds. It resembles the rubbing or scraping together of two roughened surfaces. 2. It is not necessarily synchronous with each sound. It is a to-and-fro sound, systolic and diastolic in time. It may, however, be only systolic or only diastolic. 3. It is heard over the body of the heart, usually in the third and fourth interspaces, or even over the right ventricle. 4. It is not transmitted away from the

heart. Its location may shift from day to day in the præcordial area. 5. It may be modified by pressure or by respiratory movement, or be influenced by the position of the patient. It may disappear entirely in the upright posture. An impression of nearness to the ear is given by the sound observed in the first stage of pericarditis. It may be increased or lessened in loudness by a deep inspiration. It disappears during the period of effusion, to return after that is absorbed.

Diagnosis. It must be distinguished from the *pleural friction*, which disappears if the patient is asked to hold his breath. The pericardial friction is of cardiac rhythm, the pleural friction of respiratory rhythm. It must also be distinguished from the so-called exocardial friction-sounds. The pleura adjacent to the pericardium may be inflamed. With each beat of the heart the rough surfaces of the pleura are agitated and generate a friction. It is seated along the edges of the right auricle or left ventricle. It is systolic in rhythm, but has the special characteristic that it is modified by respiration. It may be arrested if the patient holds his breath. It is increased by inspiration, or diminished in expiration when the lungs recede from the heart in expiration. The pericardial friction must be distinguished from the crepitations and râles of cardiac rhythm produced by the impact of the heart against the lung. They disappear when the breath is held. The distinctions between pericardial frictions and cardiac murmurs will be considered later.

Splashing sounds are heard when there are air and fluid in the pericardium. They may be bubbling or gurgling or resemble the sound of a water-wheel. They continue when the breath is held.

Abnormal Sounds in the Heart and Vessels. *Murmurs.* If the student listens with the stethoscope over a large superficial vessel, and does not employ pressure, he will not detect any sound. If, however, pressure is employed, a sound or murmur is produced. The passage of the blood through the vessel produces no sound because the vessel or tube is of equal calibre. The pressure of the stethoscope alters the calibre and compels the fluid to pass through a narrow orifice into a wider space. In this manner a *fluid vein* is produced. The vibration of the molecules of the agitated fluid vein produces a sound or murmur. The loudness of the sound depends upon the swiftness of the flow. The sound in this instance is carried in the direction of the blood-current, hence the murmur is known as an *onward murmur.*

The reverse may take place. The fluid may flow backward from a wider into a narrower space without the production of sound; if, however, the fluid breaks on bevelled edges, as the leaflets of heart-valves projecting into the current, the fluid is again thrown into vibration and produces noise. If there is considerable constriction by the bevelled edge, the sound is carried farthest against the natural flow of the fluid —hence the term *backward murmur.* Some authors hold that murmurs are also due to lateral vibrations of the walls of the heart or of the vessels. Some murmurs may resemble tones, and are called *musical murmurs.* Such murmurs are due either to the vibrations of the solids set up by the vibrating fluid vein, or to the vibrations of the fluid vein alone.

Murmurs are divided into two classes, in accordance with their seat of development. Murmurs originating in the heart are known as cardiac murmurs. Murmurs originating in the bloodvessels are vascular murmurs. (See The Arteries.) Cardiac and vascular murmurs are divided into (1) *organic* murmurs, if due to physical changes of the heart or vessels ; (2) *inorganic, functional,* or *hæmic,* if due to changes in the quality of the blood. (See Functional Murmurs.) Cardiac murmurs are always generated at the orifices from disease or from incompetency of the valves, or from patulous non-valve opening. The orifices are *valvular* and *non-valvular.*

Murmurs at Valvular Orifices. The valvular orifices and their anatomical relations have been described. Murmurs are produced at these orifices when they are open or when normally they should be closed. If the murmur is produced when the orifice is open it is because there is narrowing of the orifice or dilatation of the cavity (relative narrowing). The murmur, then, is always produced *with* the natural current of blood, and hence is known as an *onward* or *obstructive* murmur. It always or nearly always implies organic disease at the valve-orifice, hæmic murmurs excluded. If the murmur is produced when the orifice should be closed, and hence when the valve leaks, it is because the valves are diseased and cannot shut the orifice, or because they are too small—incompetent—to shut it. Such murmurs are produced *against* the natural current of blood, and are known as *backward* or *regurgitant* murmurs.

Murmurs at Non-valvular Orifices. The orifices of the vena cavæ and of the pulmonary veins, and of the perforations of the septa in congenital heart disease, are non-valvular. They are at times the seat of murmurs—as in open foramen ovale or perforated ventricular septum.

Diagnosis of Murmurs. The student has learned that an abnormal sound or a murmur is present. It is necessary then to determine, *first,* at which orifice the murmur is produced (the seat of the murmur) and, *second,* the kind of murmur—obstructive or regurgitant. Murmurs are therefore studied as heart-sounds are studied, as to their position of maximum intensity, their *time,* and the direction of their *transmission.* The position of the murmur indicates which valve-orifice is affected, the time and the direction of transmission, and the kind of murmur.

The Position of Maximum Intensity of the Murmur. *The Orifice Affected.* We are enabled accurately to determine the orifice at which the murmur is generated by noting the position of maximum intensity of the murmur. This corresponds to the area at which the normal sound of the respective valve is heard loudest. It may be remembered that the cardiac orifices are closely situated, and that, therefore, the murmurs must be generated within a small area, so small that it would be impossible to ascertain at which valve-orifice the murmur is created, were it not for the fact that under the laws of conduction of sound the murmurs are conducted away from their point of origin to certain definite stations, where in health the respective valve-sound is also heard loudest.

1. *Murmurs at the Apex—the Mitral Area.* A murmur heard loudest, or with the greatest intensity, at the apex is known as a mitral murmur. It is created at the mitral orifice, but is conducted to the apex by the left ventricle, which is nearest the chest-wall at this point. (See 1, Fig. 164.)

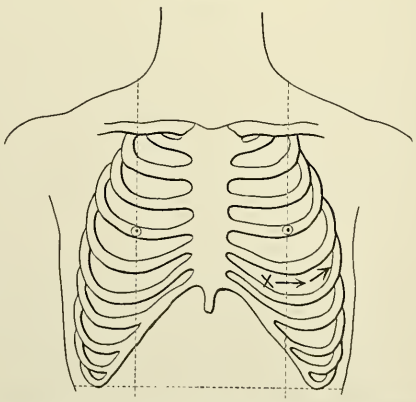
2. *Murmurs at the Xiphoid Cartilage—the Tricuspid Area.* The murmur is heard loudest at the xiphoid cartilage or the head of the fourth or fifth rib. It is created at the tricuspid orifice, and is heard most distinctly over the lower portion of the sternum, and along the left edge, because the right ventricle is in apposition with the chest-wall at this spot. (See 2, Fig. 164.)

3. *Murmurs at the Second Costal Cartilage or Second Interspace on the Right—the Aortic Area.* When a murmur is heard with greatest intensity at this point it is usually generated at the aortic orifice, and is conducted to this region by the aorta, which comes nearest to the surface of the chest at this point. (See 3, Fig. 164.)

4. *Murmurs in the Second Left Interspace—the Pulmonic Area.* A murmur heard loudest at the second interspace along the left edge of the sternum is generated at the pulmonary orifice ; it is heard loudest in this area because the pulmonary artery is nearest the chest at this point. (See 4, Fig. 164.)

The Rhythm or Time of the Murmur. *The Kind of Murmur.* Having determined the point of maximum intensity of the murmur, hence the valve at which it has its origin, we next wish to determine the kind of murmur. A murmur which is produced at orifices when they should be closed is known as the murmur of regurgitation, as the valve permits the blood to flow backward. A murmur that occurs

Fig. 170.



Maximum inteusity of murmur of mitra l regurgitation ; systolic ; transmitted to the left.

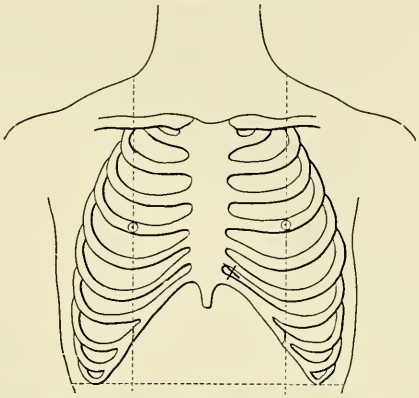
when the blood should in health be passing through an orifice is known as a murmur of obstruction, as the flow of blood is obstructed. We have to determine whether the murmur at an orifice is due to *regurgitation* or to *obstruction*. This is ascertained by the *time* of the murmur.

The time of the murmur is determined by the heart-sounds, by the impulse, and by the pulse.

Murmurs with the Systole.

1. *In the Mitral Area.* In health, during this time, the auriculo-ventricular valve is closed. The murmur indicates there is such disease as to permit of a backward flow of blood, or of regurgitation, into the auricle. It is the murmur of *mitral regurgitation.* It may be due to disease of the valves or to incompetency. (See Fig. 170.)

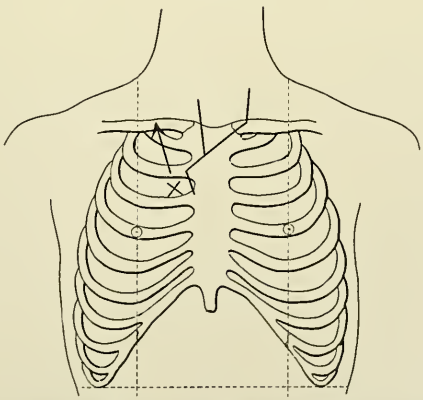
FIG. 171.



Maximum intensity of murmur of tricuspid regurgitation ; systolic.

2. *In the Tricuspid Area.* As on the left side, the murmur in this area is due to valvular disease or valvular incompetency, which permits of regurgitation, *tricuspid regurgitation.* (See Fig. 171.)

FIG. 172.



Position of maximum intensity and directions of transmission of murmur of aortic obstruction.

3. *In the Aortic Area.* During this time the blood is flowing from the ventricle into the aorta. If there is disease which causes obstruction at the orifice the murmur of *aortic obstruction* is produced. The

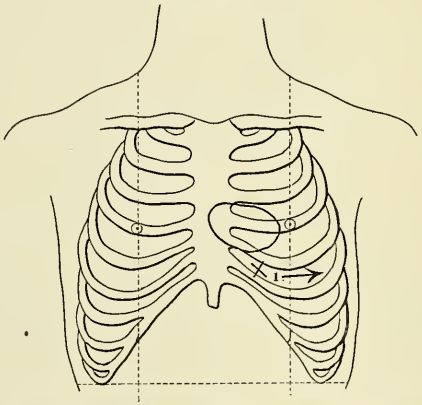
murmur may be due to anæmia ; to disease of the aorta, or to its malposition. (See Fig. 172.)

4. *In the Pulmonary Area.* The pulmonary orifice is affected in the same way as the aortic orifice under the same circumstances. The murmur is due to *pulmonary obstruction.* It is exceedingly rare. It is more frequently hæmic. (See Fig. 175.)

Murmurs with the Diastole.

1. *In the Mitral Area.* The blood is flowing from the left auricle to the left ventricle. Disease of the valves obstructs the flow. The murmur occurs in the beginning, in the middle, or at the end of the long silence. Mid-diastolic and late diastolic, or because it occurs before the systole, presystolic, are the terms applied to this murmur. It is the murmur of *mitral obstruction.* (See Fig. 173.)

FIG. 173.



Maximum intensity of murmur of mitral obstruction ; presystolic, localized or transmitted as area shows.

1. Normal impulse. ○. Area of reduplication of second sound.

2. *In the Tricuspid Area.* It occurs for the same reason and at the same time as the diastolic murmurs generated at the mitral orifice. It is rare, although more common than usually supposed, to find *tricuspid obstruction.*

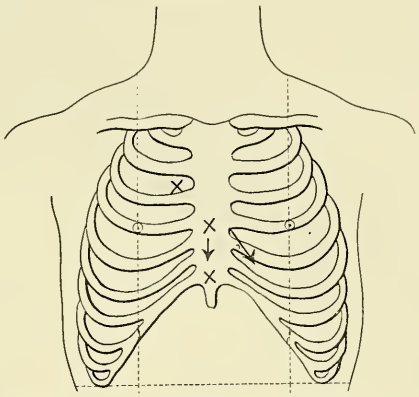
3. *In the Aortic Area.* The aortic valve closes in the diastole. A murmur indicates it is so diseased that it cannot prevent blood flowing backward or regurgitating into the ventricle. It is the murmur of *aortic regurgitation.* A murmur of the same time and in the same situation may be due to dilatation or aneurism of the aorta. (See Fig. 174.)

4. *In the Pulmonary Area.* A diastolic murmur in this area is due to *regurgitation* at the *pulmonary orifice.* (See Fig. 175.)

Murmurs are divided as to time into systolic and diastolic murmurs. The above shows that we may have practically only three systolic and two diastolic murmurs. The systolic murmurs are *aortic obstruction* and *mitral* and *tricuspid regurgitation.* The diastolic murmurs are *aortic regurgitation* and *mitral obstruction.*

THE DIRECTION OF TRANSMISSION. It depends upon the situation of the murmur and the time at which it is produced. Some murmurs are not transmitted. The transmission is usually in the direction of the currents which produce them.

FIG. 174.



Positions of maximum intensity and directions of transmission of murmur of aortic regurgitation.

Murmurs in the Mitral Area. To the *axilla.* A murmur which is produced at the apex with the systole, caused by *regurgitation* at the mitral orifice, is transmitted into the axilla, and may be heard at the angle of the scapula. The murmur which is produced in the same area before the systole—*obstruction*—is usually not transmitted. It is heard at the apex, or a little inside of the apex; or may rarely have its point of maximum intensity in the third interspace. Sometimes it is transmitted to the axilla and to the angle of the scapula. (See Figs. 170 and 173.)

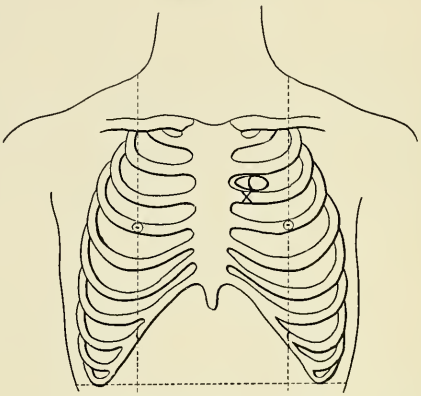
Murmurs in the Tricuspid Area. The murmur of tricuspid regurgitation is not transmitted. It is heard over a relatively large area, depending upon the intensity of the sounds.

Murmurs in the Aortic Area. Upward and Along the Vessels. The murmur, *systolic* in time, heard at the second costal cartilage on the right, due to aortic obstruction, is transmitted in the direction of the blood-current. The sound is conducted by the vessels and by the fluid; it is, therefore, heard along the course of the aorta and in the carotid arteries. *Downward to the Apex.* The murmur of aortic regurgitation, heard in the same area, is transmitted downward along the course of the sternum. It may be transmitted to the apex, or may be heard along the sternum only. The left ventricle conducts this murmur. (See Figs. 172 and 174.)

CHARACTER OF THE MURMURS. Murmurs are further distinguished by their character and the degree of *loudness.* By the character of the murmurs we are aided (1) in distinguishing them from heart-sounds; (2) in estimating the nature of the lesion that produces the murmur; (3) in judging, in the case of murmur of mitral obstruction, of the presence or absence of that disease.

Distinction from Normal Sounds. Normal sounds are sounds of tension; murmurs are sounds of rhythmical vibration. The normal sounds of the heart have been described by the syllable "*ubb*," "*dupp*," "*od*," and abnormal sounds of endocardial origin by "*uf*," "*uv*," "*us*," "*ush*," or by full vowel sounds as "*oo*," "*u*," "*ah*," and "*aw*," by musical tones, or by interrupted tones, or by general sounds, as "*urr*" or "*orr*."

Fig. 175.



Maximum intensity of pulmonary systolic murmur.
○. Area of murmur of anæmia.

The Nature of the Lesion. The murmurs may be rough or rasping, musical or whistling in character. They may be high or low in pitch. Murmurs that are rough and high in pitch are usually due to disease of the valves, causing thickening or stiffening of the leaflets, or to the projection of an atheromatous plate into the lumen of the orifice. Such conditions occur in chronic endarteritis and chronic endocarditis or valvulitis. On the other hand, murmurs that are soft and low in pitch are usually due to a physical condition which causes swelling of the valve or occlusion by soft exudations; they are heard in endocarditis of rheumatic origin, or the malignant form of endocarditis. The only murmur which has special characteristics is the murmur of *mitral obstruction*. It is a prolonged murmur of a churning or grinding character, sometimes rippling, and as if fluid were being forced through a narrow channel. It is usually presystolic, but may occur in the middle of the diastole.

Loudness. The *loudness* of the murmur is not of special significance, although, in general, it may be said that it indicates good compensation, and that the heart muscle is sufficiently strong to meet the demands of the circulation. Murmurs are louder in the recumbent than in the erect posture in some instances, especially mitral and tricuspid murmurs. Murmurs are often more distinct after exertion. Loud murmurs may become weak, and this change in character of the sound is of serious omen. They may disappear in the course of fevers and in the dying state.

Disappearance of Murmur. The student will often find that after a patient has been under treatment for a short time the murmurs disappear. This is probably due to the fact that there is complete compensation. In the terminal stages of cardiac disease they disappear because of weakness of the heart muscle. Rarely they disappear because the roughened valve causing them has been repaired. (See "Disappearance of Murmurs," by the author. *British Medical Journal*, 1897.) In other cases it may be necessary to bring out a faint murmur or increase its intensity by having the patient move about; this renders it more distinct by inducing more rapid action of the heart.

The Significance of Murmurs. Murmurs heard at the various orifices indicate either (1) disease of the valves; (2) incompetency of the valves; (3) disease of the blood; or (4) disease of the vessels in intimate relation with the heart. The systolic murmur at the second costal cartilage on the right may be heard when there is disease at the aortic orifice, causing obstruction; in atheroma of the aorta; in cases of aneurism just above the valves; in anæmia, and chlorosis, and in some vasomotor neuroses, as Graves' disease. Before concluding that the murmur is due to disease of the valves we must be able to exclude the other conditions. *Atheroma of the aorta* is most difficult to distinguish from obstruction, because the character of the murmur is the same and the associated conditions are similar. In both there may be a previous history of gout, rheumatism, syphilis, or alcoholism. The latter are associated with atheroma in other arteries of the body, and with degenerative changes that accompany atheroma. In young subjects, in whom there has been a direct history of rheumatism, or when the process has followed septicæmia, the probabilities are, in nearly all the cases, that the murmur is due to aortic obstruction. To distinguish the murmur of anæmia, chlorosis, or Graves' diseases is often difficult. The associate symptoms in each case are different, however, and with the changes in the blood indicate the nature of the murmur.

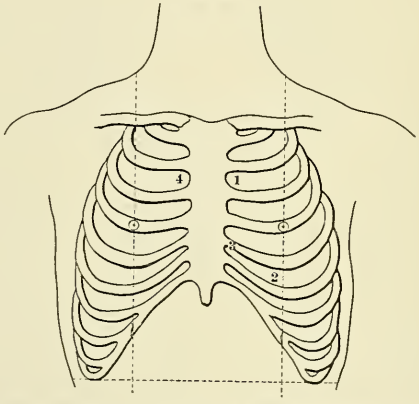
In other valve areas the chief task is to decide whether the murmur is *organic*, due to valvulitis, or whether it is *functional*, due to incompetency or to anæmia.

Murmurs due to Incompetency. The valves are sometimes unable to close properly. The cavity of the ventricles may increase in size, so that the valves do not coaptate to close the widened orifice. The tricuspid and mitral valve leaflets often become thus incompetent. Mitral and tricuspid regurgitation ensue. The murmurs are soft and low in pitch and not widely transmitted; the heart is dilated.

Murmurs of Anæmia. The murmurs of anæmia have some characteristics which aid in distinguishing them from true organic murmurs. The most important of these are: (1) The situation of the murmur; (2) its character; (3) the direction in which it is transmitted; (4) the time; (5) the associate signs; (6) the secondary heart-muscle changes. 1. The murmurs of anæmia may be heard at any orifice, but are usually heard at the second costal cartilage, or the third interspace, on the left side. They are generated at the pulmonary orifice, or in the cone of the right ventricle. The murmur at the pulmonary orifice may be heard as high as the second interspace, but otherwise is not transmitted.

Murmurs of anæmia are also heard at the apex, at the aortic cartilage, and over the tricuspid area. They are comparatively infrequent in these situations, but partake of the same nature as the murmur heard at the pulmonary orifice. 2. They are soft in character and low in pitch. They are louder in the recumbent than in the upright position. Their loudness is increased by violent cardiac action. They are loudest just at the end of expiration or beginning of inspiration. 3. They are not transmitted away from the heart. 4. They are systolic in time. 5. They are associated with murmurs in other parts of the vascular system, as the murmur in the jugular veins. Its characteristics and mode of recognition will be described elsewhere. 6. Mural changes, as general dilatation, fatty degeneration, or hypertrophy may be present; but single chambers do not undergo change. The murmur of anæmia may usually be considered to be temporary.

FIG. 176.



Maximum intensity of murmurs of anæmia, systolic. (SANSOM.)

1 Pulmonary artery, 59 per cent. 2. Apex, 7 per cent. 3. Right v. and conus, 11 per cent. 4. Aortic area, 11 per cent. 1 and 2. Pulmonary and apex coexisting, 9 per cent.

FUNCTIONAL MURMURS NOT ANÆMIC. Drummond divides functional murmurs into three classes: cardio-hæmic or anæmic; cardio-muscular or neuro-typtic, and cardio-respiratory. The first has been considered above. The *cardio-muscular murmur* attends excited action of the heart. It is heard loudest at the fourth left interspace close to the sternum; loudest in the upright posture; loudest at the end of expiration. It disappears at the end of inspiration, or when the patient lies on the side. Of course, it is increased by exertion and excitement. It is rough or whizzing in character. The *cardio-respiratory murmur* is fairly common. It is most marked in inspiration, but may be heard in both acts. It is systolic in time, and is heard loudest at the apex, but I have often heard it along the left border of the heart, as high as the second rib and in the axilla, and at the angle of the scapula. It is short and whiffing, and the sound gives one the impression that the heart is striking the lung.

Influence of Pressure. Pressure exerted, Sewall says, while using the flexible stethoscope over the second costal interspace annuls in part,

or wholly, the second sound of the heart; but if the ascending aorta be dilated or the site of an aneurism, the second sound persists strongly notwithstanding firm pressure.

Further, firm pressure removes –

(*A*) 1. Hæmic murmurs over the base of the heart (save Jenner's pulmonary murmurs).
2. An aortic obstructive murmur of the apex.
3. When mitral and aortic regurgitant murmurs coexist, the aortic murmur is diminished in the greater degree.
4. Aortic regurgitant murmurs over the second right intercostal space.

While it does not markedly affect—

(*B*) 1. Mitral regurgitant murmurs heard over the apex; or
2. Mitral obstructive murmurs over the same spot.
3. Tricuspid regurgitant murmurs over the area of greatest intensity.
4. Aortic regurgitant murmurs over the apex (see (*A*), No. 3).

Secondary Effect of Valve-lesions on the Heart and Pulse. The secondary effect of valve-lesions on the heart and pulse aid in the diagnosis. While we are enabled by the time of the murmur, the position, and the direction of transmission to affirm the nature of the disease at the respective valve-orifices, other physical signs further aid us in determining more precisely the lesion and its seat. They are derived from the heart and the pulse. They depend upon the secondary effect of the lesion upon the heart and upon the circulation. In *aortic obstruction*, on account of obstruction to the flow of blood, the left ventricle hypertrophies; moreover, the blood stream is lessened in volume, and hence the pulse is small and of high tension. The physical signs of hypertrophy and small pulse are corroborative evidence of this lesion at the left orifice. In *aortic regurgitation* the blood flows back into the ventricle. On this account, therefore, some dilatation takes place, a dilatation which, if compensation is perfect, is overcome by hypertrophy. The signs, however, of enlarged left heart are present, as shown by inspection, palpation, and percussion. But the pulse of aortic regurgitation is of the greatest diagnostic significance. With the finger on the radial, the impression is at once received of recedence of the pulse-wave as soon as it strikes the finger. This is more marked if the hand is elevated. It is the water-hammer, or Corrigan's, pulse. In *mitral regurgitation* the left auricle does not change, but the stress is thrown upon the right side of the heart, and we have the signs of right-sided hypertrophy and dilatation; but more marked than this is the evidence of high tension of the pulmonary artery, shown by accentuation of the second sound. (See p. 625.) In mitral regurgitation, the blood flows back into the auricle, and when the right heart weakens *engorges* the venous system. The arterial system is in consequence devoid of blood, and hence the arteries are empty. The pulse is small and feeble. The depleted coronary arteries do not nourish the ventricles, hence dilatation or failure in nutrition soon ensues, and the

heart is further weakened. In addition to being small and feeble, the pulse, on account of inefficient and hurried contractions of the ventricle, is irregular and intermittent.

In *mitral obstruction*, in addition to the characteristic murmur, the thrill is of great significance. Moreover, the left auricle hypertrophies, and shortly afterward the right heart. It is accompanied by an accentuated pulmonary second sound, and frequently by doubling of that sound. The pulse is small and feeble.

Multiple Cardiac Murmurs. More than one murmur may be heard over the heart. The number depends upon the number of valves that are the seat of disease and the lesions at the orifices. We may have valvulitis of the aortic, mitral, and tricuspid valves conjoined. More commonly one valve is diseased, giving rise to a murmur, while another valve is incompetent, on account of dilatation, and a murmur thus generated at its orifice It is common to see aortic obstruction from valvulitis and mitral regurgitation from incompetency; mitral obstruction or regurgitation from valvulitis, and tricuspid regurgitation from incompetency. I have seen double aortic disease (combined obstruction and regurgitation), double mitral disease, and tricuspid regurgitation. The diagnosis of the various murmurs will be discussed in the chapter on Valvulitis.

The Arteries. The stethoscope should always be used in examining the arteries. The double stethoscope is preferable, as strong pressure must be avoided upon the vessels. When the single stethoscope is used some diagnostic value attaches to the character of the shock that is transmitted to the head. The arteries open to auscultation are the carotids when the neck is slightly extended; the subclavian; the innominate above the sterno-clavicular articulation; the brachial artery in the bend of the elbow, with the arm slightly extended; and the crural artery just below Poupart's ligament. The normal systolic and diastolic heart-sounds are often heard in the carotid and subclavian arteries. The systolic sounds may be heard over the abdominal aorta, due to tension of the vessels. The diastolic sound is rarely heard in this situation. In the other vessels no sounds are heard.

Induced or pressure-murmur. By pressure with the stethoscope over one of the vessels its calibre is modified and a murmur created. This murmur corresponds in time with the pulse, hence it is systolic, and increases or diminishes in intensity, depending upon the amount of pressure placed upon it. Just here may be mentioned the systolic humming which is heard in children between the third month and the sixth year over the fontanelles and sometimes over the rest of the head. (See The Head.)

Abnormal Sounds. Abnormal sounds or murmurs are due to alterations of the blood, disease outside of the vessels causing pressure, and disease of the vessels. Murmurs from disease of the vessels, as the aorta, are discussed under the head of arterio-sclerosis or aneurism.

Conduction Murmurs. Murmurs may be propagated into the arteries. A systolic murmur created at the aortic orifice may be heard in the vessels of the neck and along the aorta. On the other hand, in aortic regurgitation, the diastolic sound normal in the carotid and

subclavian disappears, and the diastolic murmur is not heard. *Double Sounds of the Vessels.* Double sounds are sometimes heard in the crural artery under the following circumstances: (1) In aortic insufficiency; (2) in mitral stenosis; (3) in lead-poisoning; (4) in pregnancy. Duroziez's double murmur, heard when greater pressure is used by the stethoscope, occurs in aortic regurgitation when there is good compensation. Many authorities refer to this as a valuable diagnostic sign in this affection. The double sound in all instances occurs with large and quick pulse. It is probably caused by sudden collapse of the artery, and the reflux blood-current which is possibly an aortic regurgitation.

Murmurs due to Alterations of the Blood. They are generated in anæmia and chlorosis. They are called functional murmurs, to distinguish them from murmurs due to disease of the vessels. They are systolic in time. They are soft and low in pitch, often of a musical character. The degree of loudness may vary with the position of the patient. They are increased by excitement. The intensity of the murmur increases in the course of fevers.

Murmurs in Relaxed Vessels. Murmurs in the vessels, apparently of functional origin, are sometimes heard. The vessels are dilated from actual disease. The increased calibre favors the development of a murmur by the creation of a fluid vein. Dilatation of the innominate artery sometimes takes place, giving rise to a murmur, which in loudness and character simulates the murmur of aneurism. A functional murmur is sometimes heard in the vessels, independently of disease, in cases of aortic regurgitation. The murmur is systolic in time.

Pressure-murmurs. Pressure of the stethoscope, or that caused by *diseases outside of the bloodvessels.* When heard over the subclavian artery, the pressure-murmur may be due to adhesions or consolidation at the apex of the lung. It is more frequently heard at the left, and may only be present during full expansion of the lung. It is due to temporary pulling or bending of the artery during deep breathing. When it occurs on both sides it is not of much significance. Murmurs in the axillary artery, or in any arteries surrounded by enlarged lymphatic glands, are created by their pressure. Murmurs in the thyroid gland have been referred to. (See Goitre.)

Murmurs due to Disease of the Arteries. In the aorta the murmurs are due to aneurism or atheroma, or both. They may be systolic or diastolic. In the smaller vessels both conditions may be present, although atheroma is the usual one. The murmur is systolic in time, rough in character, strong or weak. It is associated with other signs of atheroma.

The Veins. In health no sounds are heard. Two conditions contribute to the creation of a *murmur* in the veins: (1) Change in the character of the blood; (2) dilatation with the occurrence of positive venous pulse.

The Venous Hum. In anæmia and chlorosis, and sometimes in healthy patients, a hum or murmur, or buzzing sound is heard over the jugular veins. It is louder on the right side than on the left. It is

soft and low in pitch, and may be musical; it has been described as humming or whizzing. It is continuous. For its detection a double stethoscope should be used, as pressure increases it, and the patient should not turn the head to one side, as it is increased when this position is taken. The murmur is modified by the respiration and by the cardiac action. It is louder in deep inspiration when the blood is going more rapidly to the thorax. It is also louder in the upright position. It is frequently louder during the diastole. The increased loudness at these periods occurs because, from the sucking action during inspiration and during the diastole, the blood is more rapidly drawn toward the heart. The murmur is caused by the flow of blood from the narrow jugular into its wider bulb, producing a fluid vein. Later authorities believe it to be due to lateral vibration of the walls of the veins. Similar murmurs are heard in other veins, as in those of the extremities when the anæmia is profound. They are stronger during the diastole of the heart. The venous hum is sometimes heard at the lower border of the liver, to the right of the median line, in cirrhosis of the liver. It is created in the enlarged collateral veins. It may be modified by pressure of the stethoscope. It may be heard in this situation in emaciated and cachectic subjects not the subject of cirrhosis. The venous hum may be heard in the innominate veins (first and second interspaces and right costo-clavicular articulation), in the subclavian and axillary veins.

Pericarditis.

Inflammation of the Pericardium. The inflammation may be acute or chronic. It is also divided according to the nature of the inflammation into simple fibrinous inflammation and inflammation with effusion. The effusion may be serous, bloody, or purulent, depending upon the nature of the inflammation. Pericarditis, either acute or chronic, is also divided into primary or secondary pericarditis. The primary form is of extremely rare occurrence. Indeed, it may well be doubted whether, in common with the inflammations of serous membranes in general, pericarditis is ever primary, or so-called idiopathic, in origin.

Causes. 1. *Extension from Neighboring Structures.* Extension of the inflammation from infected tissues in the vicinity is a common cause of pericarditis. It may follow a pleurisy and partake of the nature of the primary pleural inflammation. It often attends empyema, either from extension of the infection to the pericardium or from rupture into the pericardial sac. It may follow all forms of inflammation of the mediastinum. Disease of the ribs adjacent to the pericardium may set up pericarditis, acute and chronic. It attends on the course of aortic aneurism, at times, but more frequently infectious endocarditis and myocarditis. Inflammations below the diaphragm frequently give rise to pericarditis. Peritonitis, when general or local; sub-diaphragmatic abscess; suppurative gastritis, with perforation of the stomach; abscess of the liver; suppurating

hydatid, and other forms of suppuration below the diaphragm, belong to the latter.

2. *General Infections.* The general diseases causing inflammation of the pericardium are those which affect serous membranes. They are: Infectious diseases, particularly scarlet fever, measles, erysipelas, and typhoid fever. All forms of septicæmia may be attended by inflammation of the pericardium. Tuberculosis is a frequent cause of pericarditis. Inflammation of this membrane frequently arises in the course of rheumatism. It may occur in the course of the disease, or attend some of the affections which are themselves manifestations of rheumatism, such as acute tonsillitis. In the course of certain dyscrasiæ the pericardium is frequently the seat of inflammation because more vulnerable. This is particularly the case in scurvy. It occurs also in Bright's disease, and may be the first manifestation to the patient of this disease, particularly in the chronic form of nephritis. It occurs in the course of gout.

The various forms of pericarditis may occur at any age, although that which attends scarlatina and rheumatism occurs in early life, while late in life it is an attendant upon chronic Bright's disease and gout.

Acute Fibrinous or Plastic Pericarditis.

This is probably the most common form that is seen. It is the variety that attends Bright's disease, rheumatism, and tuberculosis. It may be wanting entirely in symptoms. An examination of the heart in the routine of duty may reveal its presence by physical signs. In the course of one of the primary causal diseases, if the temperature rises a little higher than it should, or convalescence is delayed, pericarditis should be suspected. Again, if the pulse is more rapid and quicker than customary at the period of disease the examination is made, or out of proportion to the temperature, the disease should be suspected. There may be altered rhythm or tumultuous action. In other instances the patient may complain of pain in the region of the heart. It is usually localized in the fourth or fifth interspace. It is not very severe and not influenced by pressure. Sometimes the pain is complained of at the xiphoid cartilage. In rare instances it may resemble angina. The pain and the occurrence of fever further call attention to the heart.

Physical Signs. Inspection. Nothing unusual is observed, although the heart may be seen to beat more violently against the chest-wall. The impulse is diffused.

Palpation. A friction-fremitus may be detected, due to the rubbing together of the roughened pericardial surfaces. It is not always present. It may be felt when the whole hand is laid over the præcordia, or by palpation with the tips of the fingers. It is most marked over the right ventricle, particularly in the fourth interspace, and is increased when the patient leans forward.

Auscultation. A friction-sound is usually present. It may be present while the fremitus is absent; but, on the other hand, if the fremitus

is present, we can always hear the friction. It is heard over the region where the fremitus is felt.

Point of Maximum Intensity. It may be heard along the course of the sternum It is usually heard in the third or fourth interspace, but may be heard as high as the second, adjacent to the sternum in either interspace. Sometimes it is heard at the second costal cartilage on the right, rarely at the apex. The point of maximum intensity may vary with the position of the patient.

Time. It is both systolic and diastolic. In some cases it may be only systolic in time, or it may be of a galloping nature, representing three sounds during the cardiac cycle. Again, the to-and-fro sound is not synchronous with the systolic and diastolic sound, although it occurs but once in the cardiac cycle. It may begin after systole, and be completed before the end of the diastole. The impression that it is a superadded sound is most positive.

Direction of Transmission. It is localized, and not transmitted.

Character. It is a to-and-fro rubbing, scratching, or grating sound; it gives the impression of being near the ear. It may be modified by the pressure of the stethoscope and by the position of the patient. It may be heard in the erect and disappear in the recumbent posture.

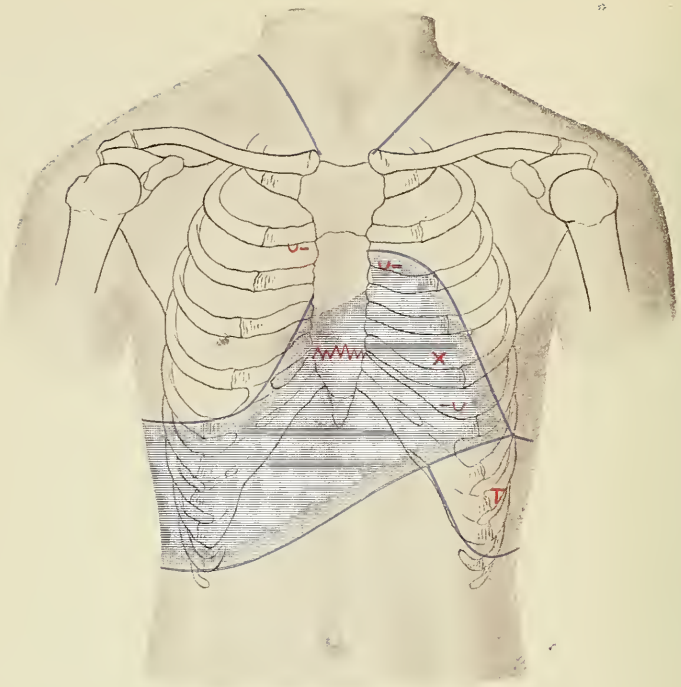
Diagnosis. Acute pericarditis without effusion is not recognized generally, because it is not sought for. In the larger number of cases, as previously intimated, there have been no indications of disease of the pericardium during life. If sought for, however, the diagnosis is usually easy. The *pericardial friction* may be mistaken for an *organic heart-murmur* or for *pleural* or *pleuro-pericardial friction*. It is often difficult to distinguish the to-and-fro friction from the murmurs of double aortic disease. If attention is paid to the general and local phenomena, the mistake is not likely to be made. The location of the murmurs in organic heart disease, the direction of the transmission, the character of the murmur, the peculiar character of the pulse, and the secondary effects upon the muscles of the heart, point to the diagnosis of valvular lesion. The pleuro-pericardial friction which simulates pericardial friction usually occurs in the course of phthisis or pleuropneumonia. It is modified by respiratory movement: (1) It may disappear, or at least diminish, if the breath is held; (2) a full expiration may cause its disappearance. While it is of cardiac rhythm it is modified by the respiratory rhythm, so that on inspiration it is usually more marked. The pleuro-pericardial friction is not so strikingly modified by position. *Pleural Friction.* This is of respiratory rhythm and ceases with cessation of breathing. The pericardial friction persists even if the breath is held.

Pericarditis with Effusion.

I know of no affection which is more frequently overlooked during life than pericardial effusion This is because it develops without symptoms. In plastic pericarditis we have referred to the occurrence of *pain*. This may occur before the effusion in the latter form, but is

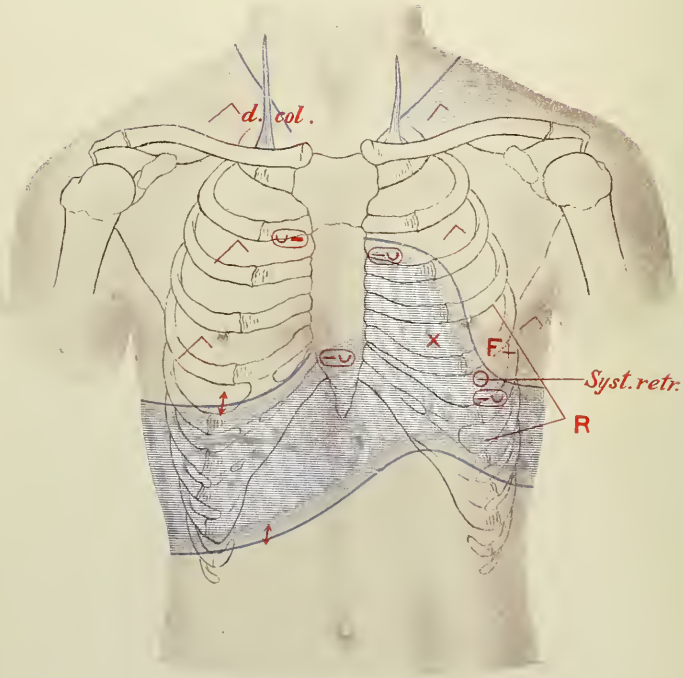
PLATE XXVIII.

FIG. 1.



Pericarditis With Effusion.

FIG. 2.



Adherent Pericardium. Chronic Left-Sided Pleurisy.

usually moderate. As with dry pericarditis, however, it may, in rare instances, be very severe, anginous in character, and be increased by pressure over the heart or on the pit of the stomach.

The *symptoms* are usually due to the special character of the inflammation and the presence of fluid in the pericardium.

1. General Symptoms. In non-suppurative cases the symptoms are usually cerebral. Delirium may be moderate or maniacal. It must not be confounded with the delirium which occurs in the course of acute rheumatism with hyperpyrexia. In addition, choreiform movements have been described. They may, however, be of rheumatic origin. Other cerebral symptoms, as hemiplegia and convulsive attacks, occur in the course of pericarditis, probably due to an associated endocarditis, causing embolism. In some cases albuminuria is found.

The general symptoms of pericardial effusion depend upon the nature of the primary disease and the character of the fluid. In *tuberculous pericarditis*, emaciation, irregular fever, sweats and prostration ensue. In *purulent pericarditis* there may be recurring chills with a temperature-range decidedly intermitting, along with other phenomena of purulent accumulation. In a case recently seen (1895) the patient was extremely debilitated and prostrated on account of pneumonia following influenza. He was extremely anæmic, and the blood-count showed diminution of red cells to one-half without other change. Every fourth day after a chill the temperature would rise to 103° or 104°. A friction-sound was detected after the second chill. It disappeared, but the physical signs of effusion were not positive. From the first the heart's action was so weak that the sounds were scarcely discernible. At the autopsy four or five ounces of pus were found in the pericardial sac. The purulent accumulation was the only lesion to account for the symptoms, and, we would say now, was no doubt a pneumococcus infection.

2. Local Symptoms The local symptoms are due to the accumulation of fluid within the pericardium. *Dyspnœa* is the most common. The degree depends upon the amount of effusion. If the latter is large, there may be extreme orthopnœa ; if the effusion is present for a considerable time, it may give rise to no symptoms. *Dysphagia.* In large effusions this may occur, on account of pressure upon the œsophagus. *Altered Cardiac Rhythm.* The effect of the effusion upon the heart is to interfere with its action. Although usually regular, on the slightest exertion or the least excitement it palpates violently or becomes irregular. The heart's action is increased in frequency ; when the effusion is very large it may be not only irregular, but also intermittent. *Aphonia* may occur from pressure upon the recurrent laryngeal nerve. *Cough* of an irritative character is sometimes noted. The *pulsus paradoxus* may be present.

3. Physical Signs. (Plate XXVIII., Fig. 1.) *Inspection.* There is bulging of the præcordia, particularly in children. The ribs and interspaces are prominent. In adults the interspaces are even with or distended beyond the surface of the ribs, and are sometimes widened.

The enlargement may extend to the antero-lateral region of the left chest. The large effusion interferes with expansion of the lung on the left side, and hence movement is diminished. The epigastrium may be prominent, on account of displacement downward of the diaphragm and liver. The apex-beat is absent or faintly seen, displaced upward and to the left. It does not extend as near the left border of dulness as in dilatation. It may be seen in the fourth interspace, or a faint impulse may be observed in the second and third interspaces beyond the mid-clavicular line.

Palpation. The impulse is feeble and diminishes in force as the effusion increases. The position of the apex as determined by inspection is confirmed. Ewart points out that the first rib is palpable at its sternal attachment in pericardial effusion. The pericardial friction which may have been present at first disappears with the effusion. Fluctuation may be detected in large effusions. The liver in large effusions is depressed and readily palpable.

Percussion. The area of præcordial dulness is increased. There is increase of the lateral boundaries and great increase of *absolute dulness.* The increase of area is usually in all directions, although increase of the dulness upward and to the left only is very common. It may extend as high as the second rib. As pointed out by Rotch, dulness in the fifth right interspace in the angle formed by the right border of the heart and the right lobe of the liver is common in effusion. It may be an early sign of effusion. Ebstein calls this region the *cardio-hepatic triangle,* and points out that the dulness is absolute in effusion, although impaired in normal states because of proximity to the liver.

Pulmonary resonance is modified posteriorly in large effusions. The dulness in large effusion includes the axillary region, so that it may simulate a pleural effusion. The dulness, however, does not extend below the eighth rib in this region, whereas, in pleural effusion, dulness always extends to the bottom of the pleural sac. In a large pericardial effusion the semilunar space of Traube is obliterated.

Auscultation. The sounds are feeble and distant. They may be scarcely heard at all over the præcordial region. The sounds at the base of the heart are diminished in intensity. If a friction-sound was heard at the beginning, it disappears entirely as the effusion is poured out. In moderate effusions the friction may be heard when the erect posture is assumed.

It must not be forgotten that the physical signs, and especially the change in impulse and the area of præcordial dulness, are modified by the position of the effusion. Accumulations occur behind the heart or above it, and in these situations interfere least with the displacement or the enfeeblement of the apex-beat. The area of dulness, however, is increased upward.

In cases of large effusion the compression of the lung may cause bronchial breathing to be heard posteriorly or in the axillary region. In a case under my care the diagnosis of pericardial effusion was readily made, but the enormous effusion so markedly simulated an

effusion into the pleural cavity that both serous cavities were believed to contain fluid. Aspiration was performed in the sixth interspace in the anterior axillary line. The fluid was removed from the pericardium, as was afterward determined. During life pressure-signs—laryngeal stridor, difficulty of deglutition, and extreme dyspnœa—were present. Early vomiting, epigastric pain and tenderness, slight delirium, albuminuria, and an excessively weak, rapid pulse occurred in the course of the disease. The patient was a male, twenty years of age. The effusion was due to tuberculous pericarditis, secondary to tuberculosis of the bronchial glands. The physical signs were prominence of the præcordia; bulging of the interspaces on the left side; diminished expansion of the left side—anteriorly, laterally, and posteriorly; increased expansion at the extreme apex of the lung. The vocal fremitus was absent below the second interspace in front, below the third in the axilla, and diminished below the spine of the scapula behind. There was dulness from the second left rib in front to the margin of the thorax; from the fourth to the eighth rib in the axilla; below the eighth rib, tympany. The dulness extended beyond the margin of the sternum on the right side, almost to the right nipple-line, in the fourth and fifth interspaces. Posteriorly, dulness from the middle of the scapula to the base of the thorax, except along the vertebræ, where, from the seventh to the ninth rib, there was tympany. The physical signs of pericardial effusion on auscultation were marked. In the axilla the breath-sounds were absent. There were bronchial breathing and bronchophony behind from the spine of the scapula to the base along the vertebræ. They were most marked opposite the angle of the scapula, where the above-noted tympany was observed. In the mid-scapular line the breath-sounds diminished from above downward, and were absent at the base. It is seen that the physical signs of pleural effusion were present posteriorly and laterally, due to the enormous effusion. At the autopsy the pericardium was found to contain sixty-four ounces of fluid.

Pleural effusions may be excluded in similar cases by the absence of dulness in the axillary region below the eighth rib; by increase in dulness beyond the right edge of the sternum; and, at the same time, by the absence of signs indicating dislocation of the heart to the right.

Diagnosis. Pericardial effusion must be distinguished from *dilatation* of the heart. Although feeble and diffuse, the expansile shock of the impulse is more distinct than in dilatation. This distinction is not generally difficult if the patient has been under observation during the development of the disease. The impulse is not always absent in dilatation. Fluctuation may be detected. The area of dulness in dilatation does not extend upward except in cases in which the right auricle is enlarged. The dulness does not extend downward in dilatation without a similar displacement of the apex impulse. The shape of the dulness differs. In dilatation the dulness is square in shape; in effusion it is triangular or pear-shaped, with the base downward. In dilatation the sounds are accentuated, and are of a valvular character; in effusion they are muffled. Dilatation does not cause the pressure-

symptoms that occur in effusion. In pericardial effusion Bamberger's sign is of importance. When the patient is sitting upright an area of dulness about the size of a silver dollar can be marked out at the angle of the scapula. Over it, dulness, increased fremitus, and bronchial breathing are made out. If the patient leans forward, the dulness and the other signs of consolidation disappear, to return when he sits upright. In children pseudo-pleuritic signs are often present posteriorly—dulness, pleuritic friction, broncho-œgophony—but will disappear if the patient is put in the knee-chest posture. It is of diagnostic significance to have change of the rhythm and the character of the sound from day to day, or of its degree of loudness on movement of the patient.

In pericarditis with effusion, after its absorption, the friction-sound may return. Often it may disappear entirely and all signs of pericardial inflammation subside. In plastic pericarditis and pericarditis with effusion adhesion of the two layers of the pericardium may take place.

Effusions into the pericardial sac of serum, of blood, or of air, may take place without previous inflammation.

Hydro-pericardium. This may occur in the course of general dropsy from kidney or heart disease. It may not prove fatal of itself, but when associated with effusion in the pleural sac it contributes to the orthopnœa, which may cause death. Rarely after scarlet fever, effusion into the pericardial sac may be the only dropsical symptom. The physical signs are those of effusion. It is not attended by fever. It is frequently overlooked, because investigation beyond the pleura is not made after an effusion into that cavity has been found.

Hæmo-pericardium. This occurs on account of rupture of an aneurism of the first part of the aorta, of the heart itself, or of the coronary arteries. Wounds of the pericardium and heart cause hæmopericardium. The extension of the ulceration of malignant endocarditis to the surface may cause gradual effusion of blood. (See Keating, *Transactions of the Philadelphia Pathological Society.*) The physical signs are those of effusion. Death usually takes place before there has been time to make a sufficiently accurate examination to determine its presence. Rapid heart-failure due to compression is the cause of death. In the case referred to above, and in cases of rupture of the heart, the patient may live for many hours with dyspnœa and progressive weakening of the heart. In tuberculosis and cancer the effusion is frequently blood-stained.

Pneumo-pericardium. This occurs very rarely, and is due to perforation from without by a stab-wound, or perforation from the lung, œsophagus, or stomach. A purulent exudation may undergo decomposition, causing an accumulation of gas. If it arises from perforation, acute pericarditis is set up. The accumulation of gas causes tympany over the movable area of percussion-dulness. The most striking sign is noted on auscultation. Churning, splashing, or metallic sounds are heard, drowning the feeble heart-sounds. Death usually occurs quickly.

Adherent Pericardium. (Plate XXVIII., Fig. 2.) Chronic adhesive pericarditis may follow the acute form or, particularly if tubercu-

lous, develop independently and progress slowly. *Inspection* and *Palpation.* Indrawing of the interspaces may be seen at the time of the systole of the ventricles; even the ribs are said to be drawn in. This indrawing is most marked at the apex, and must not be confounded with the retraction that occurs in the third and fourth interspaces with the ventricular systole. The recession is synchronous with the systolic shock. In some cases the systolic movement over the præcordia is of an undulatory character. Walter Broadbent calls attention to systolic retraction of the back in the region of the eleventh or twelfth rib as a valuable sign. The apex is displaced outward and the area of impulse is increased. The increase in area of impulse is due to the hypertrophy which always attends universal adhesion of the pericardium. After the systole there is frequently felt a quick rebound, known as the diastolic shock, which is said to be characteristic of pericardial adhesions.

In pericardial adhesions Friedreich's sign, *collapse* of the *cervical veins,* during the diastole of the heart, is seen. We may also see inspiratory swelling (Kussmaul). In addition, the *pulsus paradoxus* is significant of the presence of pericardial adhesions, or rather of the dilatation that succeeds the adhesions. The pulse is small and feeble during inspiration, assuming greater strength during the period of expiration.

Percussion. The area of cardiac dulness is increased usually upward, extending as high as the first interspace. The area of dulness is frequently not modified by respiration—that is, it is not lessened when the patient takes a full breath, when the lungs should expand over the præcordial region. This is particularly the case when there is pleuritis associated with pericarditis, a common association in the large majority of cases.

Auscultation. On auscultation the signs vary. The sounds are due to hypertrophy or to dilatation; and it must not be forgotten that they frequently arise on account of pericardial adhesions. In the former condition the first and second sounds are accentuated; in the latter, a murmur may be heard at the apex, loud and systolic in time.

In pericardial adhesions the physical signs depend upon the condition of the heart muscle at the time of the examination. At first we have the physical signs of hypertrophy, with retraction of the interspaces, particularly at the apex, or the space at the xiphoid cartilage. This is particularly the case in young subjects. In the later period of the disease the physical signs of dilatation arise, indicated by increase in transverse dulness, enfeeblement of impulse and of sounds, with the development of a murmur at the apex, undulation of the veins in the neck, and the pulsus paradoxus. The physical signs of associate pleurisy aid in the recognition of adherent pericardium. Diminution of the breath-sounds, increase in the area of cardiac dulness, lessened fremitus in the neighborhood of the heart pointing to pleural thickening, are associate evidence. Sansom considers the presence of pulmonary tuberculosis of value, as pointing to the occurrence of pericardial adhesions, for the associate pleural adhesions are likely to be attended by tuberculous pericarditis.

I have learned to suspect adhesive pericarditis in a young subject the victim of valvulitis, when the symptoms do not yield to treatment—in short, when the heart is not affected by digitalis. Unfortunately, the physical signs are often not conclusive.

The *subjective* symptoms of adherent pericardium are those of dilatation or hypertrophy of the heart, whichever one of the two is in excess.

Indurative mediastino-pericarditis with adhesion may occur with or without fibrous inflammation and adhesion of the structures in the anterior mediastinum. The pericardium is adherent and thickened. Rarely the anterior mediastinum alone is a mass of fibrous inflammation. Peritonitis and perihepatitis may be found. The entire process may be tuberculous. The *symptoms* are dyspnœa, venous engorgement, cyanosis, enlargement of the liver, ascites, and dropsy. The *physical signs* are those of extreme cardiac dilatation; the pulsus paradoxus; collapsing jugular veins during diastole, due to the dragging upon the innominate veins and cava by the fibrous adhesions, or to stretching and narrowing of the aortic arch by these adhesions; or inspiratory swelling of the veins of the neck. A friction-sound, systolic in time, heard over the sternum, increased when the arm is held up—*mediastinal friction*, so called, has been described in this affection.

It usually follows an acute chest-affection, occurs most frequently in young adults, and in males. It should also always be suspected in cases of dilatation and valvulitis in which compensation does not take place, notwithstanding the best treatment.

Endocarditis.

Endocarditis may be acute or chronic. In either form it is usually secondary. The acute form is divided into simple and so-called malignant, infectious, or mycotic endocarditis.

Simple Endocarditis. Acute endocarditis rarely occurs primarily. It usually occurs secondarily to general morbid processes. The pathological antecedents are acute rheumatism, tonsillitis, whooping-cough, scarlet fever, gonorrhœa, rarely smallpox and typhoid fever. It is of common occurrence in pneumonia and tuberculosis. It is frequent in chorea. In the simple form it occurs in septic inflammations and in debilitating diseases, as cancer. It may occur in gout and develop in the course of Bright's disease.

Symptoms. The symptoms of simple endocarditis are scarcely observed during the early course of the disease. The process is latent, and there are no indications of cardiac disease. The physical signs alone betray its presence. Unless these are sought for the disease is overlooked. The subjective symptoms are negative. In the course of rheumatism or chorea, or during convalescence from the former, the patient may complain of palpitation, and increased frequency and irregularity of the heart. At the same time there may be a rise in temperature, not attended by any increase of the rheumatic symptoms, which should call attention to the cardiac complication. The rise is not marked, and may not assert itself during the severity of the disease.

Physical Signs. On examination a murmur is detected in one of the cardiac areas. The murmur is soft, low in pitch, and follows the laws of transmission, according to its situation. Instead of a distinct murmur a roughening of the first sound alone may be heard. Preceding the murmur the heart's action may be quickened and arhythmical; the first sound may change in character from day to day or be accentuated; the second reduplicated at the apex and accentuated. The new sounds may disappear at first when the patient sits up; later they persist. The murmur must not be mistaken for the murmur at the apex in cardiac dilatation; or the murmur which may be heard in the course of fevers; or the murmur of anæmia, which may rapidly develop in rheumatism and other affections.

Malignant Endocarditis. Unlike simple endocarditis, the malignant form very rarely develops in the course of rheumatism and chorea. (See the Infections.) It occurs more frequently in pneumonia than in any other disease. It arises in the course of erysipelas, septicæmia, puerperal fever, and gonorrhœa. It may occur in dysentery. It is usually a streptococcus infection.

Symptoms. The symptoms are (1) those due to the morbid process —the infection; (2) the physical signs; (3) those due to emboli. The *general symptoms* due to the specific morbid process are septic in nature. The febrile phenomena may be one of four groups: (1) The fever is paroxysmal. Chills and fever occur daily or at intervals of two or three days, resembling types of malarial fever. Each paroxysm is attended by profuse sweats. Rapid exhaustion ensues. The fever, instead of being distinctly intermittent, may be irregularly intermittent. (2) The fever is excessive and continued, and a typhoid state frequently sets in. The temperature is irregular; extreme prostration, low delirium, sordes, subsultus, and other symptoms of that state arise. (3) The fever is moderate and continued. Physical examination, however, reveals the presence of marked endocarditis. In this group chronic heart disease has usually preceded the affection. The duration may be prolonged. (4) The fever may be remittent. Petechial *rashes* and *erythema* are common, so that, as pointed out by Osler, the disease may resemble the eruptive fevers. The *sweating* is profuse, contributing to the profound exhaustion which usually ensues. A septic *diarrhœa* occurs. In a few rapidly fatal cases *jaundice* has occurred. Again, the symptoms may be almost exclusively cerebral, resembling cerebro-spinal or basilar meningitis.

The *embolic phenomena* are due to escape into the blood-current of soft vegetations from the valves of the left heart (for the right heart is rarely affected), which are carried by the blood-stream into distant points of the circulation. Emboli occur in the brain, producing aphasia or hemiplegia; they occur in the retina, causing some complaint as to vision, but are accurately recognized by ophthalmoscopic examination. They occur in the kidneys, producing bloody urine and renal pain. In nearly all cases the spleen is the seat of embolism, and in some instances infarctions may take place in this organ alone. The spleen is always enlarged, and the infarct may cause pain and increased

tenderness on pressure. Emboli in the skin and mucous membranes present the most striking phenomena. The hemorrhages underneath the skin are minute. They are seen in the extremities, but may also be found on the trunk. They occur in the mucous membranes, as those of the mouth and tongue. They are seen in the bulbar conjunctivæ, and in the conjunctivæ of the lids.

Physical Signs. Repeated examinations are necessary in some cases, to determine the presence of a murmur, or to decide whether a previously existing organic lesion is the seat of an acute process. Variations in the character of the murmur from day to day are characteristic of malignant endocarditis. In organic *heart disease* with dilatation and failure of compensation, irregular fever followed by embolic phenomena points to the occurrence of an infectious process on the antecedent valvulitis.

Diagnosis. This form of endocarditis is of infectious origin. The diagnosis rests upon proof that an infection is present, and is made by the methods described in Chapter XIX., Part I., which should be reviewed by the reader. The history of an infection in some part of the body is most important in the diagnosis. The presence of the infection, as well as its nature, may be disclosed by an examination of the blood. When embolic phenomena are present the diagnosis is made without much difficulty. The more pronounced general symptoms distinguish it from *simple endocarditis.* The temperature-range, the septic and typhoid symptoms, belong to the malignant form. The more prolonged cases with moderately continuous fever, without apparent primary cause, are frequently confounded with *typhoid fever.* This is readily appreciated when the symptoms of the two are compared. In both there is fever of a continued type, with the symptoms of the typhoid state, including delirium. In both there are enlargement of the spleen, diarrhœa, and abdominal tenderness. In both there may be infarctions, although they are extremely rare in typhoid fever, and only occur late in the disease. In both there is progressive exhaustion. But in endocarditis the onset may be more abrupt. The fever does not present the regularity of type that is seen in the development of typhoid. In endocarditis there is more chest oppression and dyspnœa early in the disease than in typhoid fever. In endocarditis the source of the infection may be discovered in the genito-urinary organs, the lungs, the bones, etc. The diazo-reaction is found in typhoid fever after the fifth day, but rarely, if ever, in endocarditis. The results of bacteriological examination, and especially of *serum diagnosis,* distinguish the two affections. This ought to be of value in endocarditis, because the process is usually due to a staphylococcus or streptococcus infection. Either micro-organism may be found in any suppurations which may possibly be present or in the blood. In a child recently seen by me in the relapse of an attack of typhoid fever, malignant endocarditis was thought to be present, because of a loud and rough murmur at the pulmonary orifice. Fortunately the murmur was present in the apyretic period, and as the child was anæmic its exaggeration was ascribed to the fever.

Malignant endocarditis must be distinguished from *cerebro-spinal fever*, and from *smallpox* of the hemorrhagic type. We must rely on the local cardiac symptoms and physical signs, and the preponderance of these over the other symptoms. Of course, the prevalence of an epidemic and a history of exposure are of service in the distinction between the diseases. Examination of the blood excludes the forms of *malaria* which formerly were mistaken for endocarditis.

Chronic Endocarditis. Chronic endocarditis may follow the acute form or develop in the course of atheroma or of endarteritis due to alcoholism, the poison of syphilis or of gout. If associated with endarteritis, the endocardial change may be part of the general degenerative changes which occur in the aging process. It may be of dynamic origin, often following prolonged heavy muscular exertion, by which the valves, particularly at the aortic orifice, have been subjected to strain. The process is slow and insidious, and leads to the changes in the valve-segments which constitute chronic valvular disease.

Symptoms. The symptoms of chronic, or sclerotic, endocarditis are the symptoms of chronic valvular disease. Insufficiency or obstruction, or both combined, take place at the affected valve-orifice. The outflow of blood is retarded in obstruction. Backward flow, or regurgitation, takes place in insufficiency in the opposite direction from the normal blood-current. When there is obstruction hypertrophy usually develops to meet it. If the obstruction is moderate, and the person remains in good health, the hypertrophy is sufficient to overcome the obstruction. In this manner the effect of the valve lesion is compensated. On the other hand, when blood is permitted to flow by regurgitation backward into the cavity—that is, in the opposite direction to its usual course—it meets a blood-current flowing to this cavity in the normal direction, and the result is overdistention, or overfilling, of the cavity. Dilatation ensues, and may persist. If the regurgitation takes place suddenly, the dilatation continues; if gradually, as in chronic endocarditis, the dilatation is attended with hypertrophy. Thus, when there is regurgitation from the left ventricle into the left auricle, on account of incompetency at the mitral orifice, the auricle becomes overdistended with blood, for it is filling with blood from the pulmonary veins at the same time. This overdistention can only be overcome by some hypertrophy. When this is not sufficient the blood is obstructed in the pulmonary circulation, with the consequences hereafter to be mentioned.

The symptoms of chronic endocarditis are latent if the lesions are compensated; if not, symptoms of failure in compensation occur or *dilatation* of the heart arises. The physical signs are those of *chronic valvulitis*. The character of the signs depends upon the lesion of the affected valve.

Disease of the Coronary Arteries.

Atheroma, associated with the process in other vessels, or distinctly localized to the coronary arteries, affects these vessels. Its causal factors are those of endarteritis elsewhere. Its influence on the nutri-

tion of the heart, either by sudden obstruction of the vessels by an embolus or by their gradual closure, is apparent.

Symptoms. If an atheromatous coronary artery is suddenly obstructed by an embolus, death may be immediate. This is a common cause of *sudden death*. In other instances thrombosis may take place, followed by anæmic infarction, myocarditis, and mural aneurism. In this class of cases the onset of the symptoms may be sudden. Præcordial oppression or angina pectoris may be the first indication. Succeeding this, dyspnœa, dilatation of the heart, and venous stasis occur. The presence of an aneurism may be made out. The heart's action is persistently rapid and may be arhythmical. If there has not been previous valvulitis, no murmurs are heard until dilatation ensues. The patient may live three or four weeks, or as many months.

In a third group of cases occlusion, either from the endarteritis or from a slowly forming thrombus, is so gradual as to lead to myocarditis only with the attending symptoms.

Diagnosis. Unfortunately, too often the diagnosis can only be provisional. Sudden death may be attributed to coronary artery disease if there has been a history of previous attacks of angina, if there is evidence of arterial disease elsewhere, and if dyspnœa or anginoid symptoms preceded the fatal termination. Thrombosis, secondary to atheroma, may be suspected if a patient, in whom there is no valvular disease, no pulmonary or renal discase, is seized with angina pectoris or dyspnœa; providing tachycardia and arhythmia follow, and in a short time cardiac dilatation, venous stasis, etc. In a male, aged forty-three years, without syphilis, but with a history of antecedent rheumatism, an attack of angina pectoris followed some unusual exertion. Prior to this he had been in the most perfect health. The attack was followed by dyspnœa and remarkably rapid heart-action without apparent cause. The physical signs of acute congestion of the lower lobe of the right lung followed within twenty-four hours of the attack of angina. The patient was ill three months. He improved somewhat, but rapidity of the heart's action and some stasis in the lung persisted. Gradually cardiac dilatation ensued, with a murmur in the tricuspid area. Death took place from pulmonary congestion. At the autopsy the coronary arteries were atheromatous; the left was filled with an old thrombus; there was extensive myocarditis and an aneurism of the left ventricle.

In another case, male, aged seventy-two years, with general atheroma but no valvulitis, sudden præcordial distress, tachycardia, and persistent dyspnœa were followed by cardiac dilatation, mitral incompetency, general anasarca.

I have said elsewhere, a persistently rapid pulse, uninfluenced by digitalis, indicates pericardial adhesion in the young; the same pulse uninfluenced by treatment points to coronary artery disease in the middle-aged and senile.

Myocarditis.

Myocarditis may be acute or chronic. The entire muscle or only a portion may be affected. General myocarditis is always acute. The

local form may be acute or chronic, depending upon the degree of the primary cause. The local variety is usually due to a thrombus in the terminal endings of the coronary artery, which cuts off the blood-supply. The changes are those of myocarditis, to which may be added necrosis of small areas and the development of aneurism. *Etiology.* Pathological antecedents of acute general myocarditis are the fevers, particularly typhoid and typhus fever, pneumonia, diphtheria, and septic fevers generally. Chronic myocarditis is usually associated with atheroma, one of the causes of which occurs in the later stages of Bright's disease. (See Atheroma.) The result of myocarditis, when acute, is dilatation of the heart, fatty heart, or aneurism of the heart. Chronic myocarditis is followed by fatty heart, by dilatation, by the so-called fibroid heart or fibrous myocarditis, and by aneurism. The above facts in etiology are important in diagnosis.

Symptoms. The symptoms of *acute* myocarditis are vague. In the course of, or in the convalescence from, an infection the patient may complain of some oppression in the præcordia and suffer from dyspnœa; attacks of syncope may occur, and sighing may be frequent. The pulse becomes more rapid and weak, but is usually not irregular. The circulation is much depressed, the hands may be cold, the face pallid. These symptoms may be accounted for by the extreme exhaustion alone that follows fever. No doubt some myocarditis accounting for the symptoms exists in all cases, particularly if there is prolonged high temperature. Often the patient does not complain of any cardiac symptoms. Death takes place suddenly, either in the course of the disease or after it has spent its force, from acute dilatation or cardiac paralysis. This is particularly true in pneumonia and diphtheria. In the latter affection the sudden appearance of cardiac symptoms, dyspnœa, cyanosis, and cold extremities may be due to paralysis of the heart.

Physical Signs. Enfeeblement of the heart-sounds, sometimes with accentuation of the mitral first sound, is observed. The impulse and apex-beat are scarcely perceptible, or absent altogether. If acute dilatation supervenes the area of dulness may be increased.

The symptoms of *chronic myocarditis* are obscure and indefinite, and in the majority of cases depend upon the secondary changes that have taken place in the heart muscle. If there is *atrophy* of the fibroid heart, the pulse is feeble, slow, and irregular. It may be as slow as thirty or forty beats to the minute. Irregularity is not necessarily present, but intermittency is of frequent occurrence. The patient complains of dyspnœa aggravated by exertion. Attacks of angina pectoris are likely to occur. The symptoms of dilatation of the heart may ensue later, with œdema, cyanosis, and congestions. A symptom-complex, known as the Stokes-Adams syndrome, is often seen, characterized by dyspnœa, coma, and slow pulse—a pseudo-apoplexy. In *fatty degeneration* of the heart the pulse is increased in frequency; there are cardiac irregularity, palpitation, and dyspnœa. These, however, are also the symptoms of dilatation, which usually succeeds the degeneration. The heart-sounds are weak. If dilatation has set in, a murmur is heard

at the apex, with gallop-rhythm of the heart. In fatty degeneration attacks of collapse with slow pulse are common. Shortness of breath on exertion may occur. Cardiac asthma occurs at night, and sighing and yawning are of frequent occurrence during the day. The patient usually sleeps badly. The cerebral functions are more or less in abeyance, the action of the mind is sluggish; the patient may have delusions or become maniacal. Cheyne-Stokes breathing was formerly thought to be of diagnostic significance.

Chronic myocarditis must be distinguished from *fatty overgrowth* of the heart. This cardiac change is frequently seen in brewers and saloon-keepers, and is usually associated with obesity. The pulse may be feeble, the heart-sounds weak and muffled. The patients are subject to attacks of asthma, and frequently have bronchitis and emphysema. Vertigo is of common occurrence. Death may occur during syncope.

Aneurism of the Heart.

Aneurism of the valves, following endocarditis, cannot be recognized during life. Aneurism of the walls usually results from chronic myocarditis. The aneurism develops in the left ventricle at the apex. The symptoms are indefinite. In rare cases a marked bulging has been noted in the region of the apex, and the tumor may perforate the chest-wall. A projection beyond the normal line of cardiac dulness may be detected by stethoscopic or plessimetric percussion. The symptoms are those of myocarditis and of dilatation of the heart.

Rupture of the heart is one of the causes of sudden death, often without previous symptoms. The accident takes place during exertion. Quain collected one hundred cases, in seventy-one of which death took place without previous warning. In other instances there was a sense of anguish, and suffocation in the cardiac region. The physical signs of slowly developing pericardial effusion may be ascertained if the leakage from rupture is slow in progress.

Chronic Valvular Disease.

Valvular disease includes valvulitis and valvular incompetency; there is either obstruction or regurgitation at the orifices affected. Valvulitis may exist with or without symptoms; valvular incompetency is always accompanied by symptoms. Valvulitis implies organic disease of the valves; valvular incompetency, regurgitation through orifices, the valves of which cannot close it, but they may or may not be diseased. Valvulitis may be recognized by physical signs of (1) the lesion, (2) the secondary effects of the lesion on the heart and circulation—hypertrophy or dilatation. Valvular incompetency occurs usually in dilatation, and may be secondary to valvulitis. It is recognized by both signs and symptoms. Valvular disease is without symptoms as long as the heart-muscle enlarges sufficiently to keep in balance the impaired circulation; compensation is then said to be complete.

When compensation is broken we then have the subjective symptoms enumerated above, all in consequence of dilatation of the heart. It may be said that valvulitis is of no significance as long as compensation is perfect. To review—valvulitis may be attended by physical signs in the heart and vessels only, or by its own physical signs, the physical signs of dilatation, and the symptoms of the latter. In the consideration of valvular disease it is more profitable to take up the symptoms of each valve-lesion, bearing in mind that two or more of the valves may be diseased at the same time, or that both obstruction and regurgitation may be present at the same time at the same valve-orifice.

Aortic Regurgitation, Insufficiency or Incompetency. This may exist for a long time without presenting any symptoms. It occurs more frequently in men than in women, and is more common in the later periods of life. It may be due to congenital malformation, to acute endocarditis, or, as is most frequently the case, to chronic endocarditis, particularly when it follows strain or undue exertion; alcoholism and syphilis are also frequent antecedents. In rare cases it follows rupture of the valves. Relative insufficiency or incompetency is of very rare occurrence. Insufficiency is frequently combined with obstruction.

On account of regurgitation, or insufficiency, at the aortic orifice the blood falls directly into the left ventricle during the diastole. There is, first, a relative diminution in the amount of blood in the artery; and, second, an increased amount of blood in the ventricle, because the regurgitated column of blood meets the blood from the auricle which is filling the chamber at the same time. Dilatation of the left ventricle ensues, and is followed by hypertrophy. Dilated hypertrophy thus arises. The heart becomes enormously enlarged. This is one of the conditions in which enormous cardiac enlargement takes place—so-called *cor bovinum*. If this valve-lesion occurs at the period of life and from the causes above mentioned, it is attended by more or less sclerosis of the arteries.

Symptoms. They may be entirely absent as long as perfect compensation exists. This is particularly the case if there is but little general arterial sclerosis. Coincident lesions of other valves tend to break the compensation. The earlier symptoms are those due to arterial anæmia, particularly anæmia of the brain. They are headache, dizziness, and flashes of light before the eyes. The patient has an anæmic appearance, and soon begins to suffer from shortness of breath. This at first develops upon slight exertion. Palpitation and oppression about the chest are complained of, readily excited by undue exertion. Pain is a common symptom. It may be in the region of the præcordia, of a dull, aching character, and radiate to the neck and down the arms, particularly on the left side. The anginoid pains may be followed by attacks of true angina pectoris. The latter are more common in aortic regurgitation than in any other valve-lesion.

As compensation fails venous stasis occurs and the dyspnœa increases. The latter is worse at night and compels the patient to sleep in a semi-erect posture. Congestion of the lungs takes place, giving rise to cough. Hemorrhage occurs, but not so frequently as in mitral

disease. Œdema of the feet sets in, but general anasarca is not common. Œdema of the feet may be due to the attendant anæmia.

In aortic insufficiency sudden death is of common occurrence. This may take place at night during an attack of dyspnœa, or occur suddenly upon the slightest exertion, such as straining at stool, or ascending a height, or walking more quickly than usual.

The Physical Signs of Aortic Regurgitation. (Plate XXIX., Fig. 1.) *Inspection.* The apex beat is downward, outward, and to the left. It may be as low as the seventh interspace, and as far out as the anterior axillary line. The area of cardiac impulse is increased. It occupies the whole præcordia, and heaving of the lower half of the chest may be seen. In young subjects there is præcordial bulging.

Palpation. The impulse is strong and heaving. After compensation fails it is indefinite and wavy. A thrill, diastolic in time, may be felt if the hand is placed about the middle of the sternum.

Percussion. The area of dulness is increased. The extent is greater than that in any other valve-lesion, and the enlargement is more particularly downward and to the left.

Auscultation. At the second costal cartilage on the right a murmur is heard, diastolic in time. This may be its seat of maximum intensity. (See Fig. 175.) It is transmitted along the course of the sternum toward the apex. In some instances the seat of maximum intensity is at the fourth left costal cartilage, or even at the apex. The second sound is absent in the large majority of cases. In some instances, however, both murmur and second sound may be heard at the same time. Other murmurs also may be associated with aortic regurgitation, not always due to disease of the aortic valves :

1. A systolic murmur at the second costal cartilage on the right, transmitted into the vessels of the neck, short, rough, and high in pitch. It is due to roughening of the valve-segments, or to atheroma of the aorta.

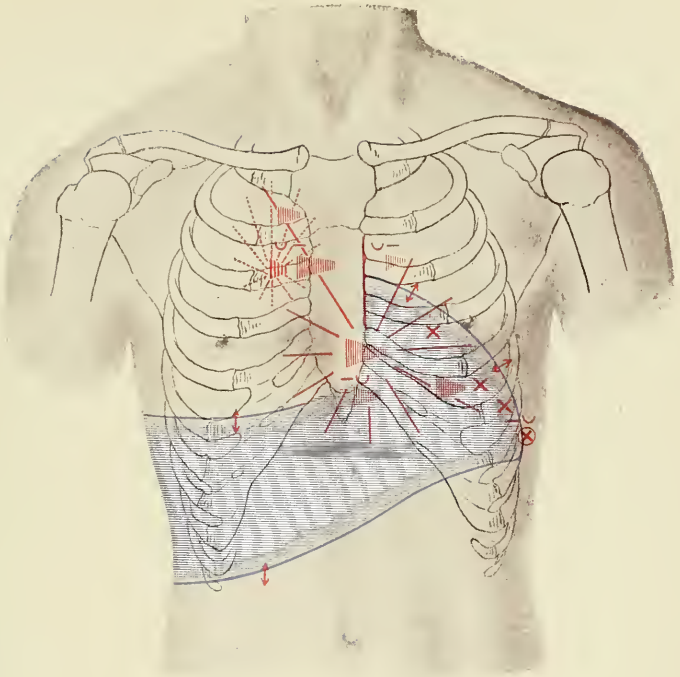
2. A murmur at the apex, rumbling in character, localized to this area, usually presystolic in time. It is the murmur described by Flint, who attributes it to flapping of the mitral segments, which during diastole are not forced back against the heart-wall, on account of the dilatation of the ventricle. They remain in the blood-current and produce relative narrowing.

3. A systolic murmur in the mitral area, low in pitch, due to dilatation. This occurs when failure in compensation takes place.

Examination of the Arteries. Pulsation of the peripheral vessels is more common in aortic regurgitation than in any other valve-lesion. The carotids throb, the temporals pulsate, the brachial and radial arteries are conspicuous. Pulsation of the retinal arteries is seen with the ophthalmoscope, and has often led to the recognition of the disease by the ophthalmologist who had been consulted for other conditions. The pulsation is of a jerking character ; in the neck it may simulate the pulsation of an aneurism. The aorta can be seen and felt at the suprasternal notch. The abdominal aorta pulsates vigorously in the epigastrium. The *pulse* is significant in aortic regurgitation. The so-called water-hammer, or Corrigan's, pulse is observed. The pulse is quick

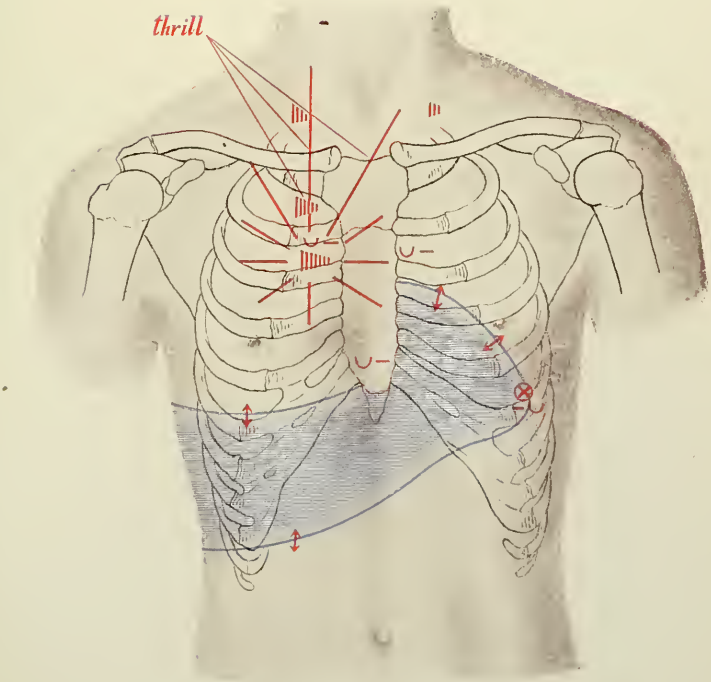
PLATE XXIX.

FIG. 1.



Aortic Regurgitation.

FIG. 2.



Aortic Obstruction.

and jerking, and after striking the finger immediately recedes. It is most marked when the arm is held up. On *auscultation* of the arteries double murmurs may be heard in the carotids and subclavians, and in rare instances they are present in the femorals. (See Pulse.)

The Capillary Pulse. This is seen beneath the finger-nails, or on the surface of the skin, as the forehead, when a line is drawn across it. The hyperæmia produced on either side of the line becomes alternately red and pale. Capillary pulse also occurs in anæmia, and at times in neurasthenia.

Aortic Obstruction. Aortic obstruction occurs in the aged, and with atheroma of the arteries. It causes some diminution in the amount of blood in the peripheral circulation, resulting in poor nutrition and the development of anæmia.

Symptoms. Anæmia develops first, and embolic phenomena may occur later. The symptoms may be latent until the occurrence of embolism. This accident is not uncommon, on account of the position of the aortic valve. The emboli are distributed throughout the arterial circuit, and may lodge in the brain, kidneys, or spleen. When the obstruction is pronounced the blood-supply in the arteries is diminished. Cerebral anæmia takes place, causing dizziness and fainting. Sleep is more disturbed than in other valve affections, because of the cerebral anæmia. Palpitation and cardiac pain occur, but are not so common as in aortic regurgitation. When compensation fails, dilatation of the left ventricle ensues, followed by pulmonary congestion and stasis in the systemic circulation.

The Physical Signs. (Plate XXIX., Fig. 2.) There is hypertrophy of the left ventricle. *Inspection.* The apex-beat is displaced downward and outward. The impulse is strong during the period of hypertrophy. When compensation fails the physical signs of dilatation ensue. In many cases, from the very first, there may be considerable hypertrophy without the visible impulse, because of associate emphysema, which is common to old men with this lesion.

Palpation. At the base of the heart, and in the aortic area, a thrill, systolic in time, may be felt. When present, it is usually very distinct, and is transmitted along the course of the vessels. The impulse is slow and heaving, if hypertrophy is present; if dilatation, feeble and indistinct.

Percussion. The area of dulness is increased, in the earlier stages, to the left and downward. After compensation is broken, dilatation with increased area of dulness ensues.

Auscultation. A murmur is heard of maximum intensity at the second costal cartilage to the right, systolic in time, and transmitted in the course of the bloodvessels. (See Fig. 174.) It is usually harsh and loud, but may be musical. As the heart weakens, the intensity of the murmur lessens and its roughening disappears. It becomes soft and low in pitch. The second sound, if there is no regurgitation, is muffled or may be absent. The *pulse* is small and regular. The tension is usually increased.

Diagnosis. A systolic murmur at the aortic orifice may be due to aortic obstruction, atheroma or dilatation of the aorta, ulcerative aor-

titis, or anæmia. Huchard describes a murmur in this situation, with vibratory thrill, due to aberrant chordæ tendineæ. The murmur of aortic stenosis is distinguished from the others by its character, by the presence of thrill, by the character of the pulse, and by its association with hypertrophy of the left ventricle. A murmur due to atheroma of the aorta, particularly in the course of renal disease, is also associated with hypertrophy of the left ventricle. The diagnosis from aortic obstruction is often difficult or impossible. Slowness of the pulse is more characteristic of aortic obstruction. The murmur of anæmia is softer and low in pitch. There is no thrill, and the left ventricle is not hypertrophied. Anæmic murmurs may be heard elsewhere. In atheroma the second sound is usually accentuated, and in anæmia also it is intensified.

Mitral Incompetency or Regurgitation. The regurgitation may be due to disease of the valves (organic) from previous endocarditis, or to inability of the segments to close the orifice (incompetency), which has become enlarged as part of the dilatation of the cavities. The latter occurs in dilatation of the left ventricle. It takes place when the muscle is weak in fevers and in anæmia. It is thus seen that the murmur of mitral insufficiency is one of the most commonly observed of all valve-murmurs. Its ready production and often equally ready removal with treatment make it the least serious. It must not be forgotten that insufficiency from disease of the valves and from disease of the muscles must, if possible, be distinguished from each other. The history of the case is essential in determining the diagnosis.

Disease at the mitral orifice producing insufficiency has more serious effect upon the pulmonic and arterial circulation than disease at any of the other orifices. These effects must be understood in order to appreciate the symptoms of mitral incompetency. They are as follows: 1. With each systolic contraction the blood flows back, on account of the insufficiency, to the auricle, where it soon meets a volume of blood coming from the lungs. The combined volumes of blood overdistend the auricle. Dilatation ensues, and because of increased work to get rid of the increased contents, hypertrophy follows. Dilated hypertrophy of the left auricle is the first effect. 2. As a result of the above, a larger amount of blood is forced from the left auricle into the left ventricle; dilatation and subsequent hypertrophy of this chamber also follow, to remove the fluid. 3. On account of the overdistended auricle the pulmonary veins are not fully emptied during the diastole of that chamber. The veins are therefore engorged and interfere with the flow of blood through the pulmonary circuit. In consequence of the impeded flow of blood the vessels in the pulmonary circuit are dilated and overdistended. The right ventricle is compelled to act more vigorously, and even then cannot empty itself freely. Dilatation and hypertrophy of the right ventricle ensue. 4. This causes obstruction of the flow of blood from the right auricle to the right ventricle; dilatation and hypertrophy of its chambers follow. If perfect *compensation* ensues through hypertrophy of both ventricles, engorgement in the lungs may not be observed. Moreover, the left ventricle is allowed to send out sufficient blood to supply the wants of

the system. This compensation may continue for years. If it fails, either from increase in the valve-lesion, or valvular incompetency, or from weakening of the muscle, a normal amount of blood is not distributed throughout the aortic area, but is thrown back upon (1) the left auricle; (2) the pulmonary circulation; (3) the right heart; and, finally, the systemic veins. For a time the pulmonary circuit will alone be engorged, subsequently the systemic veins become congested because of dilatation of the right auricle and incompetency of the tricuspid valves. We then have the secondary effects of stasis upon the various organs of the body, with cyanotic induration and the development of dropsies. *Mitral incompetency* without disease of the valves is of frequent occurrence in emphysema of the lungs and in Bright's disease, and is a condition which always attends hypertrophy and dilatation, or may take place from various causes. (See Hypertrophy and Dilatation.)

Symptoms. As to the general symptoms: In a large number of cases perfect compensation may continue for a long time. No subjective symptoms arise nor are there symptoms due to dilatation. If compensation is not perfectly effected from the first, or is broken suddenly or gradually, the symptoms of dilatation arise.

In patients in whom compensation remains only fairly good we have the characteristic appearances of heart disease. It is to this class of patients that the general descriptions of heart disease apply. The face is pale and pinched, the lips and ears dusky, the capillaries of the cheeks enlarged, the finger-nails clubbed, particularly in children; shortness of breath on exertion may be the only symptom complained of, and this may exist for years. The patients are, however, liable to attacks of bronchitis and of pulmonary hemorrhage. Palpitation may occur in this as in other forms of heart disease, and from the same cause.

When the compensation is broken, symptoms referable to the heart and to engorgement of systemic and pulmonary veins occur. Of the former palpitation with a sense of oppression is the most common; pain is rare.

Venous engorgement leads to *congestions, cyanosis,* and *dropsies.* We now have the symptoms of dilated right heart superadded. The lungs are the first to be congested. Dyspnœa becomes constant and is aggravated by exertion. Cough is present, excited by exertion or speaking. With the cough there is bloody expectoration. Cyanosis occurs. Congestion of other organs follows. The liver is enlarged; obstruction in the portal area is prominent; chronic gastritis or gastro-intestinal catarrh ensues. The spleen is enlarged; ascites develops, and hemorrhoids and congestion in the rest of the portal area are seen. The kidneys are congested; the urine is scanty, albuminous, and contains casts and blood-corpuscles. At the same time that the internal viscera are congested dropsies take place, beginning in the feet and extending to the rest of the body. Dropsy may have been present in the feet before symptoms of portal congestion ensued.

The patient may be relieved and compensation continue for a long time. Frequent attacks of dilatation of this character may take place, their recurrence being due to lack of care in hygienic matters, or

failure in health from other causes. Finally, however, the compensation cannot be restored; the stases persist; the dropsies become more marked, and the symptoms of cyanotic induration and secondary scleroses of the internal organs follow. It must not be forgotten that this is the chief form of organic heart disease seen in children.

Physical Signs. (Plate XXX., Fig. 1.) On *inspection* the præcordial area appears prominent; the apex-beat is displaced to the left and downward, rarely below the sixth interspace. It may extend to the anterior axillary line. The cervical veins pulsate and are distended. The area of impulse is increased.

Palpation. The character of the impulse depends upon the stage of the disease at which the case is examined. At the time of full compensation it is strong and even. When this is broken, it is feeble and diffuse. A thrill is extremely rare.

The Bloodvessels. The amount of blood in the arteries is diminished. There is notable absence of visible pulsation in the arteries. The pulse at first is full and regular. It is notably small in volume and soft. As soon as failure of compensation takes place the pulse becomes irregular. The irregularity may be that of time as well as of volume.

Percussion. The area of dulness is increased to the left. The transverse diameter of the heart is much increased because of dilatation of both chambers. The area extends beyond the right margin of the sternum to the extent of an inch or more and to the left as far as the mid-clavicular line, sometimes to the anterior axillary line. The cardio-hepatic triangle is preserved.

Auscultation At the apex, *the mitral area*, a murmur is heard. The point of maximum intensity is in this region. It is systolic in time; it may replace the first sound entirely. It may be soft and low in pitch, or rough, high in pitch, even musical in character. It is transmitted to the axilla and the angle of the scapula. (See Fig. 171.) In some instances it may be heard loudest along the left border of the sternum. The pulmonary second sound is accentuated; the accentuation is loudest in the pulmonary area at the second left interspace. It may be very loud over the right ventricle, between the parasternal line and the left edge of the sternum. The murmur of mitral insufficiency is modified by the position of the patient and intensified after exertion. It may be present when the patient is lying down, and disappear in an erect posture. It may disappear when the patient is quiet and return after exertion. Other murmurs are sometimes heard:

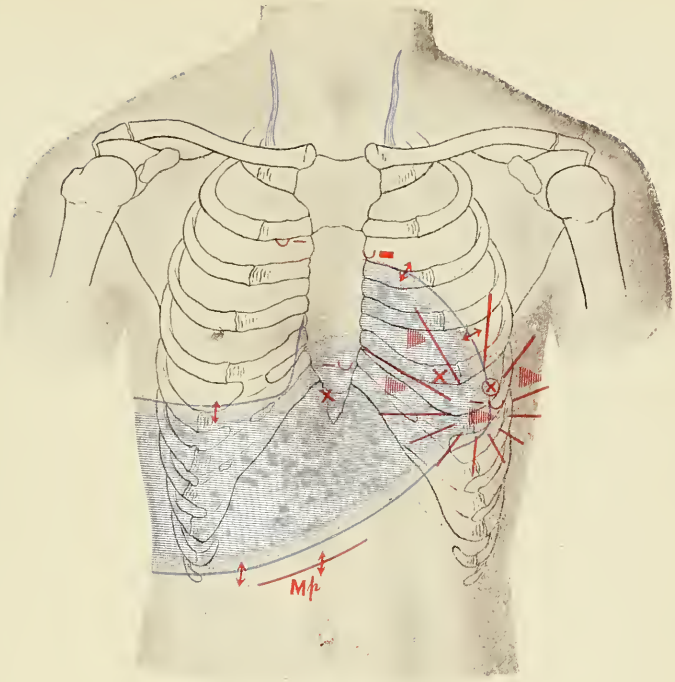
1. A presystolic murmur, soft or rumbling. 2. When dilatation ensues a low-pitched systolic murmur is heard at the ensiform cartilage and at the lower left border of the sternum. It is due to tricuspid regurgitation.

Of special *diagnostic significance* are: the position of the murmur and the direction of its transmission; accentuation of the pulmonary second sound; enlargement of the transverse diameter of the heart, due to dilatation of both ventricles.

Diagnosis. This is usually easy if the physical signs are sought for. Very often examination of the heart is neglected, and the patient

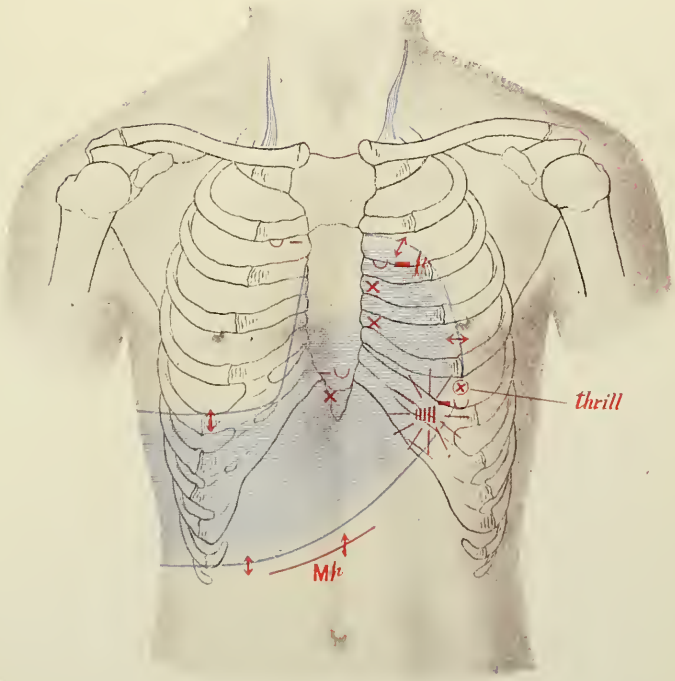
PLATE XXX.

FIG. 1.



Mitral Regurgitation.

FIG. 2.



Mitral Stenosis.

is treated for the symptoms that arise from congestion of the viscera. We have often seen chronic gastritis or gastro-intestinal catarrh, due to mitral insufficiency, not relieved because the primary lesions had not been ascertained. In the same way cardiac cough or dyspnœa may be overlooked. It is important in the diagnosis to determine, if possible, the nature of the insufficiency, whether it is due to disease or incompetency of the valves. As previously mentioned, the history is possibly the only means by which a diagnosis can be made. If a mitral murmur ensues in old people, in whom there has been physical cause for the development of dilatation and hypertrophy, as in emphysema or arterio-sclerosis, it is usually due to relative incompetency of the valve. It must not be forgotten that the mitral area is the seat of a number of murmurs due to various causes. (See Auscultation.)

Mitral Stenosis. Obstruction to the flow of blood from the auricle to the ventricle is due to valvulitis, or endocarditis, and particularly the endocarditis of early life. It is of much more frequent occurrence in women than aortic disease. It is much more often seen in young adults and children, because its etiological factors, rheumatism and chorea, are then more prevalent.

On account of the obstruction at the orifice changes ensue in the auricle. These changes depend in a measure upon the nature of the lesion. In the so-called buttonhole contraction they are very marked. The orifice may be so obliterated in rare cases as to admit only a small probe. Dilatation and hypertrophy of the left auricle ensue if the valve-changes take place gradually. The walls of the auricle are thickened to three or four times their natural size. On account of the dilatation of this auricle the outflow from the pulmonary veins is impeded, which in turn obstructs the circulation of blood through the lungs. As a consequence, dilatation and hypertrophy of the right ventricle occur. As a result of this we have, later on, the occurrence of relative incompetency at the tricuspid orifice, with engorgement of the systemic veins. The left ventricle does not take part in any changes. It retains its normal size, but it may look small in comparison with the right ventricle.

Symptoms. If hypertrophy of the right ventricle ensues, the compensation may be sufficient to prevent the occurrence of symptoms for many years. The disease may exist for a number of years without discomfort to the patient. Because of its rheumatic origin a fresh endocarditis may develop, particularly as most of the subjects are young. The old valve lesion invites infection, and so a recurrent form of endocarditis is induced. If fresh endocarditis occurs, embolic symptoms are likely to follow. Embolism takes place particularly in the brain, causing hemiplegia or aphasia. When failure of compensation takes place the symptoms described in mitral incompetency arise. They are the symptoms of dilatation of the heart, and may recur frequently during a long period of years.

Dropsy, however, is not so common as in mitral regurgitation. Visceral stases are common when compensation fails, and in many cases we find enlargement of the liver continuing for a long period. Ascites may in rare cases be the only manifestation of mitral obstruction.

Physical Signs. (Plate XXX., Fig. 2.) The physical signs of mitral obstruction are more striking and more diagnostic of the lesion than the physical signs of any other form of organic heart disease.

Inspection. As the disease develops in children with soft ribs the local deformities are very marked. For the same reason præcordial bulging is more prominent. Because the right ventricle is hypertrophied, the sternum and the fourth, fifth, and sixth costal cartilages protrude. The apex impulse is not usually displaced, certainly not beyond the mid-clavicular line. The impulse is not marked at the apex. In the third and fourth interspaces a visible impulse is seen along the margin of the sternum. After dilatation the extent of impulse diminishes and the veins of the neck become engorged, the blood regurgitating into them during the systole.

Palpation. In the large majority of cases a distinct fremitus or thrill is felt—more marked in the fourth or fifth interspace, inside of the nipple. It is usually localized to a small area, is increased during expiration, and is of a twisting, grating, or grinding character. It is made up of a series of small shocks increasing in intensity, culminating in a sudden, sharp shock, which occurs at the time of the impulse. The thrill and systolic shock are pathognomonic, and may be present when other signs, as the murmur, are absent or indistinct. The cardiac impulse is felt strongest at the lower margin of the sternum and in the third and fourth interspaces, in some cases even in the second. It is due to an enlarged and dilated right ventricle.

The Pulse. With perfect compensation the pulse is slow, regular, and firm, although small. If the orifice is much narrowed, small, weak, and irregular in force and rhythm. When compensation fails and the right heart is dilated the pulse becomes rapid, quick, weak, small in size, and irregular in force and rhythm. The dilatation may be so great that the right auricle and overdistended veins may press upon the aorta or the innominate and subclavian arteries. The pulse on that side will be lessened in volume.[1]

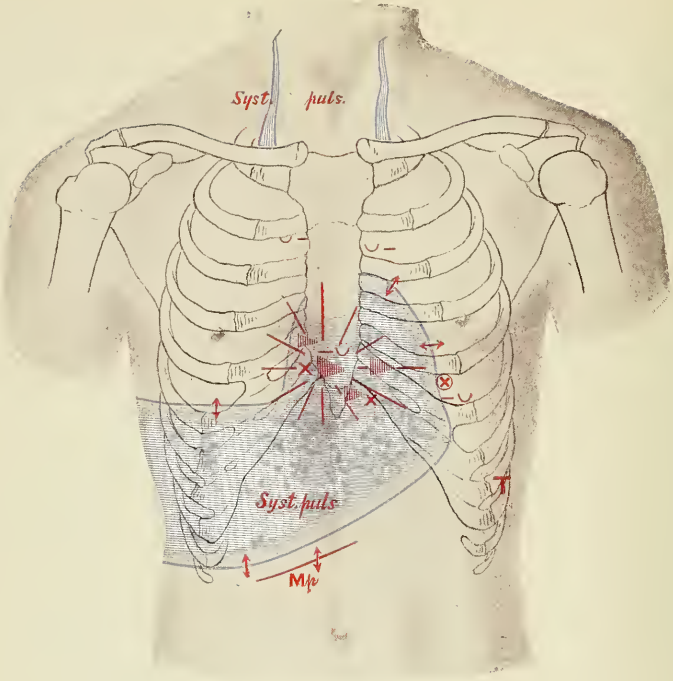
Percussion. The area of cardiac dulness is increased upward and to the right and left of the margin of the sternum. Sometimes it extends upward as high as the second rib ; this increase is quite characteristic.

Auscultation. At the apex, or just inside of the position of the apex-beat, a murmur is heard, its point of maximum intensity distinctly localized to this spot. It is usually not transmitted. (See Fig. 172.) It is of a churning and grinding character, or vibratory and purring. It is usually high in pitch and rough. It occurs synchronously with the thrill, and terminates with a loud shock that is heard simultaneously with the first sound. It is, therefore, presystolic in time. As has been said of the thrill, so it may be said of this murmur, that it is the only murmur that is pathognomonic of a special lesion. It indicates narrowing of the mitral orifice. The only exception, in which the lesion is absent, although the murmur is present, is found in the class of cases described by Flint, referred to in the section on

[1] Popoff: British Medical Journal, 1893.

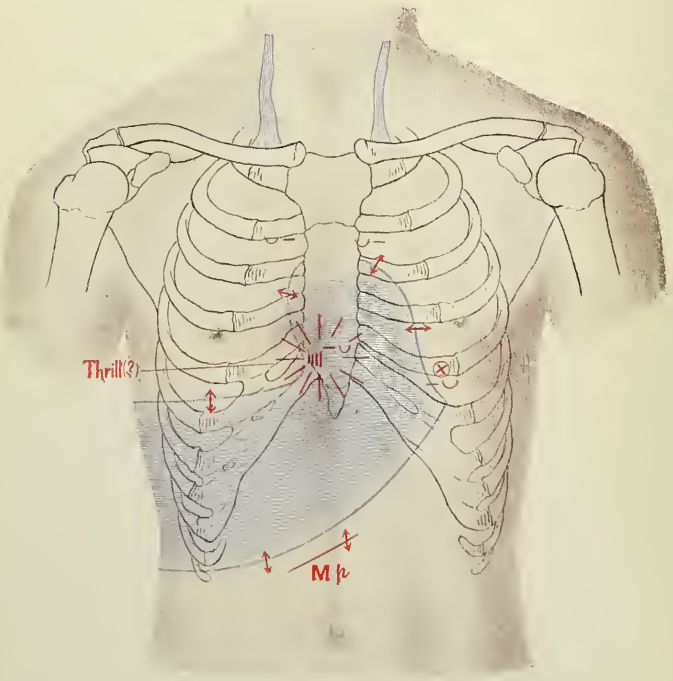
PLATE XXXI.

FIG. 1.



Tricuspid Regurgitation.

FIG. 2.



Tricuspid Stenosis.

aortic regurgitation. The first sound is loud, clear, and abrupt; it may be thumping.

The presystolic murmur may occupy the entire period of the diastole, but in the large majority of cases it occurs in the latter half only, during which the auricular systole occurs. In some instances it is heard in the middle of the diastole.

Associate Murmurs. 1. At the same time a systolic murmur may be heard at the apex, soft and low in pitch. It may be transmitted into the axilla. It is usually due to associate mitral regurgitation. 2. At the lower portion of the sternum a systolic murmur may be heard, due to dilatation and incompetency at the tricuspid orifice. Murmurs in the aortic region are not usually heard.

The *second sound* at the pulmonary orifice is usually *accentuated.* It is heard in the second and third interspaces along the left edge of the sternum; it may be heard at the apex. *Reduplication* of the first sound is often observed. Reduplication of the second sound is very common. After compensation is broken other murmurs may be heard, and the presystolic murmur changes in character. It may disappear entirely and be replaced by a sharp first sound. The short, high-pitched systolic shock may continue, although the murmur disappears. It disappears probably because the left auricle has become weakened. The tricuspid murmur continues during this period.

The points of distinction of mitral obstruction are (1) the position of the murmur; (2) its restricted area; (3) its peculiar character; (4) the systolic shock which takes the place of the first sound; (5) the thrill; (6) the impulse and increased area of dulness upward; (7) accentuated pulmonary second sound; (8) reduplication; (9) the absence of the pulse of aortic regurgitation and of hypertrophy of the left ventricle.

Presystolic Murmur not due to Valvulitis. A presystolic murmur without mitral obstruction may occur in aortic regurgitation and in adherent pericardium.

Tricuspid Regurgitation or Incompetency. Structural disease at the tricuspid orifice is of comparatively rare occurrence. Insufficiency is more frequent, and is due to dilatation, with relative insufficiency of the valve-orifice. It occurs secondarily to obstructive lung diseases, as emphysema and cirrhosis, and is secondary to regurgitation at the mitral orifice, which leads to stasis in the lungs.

Symptoms. The symptoms were detailed in speaking of the mitral valve affections. They are those of obstruction in the pulmonary circulation and engorgement of the systemic veins.

Physical Signs. (Plate XXXI., Fig. 1.) *Inspection.* The physical signs of dilatation of the right heart are seen. An impulse in the epigastrium is noted. This is seen especially between the xiphoid cartilage and the left margin of the ribs. Pulsation to the right of the sternum and in the second and third intercostal spaces may also be observed. The veins of the neck are also seen to pulsate. In addition to the wavy pulsation, regurgitation of the blood into the right auricle causes transmission of the pulse-wave into the veins. The pulsation is systolic in time. It is more marked in the right jugular than in the left, and in the external than in the internal veins. With the pulsation, regurgi-

tation is readily observed by emptying the external vein. Place the finger firmly on the vein just above the clavicle, move it along the course of the vein in the direction of the inferior maxillary bone. The vein is thus emptied of blood, and with each systole of the heart it will be seen to fill up from below in rhythmical pulsation. The veins are increased in size. This is more noticeable during the act of coughing or when the patient holds his breath in full inspiration. In rare instances the pulsation is transmitted to the subclavian and axillary veins.

Palpation. By palpation the above conditions are also determined. The impulse over the lower sternum and in the epigastrium is noted to be forcible.

The regurgitant pulsation is transmitted to the descending vena cava as well as to the ascending. The hepatic veins also distend during the systole. So-called pulsation of the liver is produced. With one hand on the fifth and sixth costal cartilages and the other over the liver in the axillary region, rhythmical expansile pulsation may be recognized. It is not of common occurrence, but is absolutely diagnostic of regurgitation at the tricuspid orifice.

Percussion. The area of cardiac dulness is increased transversely and upward, as described in mitral stenosis. It extends often far beyond the right edge of the sternum.

Auscultation. At the xiphoid cartilage, the lower end of the sternum or the head of the fourth rib, a murmur is heard. It is systolic in time, usually low in pitch, and is heard loud to the left of the sternum, within an inch of the apex, and to the right of the sternum and the outer limits of percussion-dulness. (See Fig. 173.) It is not further transmitted. Other murmurs are heard, due to the primary organic disease. If the heart is weak, the lesion may not be productive of a murmur. The pulmonary second sound is accentuated.

Tricuspid Stenosis. Stenosis at this valve-orifice is generally of congenital origin. In rare instances it may be secondary to lesions in the left heart. It is accompanied by dilatation of the right auricle.

The *physical signs* (Plate XXXI., Fig. 2) are the same as in stenosis at the mitral orifice, except for the alteration in their position. In some instances a presystolic thrill has been observed, and with it a presystolic murmur at the lower end of the sternum or toward the right of it. The area of dulness is increased as in right-sided dilatation. Cyanosis is a prominent symptom and may be intense.

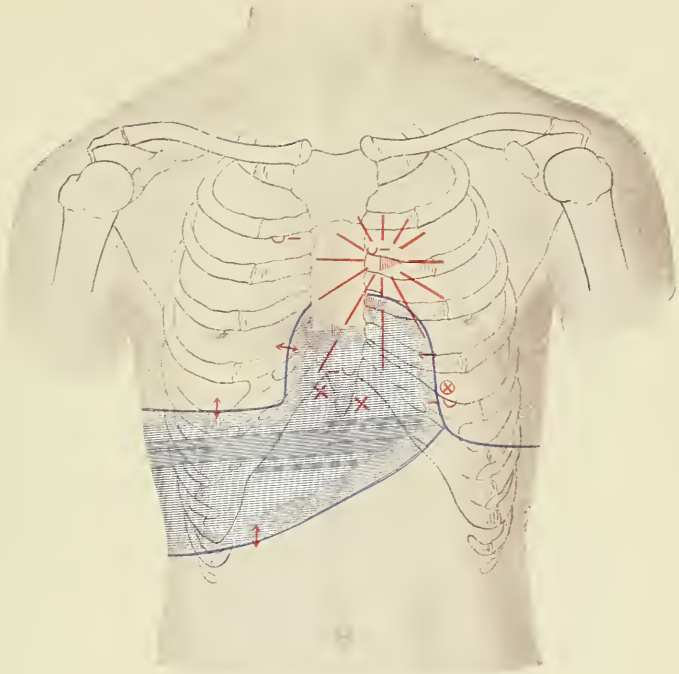
Disease of the Pulmonary Valve. Diseases of the pulmonary valve are extremely rare and are almost always congenital.

Pulmonary Insufficiency. (Plate XXXII., Fig. 1.) The *physical signs* are due to regurgitation into the right ventricle. The maximum intensity of the murmur is in the second pulmonary interspace, and it is transmitted down the sternum. It cannot be distinguished from aortic regurgitation, except by the pulse.

Pulmonary Stenosis. (Plate XXXII., Fig. 2.) In stenosis of the pulmonary valve a systolic murmur and thrill are detected to the left of the sternum in the second interspace. The murmur is not transmitted to the vessels of the neck. The pulmonary second sound is weak. The effect on the heart is the production of right-sided hypertrophy.

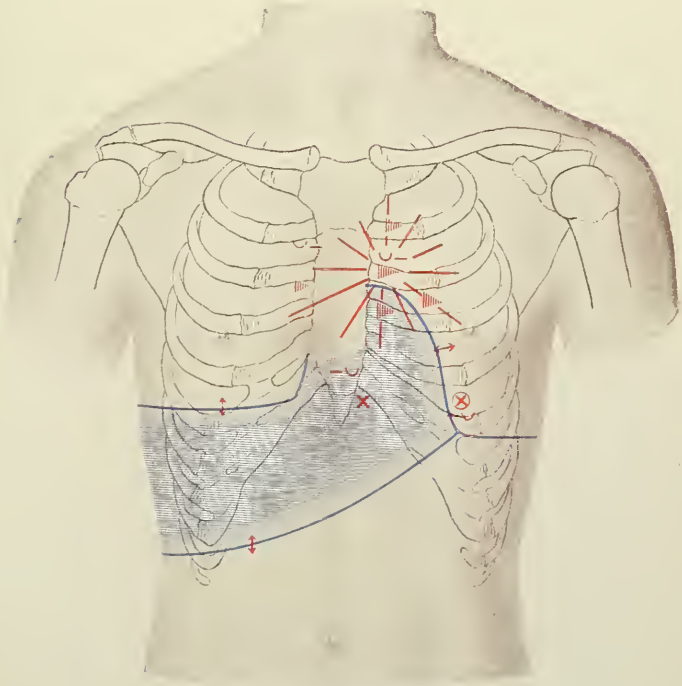
PLATE XXXII.

FIG. 1.



Pulmonary Insufficiency.

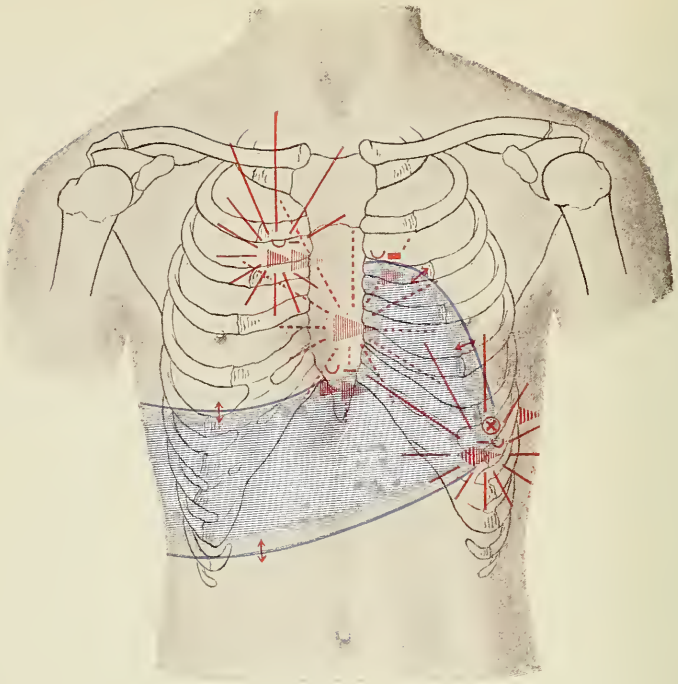
FIG. 2.



Pulmonary Stenosis.

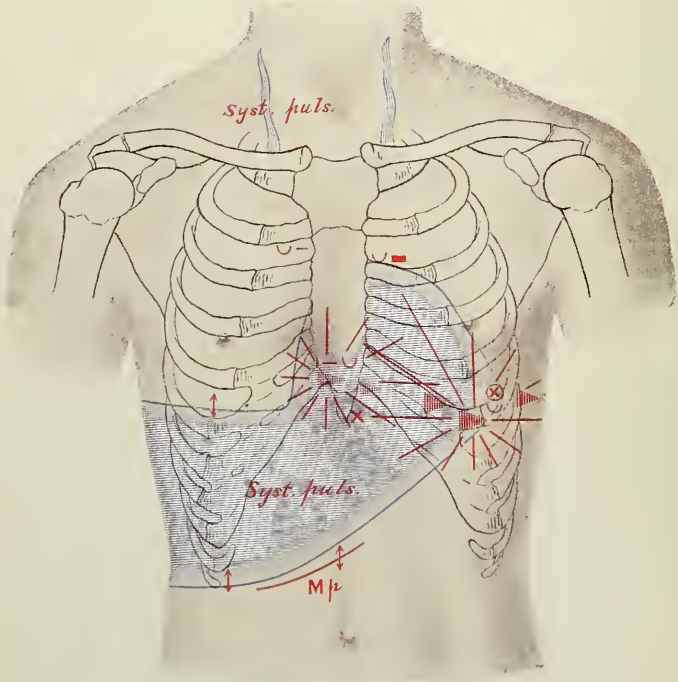
PLATE XXXIII.

FIG. 1.



Combined Mitral and Aortic Insufficiency and Stenosis.

FIG. 2.



Combined Mitral and Tricuspid Insufficiency.

Combined Valvular Lesions. (Plate XXXIII.) It must not be forgotten that there may be disease causing both obstruction and regurgitation at the same time and at the same orifice, or that two or more valves may be the seat of disease in the same individual. It is not impossible, for instance, to have aortic obstruction and regurgitation, mitral obstruction and regurgitation, and tricuspid regurgitation. Aortic obstruction or insufficiency is frequently combined with mitral insufficiency. Aortic and mitral insufficiency occur together most frequently in children ; aortic obstruction and mitral obstruction in adults.

When more than one valve is diseased the site of the various lesions is based upon the time, the position of maximum intensity, and the direction of transmission of the murmurs. Students often experience difficulty here. A systolic murmur may be heard in the aortic area and in the mitral area at the same time. If it is observed that each progressively weakens as the stethoscope is moved toward the middle of the præcordial area, it may be inferred that the murmur, systolic in time, is due to two lesions. As previously intimated, the direction of the transmission of the murmur further aids in the diagnosis.

Enlargement of the Heart.

Enlargement of the heart is due to *hypertrophy* or to *dilatation*. In hypertrophy there is increased thickness of the muscular walls. This may be general or limited to the walls of one chamber. Hypertrophy is further divided into simple hypertrophy, in which the cavity or cavities are of normal size, and eccentric hypertrophy, in which, with increase in the wall, there is enlargement of the cavities. This is hypertrophy with dilatation. The left ventricle is most frequently the seat of hypertrophy when one chamber is involved. The cause of hypertrophy is obstruction to the flow of blood ; increased work is followed by increased size of the muscle. *General hypertrophy* or *hypertrophy of the left ventricle* occurs from diseases of the heart itself, or from affections of the bloodvessels.

A. *Diseases of the heart.* 1. Disease of the aortic valves. Hypertrophy of the left ventricle always follows. 2. Mitral regurgitation. 3. Pericardial adhesions. 4. Myocarditis of the fibrous variety. 5. Neuroses with overaction and frequent palpitation, as in exophthalmic goitre and from the effects of tea, tobacco, and alcohol. In pericardial adhesions and myocarditis hypertrophy arises because of the inability of the heart to do the work expected of it. There is no obstruction in the course of the vessels or at the orifices. The struggle to keep up causes the hypertrophy. In neuroses there is absence of obstruction, but the rapid action causes hypertrophy.

B. *Affections of the bloodvessels* which cause hypertrophy are : 1. General arterial sclerosis. 2. Increased arterial tension due to contraction of the peripheral arteries, as in Bright's disease, and in toxæmias from lead, the poison of gout and of syphilis. 3. Increased blood-pressure from prolonged muscular exertion. 4. Narrowing of the aorta from external pressure and from congenital stenosis or the development of an aneurism.

Hypertrophy of the Right Ventricle. Obstruction to the flow of blood in the pulmonary area is the usual cause of hypertrophy of the right ventricle. This obstruction occurs in lesions of the mitral valve, causing pulmonary stasis; and disease of the lungs, causing compression of the bloodvessels, as in emphysema or cirrhosis. It occurs if there is disease of the right heart with obstruction of the valves. Thus in obstruction at the pulmonary orifice the right ventricle undergoes secondary hypertrophy.

Hypertrophy of the Auricles. Simple hypertrophy of the left auricle with dilatation develops in mitral stenosis. Hypertrophy of the right auricle occurs in tricuspid obstruction and in right-sided dilatation with tricuspid regurgitation.

Symptoms. The symptoms of hypertrophy of the heart are *general* and *local*. The former are not common. They are due to increased tension in the cerebral vessels because of increased force of the heart, usually causing congestive headaches, noises in the ears, flashes of light, and flushing of the face.

General symptoms arise in hypertrophy of the left ventricle because the increased force causes reactive spasm of peripheral vessels, and hence increased tension in the vascular system. In Bright's disease, for instance, or heightened arterial tension from other causes, endarteritis develops in the large vessels, on account of the strain put upon them. This is seen particularly in the aorta and its divisions. Whether atheroma is primary or secondary, its presence, with hypertrophy of the left ventricle, indicates that rupture of the vessels somewhere in the periphery may take place. This occurs most frequently in the brain, causing apoplexy.

Locally, the patient complains of fulness and discomfort, particularly marked when lying down on the left side. In the hypertrophy that accompanies the tobacco-heart, or the irritable heart of soldiers, there may be some pain. On the other hand, the organ may be enormously enlarged without the patient complaining of discomfort about the heart. Palpitation is not of common occurrence except in neurasthenic subjects.

Physical Signs. The hypertrophy causes præcordial bulging, if it has developed early in life, when the ribs are soft. The intercostal spaces are widened and the area of impulse is much increased. The normal impulse is changed in position. It is downward and to the left, often extending as far as the axilla in hypertrophy of the left ventricle.

Palpation. The impulse is forcible and heaving. The head is visibly raised with each systole when placed upon the chest for auscultation. The impulse is slow. This slow, heaving impulse distinguishes it from the forcible impulse of dilated hypertrophy, which is sudden and abrupt. Inspection is confirmed as to the position of the apex. In moderate hypertrophy the apex extends to the sixth interspace in the mid-clavicular line. In large-sized hypertrophy it may extend to the seventh interspace. The heart may be apparently hypertrophied in fibrous and fatty myocarditis. The impulse may be absent in emphysema, in fatty overgrowth of the heart, and in persons with thick chest-walls.

The Pulse. The frequency of the pulse is not affected. It is full, regular, and strong. The tension is increased. In dilated hypertrophy the pulse is full but soft, and more rapid than in simple hypertrophy. When failure of the heart takes place the pulse increases in frequency and becomes intermittent and irregular. When valve-lesions are present the pulse is modified accordingly.

Percussion. The area of dulness is increased both upward and transversely. It may begin as high as the second interspace and extend two inches beyond the left mid-clavicular line, and an inch beyond the right edge of the sternum transversely. In simple hypertrophy the area is ovoid.

Auscultation. When the valves are healthy, prolongations of the first sounds occur. They are also at times duller than in health. The dull, prolonged first sounds distinguish hypertrophy from dilatation, for in the latter they are clear and sharp. The second sounds are clear and loud. The degree of accentuation depends upon the state of the peripheral arteries. If there is heightened tension, the second sound may be reduplicated. If valvular disease is present, the sounds are modified.

Hypertrophy of the Right Ventricle. Increased pulmonary tension from resistance in the pulmonary circulation may always be looked for. If there is complete compensation, no symptoms are observed, or only those of dyspnœa on extra exertion. Hypertrophy of this ventricle persists for a long period of time without the grave local changes in the heart, or secondary changes in the peripheral vessels, which occur in left ventricle hypertrophy. In dilated hypertrophy, when the dilatation is in excess, tricuspid regurgitation takes place, with the development of venous stases. Induration of the lungs may succeed the persistent engorgement of the capillaries. Pulmonary congestions and apoplexy may also occur.

Physical Signs. The physical signs of hypertrophy of the right ventricle have been partially referred to under the various valve affections. There is bulging of the lower part of the sternum and cartilages. The epigastric impulse in the angle between the ensiform cartilage and the ribs has been referred to. The impulse may be in the sixth interspace. It is diffuse; it may extend upward as in mitral stenosis. Cardiac dulness is increased toward the right an inch or more beyond the border of the sternum. The heart-sounds are not much changed unless there is dilatation. The tricuspid sound is clear and sharp when this occurs. The pulmonary second sound is accentuated, and reduplication may take place. The radial pulse is small. If there is tricuspid regurgitation, the physical signs that attend it are present.

Hypertrophy of the Left Auricle. This is present in mitral stenosis, but cannot be determined by physical signs, save possibly by greater increase of dulness to the left of the sternum in the second and third interspaces. Barr states that dulness above the "suprasternal mammillary line" toward the left clavicle indicates enlargement of the left auricle, as in mitral stenosis. The line above mentioned is drawn from the middle of the suprasternal notch to the normal site of the left nipple on the fourth rib.

Hypertrophy of the right auricle with dilatation occurs under the same circumstances as hypertrophy of the ventricle. It usually dilates more than the left auricle in left ventricle hypertrophy. There is increased area of dulness in the third and fourth right interspaces; abnormal pulsation is sometimes observed in this situation before the systole, with the signs of tricuspid regurgitation.

Diagnosis. The forcible impulse in nervous palpitation of the heart must not be confounded with true hypertrophy, although it must not be forgotten that hypertrophy frequently follows neurotic palpitation, as in the smoker's heart, or in exophthalmic goitre. The enlargement must not be confounded with enlargement of the area of cardiac dulness in the præcordial region from other causes, such as pericardial effusion; aneurism and mediastinal tumor, pushing the heart against the chest-wall; disease of the lungs, on account of which they are withdrawn from the surface of the heart, as in phthisis or chronic pleurisy; and displacement of the heart from pressure, as in effusion on the left side of the chest, or in disease below the diaphragm. The cause of hypertrophy should be ascertained, for it is a valuable aid in diagnosis. It must not be forgotten that emphysema of the lung may mask a considerable hypertrophy of the heart by causing diminution of the area of dulness.

Dilatation of the Heart. Enlargement due to dilatation of the heart is common. The condition usually succeeds hypertrophy. Thickening of the muscles attends dilatation of the cavities, as in dilated or eccentric hypertrophy. The dilatation occurs because of increased pressure within the cavities or because of weakening of the heart-walls, the pressure within being normal.

1. Increased pressure within the walls is due to an increased amount of blood within the chamber from regurgitation, or from an obstacle to the outward flow of blood. Simple hypertrophy occurs first in many cases; in others, hypertrophy with dilatation; in not a few, dilatation takes place at once. In dilatation the chamber does not empty itself during the systole. It is seen physiologically after the exertion of ascending a great height. It may remain within the bounds of physiological action. Temporarily, as any one can show by running violently, the dilatation is attended by increased epigastric pulsation and increased cardiac dulness. The tricuspid valves temporarily become incompetent, owing to their safety-valve action. The latter may continue after the acute strain, the heart always showing symptoms of the condition, or it may disappear entirely. An excessive dilatation results in heart-strain, with cardiac distress and dyspnœa, symptoms due to overdistention and paralysis of the heart. (See Symptoms.) Dilatation occurs in all forms of heart-lesions previously described. The most typical is seen in aortic regurgitation, when the left ventricle becomes the seat of dilatation, and in mitral regurgitation when the left auricle becomes the seat of dilatation.

2. Disease of the heart-walls, lessening the resisting power, the normal pressure within the cavities being maintained, invites dilatation. In myocarditis, in infections, acute dilatation may ensue. It occurs in scarlatinal dropsy, typhoid fever, rheumatic fever, and erysipelas.

The heart-muscle changes in acute endocarditis and pericarditis, on account of which dilatation may ensue. In anæmia and chlorosis the same process may take place. In chronic myocarditis dilatation takes place at the apex. When pericardial adhesions are present the fibrous overgrowth invades the interstices of the myocardium, thereby weakening the heart-muscle. Dilatation may follow.

Symptoms. The symptoms of dilatation are the reverse of those of hypertrophy. When the latter fails the blood is not expelled from the chambers in systole, so that the cavity is overdistended with blood that accumulates in the diastole. Weakening of the muscles also favors the development of dilatation. As soon as dilatation becomes permanent, incompetency of the valves takes place. In obstructive heart disease the left side is first affected. It may be compensated for by hypertrophy of the right side. When this fails venous engorgement and dropsy ensue. The symptoms have been described under chronic valvular disease. In *acute dilatation* there is a sudden occurrence of dyspnœa. Pain, or at least præcordial oppression, may be complained of. The heart's action increases in frequency. The pulse is rapid, feeble, irregular, and may scarcely be felt at the wrist.

Physical Signs. *Inspection.* The apex is displaced to the left, even as far as the axillary line, but rarely downward, unless hypertrophy precedes the dilatation. The impulse is diffused and undulatory in appearance. The apex-beat may be defined with extreme difficulty. It may be visible when the patient leans forward, yet not felt.

With the diffused area of impulse a quick apex-beat may be felt—much weakened, however. When the right ventricle is dilated, the impulse is seen and felt to the right or left of the xiphoid cartilage, and there is a wavy pulsation along the left edge of the sternum in the fourth, fifth, and sixth interspaces. If the dilatation is extreme, involving the right auricle, a pulsation at the third right interspace close to the sternum may be felt. Tricuspid regurgitation is then present.

The area of dulness is increased in the same directions as in hypertrophy, if the two coexist. In general, it may be said the increase extends outward to the right or left, the direction corresponding to the ventricle affected. It is increased upward along the left edge of the sternum in left auricle dilatation. (See Mitral Valvulitis.) When the whole heart is dilated the increase of dulness is in a transverse direction on both sides. The apex is rounded or square, not pointed, as in hypertrophy; indeed, it retains the oval shape of the dulness of a normal heart. As dilatation occurs so frequently in emphysema of the lungs, the modification of the percussion-sound must be remembered.

Auscultation. The systolic sounds are short and sharp. They are high-pitched and resemble the diastolic. The latter may become enfeebled when the dilatation becomes excessive. The right and left first sounds may differ somewhat in intensity, and reduplication may occur. The sounds may be obscured by murmurs. The murmurs are due to previous valve disease or to incompetency, on account of dilatation. The action of the heart is irregular and intermittent. The

pulse is correspondingly small. In dilatation the alteration of the rhythm is extreme. There may be *embryocardia* or fœtal-heart rhythm, in which the first and second sounds are alike, and the long pause is shortened. More frequently we have galloping rhythm of the heart. It must not be forgotten that, as dilatation ensues, murmurs of various valve-lesions may disappear, particularly the murmur of mitral stenosis. On the other hand, in the earlier stages particularly, murmurs develop, on account of incompetency at the auriculo-ventricular orifices, in addition to the primary organic murmur. These murmurs in turn may disappear, if the dilatation is controlled by careful treatment.

Diseases of the Arteries.

Arterial Sclerosis or Arterio-capillary Fibrosis. This occurs as the result of wear and tear of life and as the accompaniment of age. The time of its onset depends upon the quality of the arterial tissue which the individual inherited, and upon the amount of wear and tear. It may occur early in life, and entire families may show this tendency. Very frequently the sclerosis develops from intoxications of the system, on account of which persistent spasm of the small vessels is set up—for blood of an impaired quality is passed with greater difficulty through the capillaries, as was taught by Bright. The blood-tension is raised thereby. The poison of alcohol, of lead, of gout, and of syphilis leads to this condition. The poison of syphilis and of gout may set up directly an inflammation and degeneration of the arteries. In renal disease arterial sclerosis is of common occurrence. The relation to the renal lesion differs. It may be primary or secondary. When primary, the morbid cause operates upon the kidneys as well as the arteries. When secondary a morbid poison is retained within the system by the diseased kidneys, the action of which is such as to cause peripheral spasm and heightened tension.

Overfilling of the bloodvessels from excessive eating and drinking is thought by some to cause arterial sclerosis through constant overdistention of the vessels. In overwork of the vessels and excessive strain there is either heightened tension or increased peripheral resistance, the effect upon the bloodvessels being the same in either case. The result of the above causes is thickening of the intima, followed by changes in the media and adventitia, terminating in endarteritis deformans of the large arteries.

Symptoms. The symptoms vary. They may be general or local. The disease may be present and the patients die from other causes. Local symptoms are due to rupture of the vessels, as in apoplexy from cerebral hemorrhage, or to their obstruction, as the coronary artery, or to rupture of an aneurism.

Physical Signs. Arterio-sclerosis is recognized by inspection, palpation, and auscultation of the bloodvessels, and by observation of the condition of the heart. The superficial bloodvessels are elongated and tortuous, and pulsate visibly. On palpation the artery feels very hard to the touch ; it resists compression ; it is corded or rounded underneath the finger, and readily rolled about. The pulse shows at

once high tension ; the wave is slow in ascent, continues long underneath the finger, and subsides slowly. If in the interval of the beats the vessel remains full, the pulse, as previously noted, is obliterated with difficulty. Sphygmographic tracings are characteristic. (See Pulse.) If, after pressure on the radial artery, it can still be felt beyond the point of compression, its walls are sclerosed ; whereas, if after such compression the artery is obliterated beyond the point of compression, the hardness and firmness of the pulse previously observed are due to vascular tension and not to thickened walls. The two conditions should be distinguished. Hypertrophy of the heart occurs early in the course of the sclerosis, on account of peripheral resistance. The hypertrophy involves the left ventricle, and is not attended by dilatation. The apex-beat is out beyond the mid-clavicular line ; the impulse is heaving and forcible. The second sound at the aortic cartilage is characteristic. It is clear and ringing ; it is heard in the course of the bloodvessels, and is most distinct at or just beyond the apex. Right-sided hypertrophy and dilatation are not generally present. Auscultation of the larger arteries, as the carotids, the abdominal aorta, and femorals, shows a systolic murmur usually rough and high in pitch. All the above-mentioned conditions may be present, and yet the patient remain in good health. The hypertrophy apparently compensates for the arterial occlusion. There may be no renal disease, or moderate renal cirrhosis may be present, indicated by transient albuminuria, polyuria, and hyaline tube-casts. The subsequent symptoms are due largely to closure of one or more vessels in the peripheral circulation, to the development of an aneurism or dilatation of the aorta, to failing hypertrophy of the heart, or to the development of renal cirrhosis.

The blocking of peripheral arteries is due to embolism or thrombosis, more frequently the latter, and to rupture of peripheral vessels, or, in all probability, miliary aneurisms. When occlusion of the vessels takes place in arteries which supply the extremities gangrene may occur. Sometimes the occlusion is due to simple narrowing of the vessels alone. Gangrene of the feet is frequently seen secondary to bad arteries. If the occlusion takes place in the vessels of the brain, various secondary lesions are produced. In more or less general occlusion from sclerosis of the smaller arteries acute and chronic softening occur. Hemiplegia, monoplegia, or aphasia may occur temporarily, if relieved by collateral circulation, or permanently, from embolism, thrombosis, or rupture of the vessels. Hence, apoplexy is almost always due to primary disease of the arteries, upon which, in the large majority of cases, miliary aneurisms have existed. If the coronary arteries are blocked, thrombosis with sudden death takes place, or chronic myocarditis may develop, with subsequent aneurism and rupture. Angina pectoris, with or without thrombosis of the coronary artery, is always associated with arterial sclerosis.

Failure of the hypertrophied heart leads to dilatation with all the symptoms as previously described, including cyanosis, visceral congestions, and dropsies. The murmur at the apex, due to incompetency from dilatation, may simulate chronic valvular disease, although the

latter may never have been present. The sclerosis may advance more rapidly in the kidneys than in the other portions of the circulation; later, on account of the contracted kidney, symptoms of interstitial nephritis may arise.

Aneurism.

A true aneurism is formed by the distention of one or more of the arterial coats. It is usually fusiform, but may be cylindrical. It may be circumscribed or sacculated. The fusiform and saccular are the forms most commonly seen. False aneurism or dissecting-aneurism arises from laceration of the internal coat of the artery. The blood dissects between the layers. It occurs in the aorta. It may begin at the heart and separate the coats as far down as the iliac arteries. *Arterio-venous aneurism* is seen when communication between an artery and a vein has been set up. If a sac intervenes, it is called a *varicose aneurism*. Sometimes communication is direct, the vein becoming dilated, tortuous, and pulsating. It is known as an *aneurismal varix*.

An aneurism may occur in the course of arterial sclerosis from diffuse distention of the coats. Its typical form is seen in dilatation of the aorta with one or more sacculated aneurisms on its surface.

Sacculated aneurism occurs from rupture of the tunica media, independently of general disease of the arteries, and in arterial sclerosis. The most common seat is the ascending portion of the aorta. It occurs early in the course of arterial sclerosis. Such form of aneurism is seen in the smaller vessels. Aneurisms also arise after the lodgement of an embolus, permanently plugging the vessel. The proximal end of the vessel becomes dilated.

Mycotic aneurism, first described by Osler and exhaustively by Eppinger, occurs in malignant endocarditis. The aneurisms are small in size and multiple, and not recognized during life. They arise from the injury produced by the local infection of bacteria in different portions of the vascular system.

Aneurism of the Thoracic Aorta. The causes which produce arterial sclerosis are operative in the thoracic portion of the aorta—chiefly physical overwork, alcohol, syphilis, and gout. It may be situated just beyond the aortic ring, at the junction of the ascending and transverse aorta, in the transverse, or at the beginning of the descending, portion of the thoracic aorta. The larger aneurisms are at the two bends of the aorta.

Symptoms. The symptoms of aneurism are largely due to pressure, and depend upon the position of the aneurism and the direction of its growth.

Aneurisms, however, may exist *without symptoms* or appreciable physical signs. Even in a patient who has been under careful observation, sudden death may take place from rupture of a concealed aneurism, the presence of which had not been suspected during life. On the other hand, cases occur with characteristic pressure-symptoms and with no physical signs. Pressure-symptoms depend entirely upon the position of the tumor.

Aneurisms of the *ascending* portion of the arch cause dislocation of

the heart outward, or toward the right pleura or forward, appearing at the second or third interspace, causing erosion of the ribs and sternum. The vena cava is compressed, causing enlargement of the veins of the head and arms; the subclavian vein may be compressed alone, causing enlargement and œdema of the right arm. Localized œdema may result, confined to the thorax. (See Œdema.) If the aneurism is large, the inferior vena cava may be pressed upon, causing œdema of the feet. The right laryngeal nerve may be involved, causing aphonia and dyspnœa. Pain attends the aneurismal process.

FIG. 177.

Aneurism of ascending portion of arch of aorta. Tumor in first and second interspaces, extending into neck. Portion of sternum atrophied.

Aneurisms of the *transverse* portion of the aorta project below, forward, or backward. When forward, they produce tumors behind the manubrium, which from pressure cause destruction of the bone; if the aneurism projects backward, marked pressure-symptoms are produced. When the trachea is pressed upon, it causes dyspnœa and cough, which

is paroxysmal. (See Dyspnœa.) The œsophagus may be pressed upon, causing dysphagia. The left recurrent laryngeal nerve may be pressed upon, causing paralysis of the corresponding cord, with aphonia. (See Larynx.) Pressure on a bronchus may produce bronchorrhœa and dilatation, which in turn may lead to localized abscess. The growth may extend upward, involving the coats of the innominate and carotid arteries on the right side, or carotid and subclavian on the left, markedly interfering with the pulse of the two sides. Pressure on the sympathetic nerve is likely to take place in this situation, with contraction of one of the pupils, although at first it is sometimes dilated. The thoracic duct is sometimes compressed, leading to rapid wasting.

In the *descending portion* the pressure-signs of aneurism are often not so marked. The vertebræ are likely to be pressed upon in this situation. The pain, therefore, is most intense. The œsophagus and left bronchus are compressed. Dysphagia and bronchiectasis, the latter causing bronchorrhœa with subsequent gangrene, are likely to occur. The *cough* and the fever in bronchorrhœa, together with emaciation, simulate phthisis, for which aneurism is often mistaken. The physical signs of phthisis are usually pronounced in this situation, and, with the presence of bacilli in the sputum, render the diagnosis easy. In these cases rupture takes place into the bronchus or into the œsophagus. In one of my cases, which had been treated for tuberculosis because of small hemorrhages, with the conditions above-mentioned, death took place from rupture into the bronchus, causing sudden profuse hemorrhage. When the aneurism is adherent to the œsophagus and slowly ulcerating into it, rupture may take place, followed by instantaneous death. The vertebræ may be eroded and symptoms of spinal compression arise.

I once saw an autopsy performed by a medico-legal expert on a case of sudden death from gastric hemorrhage. The source of the hemorrhage could not be ascertained. There was blood in the stomach. When he was about to give up the search, the œsophagus and aorta were suggested for examination. A small aneurism was found which had ulcerated and then ruptured into the gullet. In another the aneurism had ruptured into the pleural sac, causing internal concealed hemorrhage and death.

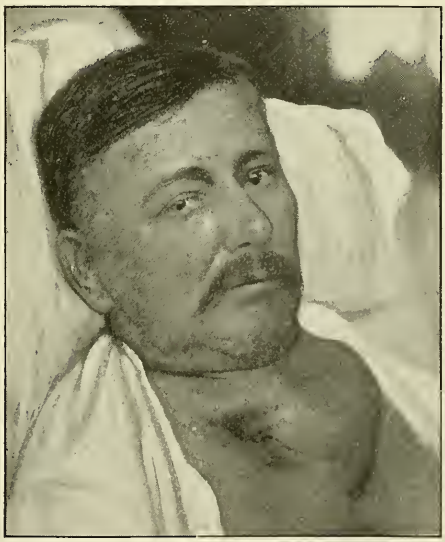
Special Symptoms. While pressure-symptoms are the most striking symptoms of this affection, *pain*, which is usually due to pressure, must be referred to. It is an important constant symptom. It is sharp and lancinating, and may occur in paroxysms. It is more severe and constant when bone is eroded by pressure on the vertebræ, or the thorax in front. The gnawing pain that attends ulceration of bone is relieved, if it, as the sternum, is perforated. Anginal attacks may attend the neuralgic pains just described. Pain sometimes follows the course of the nerves, extending down the arm or to the neck, or along the course of the intercostal nerves.

Cough. The cough is peculiar. It is paroxysmal in many cases and of a brazen, ringing character, indicating its laryngeal origin, due to pressure upon the recurrent laryngeal nerves. It is frequently

paroxysmal when the pressure is directed upon the windpipe or bronchus. In the former instance the cough is dry, in the latter tracheal and bronchial. It is attended by a thin, watery expectoration which, if bronchiectasis with fermentation ensues, becomes thick and ropy. *Dyspnœa* occurs more frequently in aneurism of the transverse portion, due (1) to pressure on the recurrent laryngeal nerves ; (2) to compression of the trachea ; (3) to compression of the left bronchus. Marked stridor attends the first form. When one of the recurrent laryngeal nerves, more particularly the left, is pressed upon, there is spasm or paralysis of the muscles of the vocal cord, causing hoarseness and loss of voice. Laryngoscopic examination should not be neglected, for paralysis of the abductor muscles without symptoms may be present.

Hemorrhage. The hemorrhage may be gradual when there is slight leakage into the trachea at the point of compression. The amount of blood lost is small. It may take place externally. (See Fig. 178.) Profuse hemorrhages, causing sudden death, occur from

FIG. 178.



Aneurism of ascending and transverse portions of aorta projecting forward, destroying ribs and sternum. The skin ulcerated, and gradual external leakage took place. The bleeding continued in small amounts for a long time.

rupture into the trachea or bronchus, and from perforation into the lung. With regard to difficulty of deglutition, it may be said that the sound should never be passed in suspected cases of aneurism, on account of the danger of rupturing the sac.

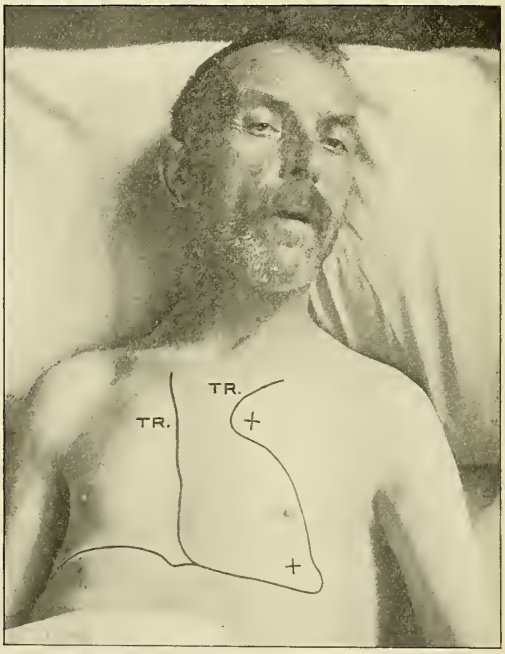
Clubbed Fingers. In intrathoracic aneurism clubbing of the fingers and incurvation of the nails of one hand are sometimes seen, although comparatively rarely.

Compression and pressure on the *sympathetic system of nerves* has been referred to. In addition to pupillary changes there may be pallor

of one side of the face. When the pupil is dilated this pallor may accompany it, on account of stimulation of the vaso-dilator fibres. When the cilio-spinal branches of the sympathetic are pressed upon, the dilator fibres are paralyzed. If the pupil contracts, there are also hyperæmia of the side of the face and unilateral sweating.

Physical Signs. (Plate XXXIV., Fig. 1.) *Inspection.* In health the position of the aorta cannot be recognized. Pulsation may be seen at the episternal notch in rare instances, particularly in women, independently of disease of the aorta; it is due to nervous palpitation. An aneurism may exist without any external visible signs. On the

Fig. 179.



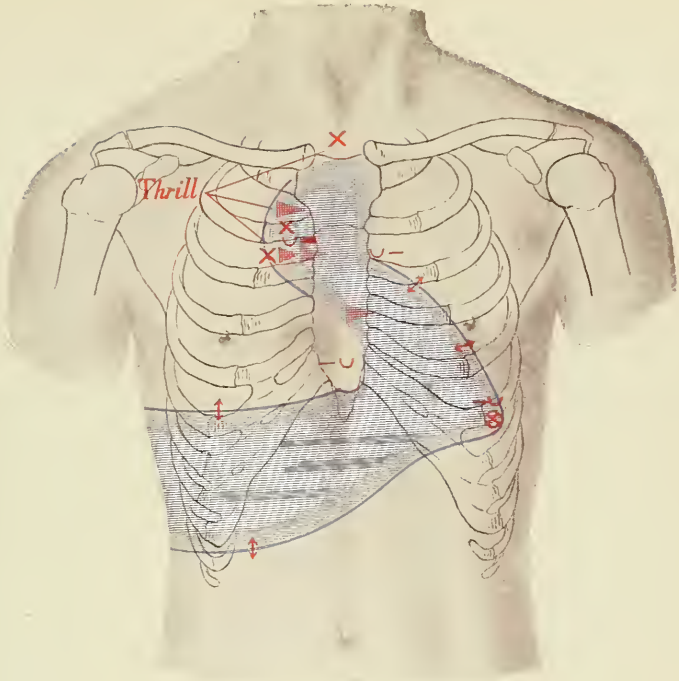
Aneurism. General endarteritis and valvulitis.
TR. = Thrill and impulse. + = Murmur.

other hand, pulsation may be seen at either side of the sternum above the level of the third rib, most commonly in the second interspace on the right side. The impulse may be seen alone without visible swelling; the chest must be viewed from different situations in order to detect it. An oblique light falling on the surface is sometimes necessary. When the innominate artery is involved the pulsation is observed in the neck, above the sterno-clavicular junction, or above the sternum.

With the abnormal impulse a swelling or *tumor* is often present. It may be large enough to press the upper portion of the sternum and adjacent ribs forward. In other instances a tumor the size of the half

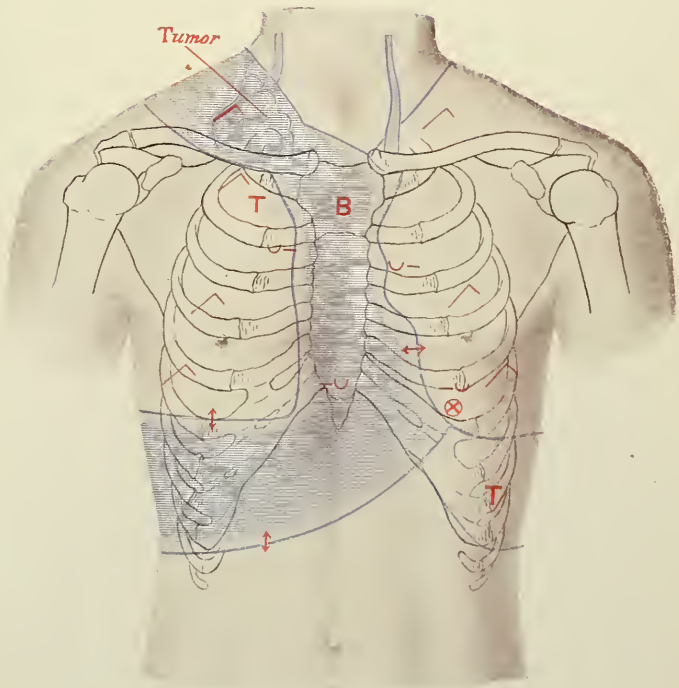
PLATE XXXIV.

FIG. 1.



Aneurism of the Arch of the Aorta.

FIG. 2.



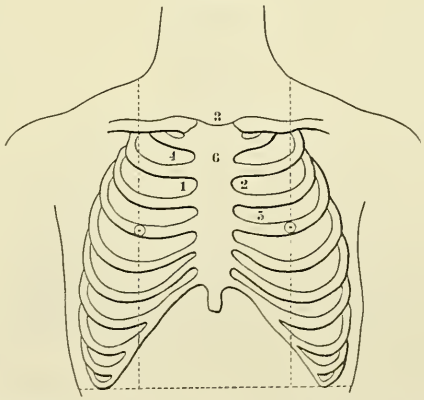
Tumor of the Anterior Mediastinum.

of a lemon may be seen along the edge of the sternum. The most frequent site is the first and second right, or the second left interspace. The skin over the tumor, as in the case of which an illustration is given, may ulcerate and be the seat of persistent small hemorrhages. The *apex-beat* of the heart is displaced downward and outward from pressure.

If the aneurism is seated in the ascending portion of the aorta, just beyond the aortic ring, a pulsating tumor may be seen in the third interspace at the left edge of the sternum. If in the ascending portion, beyond the heart, the tumor is in the first or second interspace along the right edge of the sternum. If the aneurism is in the transverse portion of the aorta, the upper portion of the sternum is frequently made to protrude, or the tumor projects upward into the fossæ of the neck. If in the descending portion, it is in the second or third interspace on the left side. In this portion of the aorta a tumor is seen in the left scapular region in rare instances.

Palpation. Palpation must be employed by the usual method; bimanual palpation must also be used, one hand placed upon the sternum and the other upon the vertebræ. Moderate pressure should be exerted. Palpation should also be employed at different periods of respiration. At times signs are only yielded at the end of complete expiration. It must further be said that palpation must be employed both with the tips of the fingers and with the palm of the hand applied to the surface.

FIG. 180.



Possible position of impulse in aneurism; arranged in order of frequency.

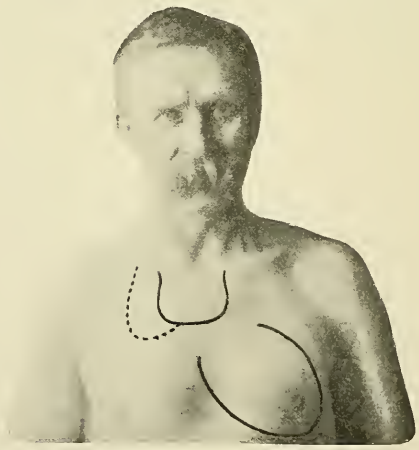
By *palpation* the area and degree of pulsation are determined. If the aneurism is large or has perforated, the impulse is expansile and heaving in character. The sac may be soft and fluctuating, but usually presents considerable resistance. In addition to the systolic impulse the diastolic shock is also felt. This is a most conclusive physical sign. A thrill is frequently present, systolic in time, usually due to dilatation of the arch; at times, to sacculated aneurism. Without visible tumor, pulsation and thrill may be felt in the suprasternal notch, if the head is bent forward, so that the tissues are relaxed, and

the fingers pushed down toward the aorta. When the aneurism is filled or filling with clot, the tumor may be seen and felt, but no impulse will be transmitted to the hand or thrill be felt by the fingers.

Percussion. Percussion furnishes the most reliable evidence of the presence of an aneurism or aneurismal dilatation in cases in which the tumor is not too deep-seated or small in size. The dulness may be relative only. (See Cardiac Percussion.) The area of dulness is increased somewhere in the course of the aorta. It may be observed projecting outward at the right edge of the sternum when the ascending portion of the aorta is the seat of disease, or over the entire upper part of the sternum, extending toward the left, when the transverse portion is diseased. It may be observed as an extension of cardiac dulness upward in the second and third interspaces. Sometimes dulness is detected in the scapular regions, particularly of the left side. The percussion-tone is flat, and there is marked sense of resistance. Percussion must be employed with the patient in the upright and in the recumbent posture.

Respiratory Percussion. The character of the tone and the shape of the dulness must be noted at the end of full inspiration and of full expiration.

FIG. 181.



Aneurism of aorta.
Area of absolute dulness, dark line. Area of relative dulness, broken line.

Auscultatory percussion is of the utmost value, and the method of percussion taught by Sansom and Ewart must be carefully followed. An aneurismal tumor may be present without thrill or murmur, but yields signs of dulness on percussion.

Auscultation. As just stated, murmurs may not always be present. They depend upon the amount of fibrin in the sac. When present the murmur is systolic in time, heard with maximum intensity usually over the abnormal area of impulse or tumor, or over the increasing area of dulness. It is transmitted in the direction of the

vessels, and may be heard louder in the vessels of the neck and along the course of the aorta. Often a double murmur is heard, the diastolic sound being due to associated regurgitation at the aortic orifice. Sometimes the diastolic murmur alone may be heard. Increase in intensity or accentuation of the aortic second sound is pronounced. The sound is ringing in character, and is rarely absent in large aneurisms.

The Peripheral Vessels in Aneurism. The pulse in the two radial arteries may show a marked difference both in volume and in time. The difference may indicate the position of the aneurism. If the pulse of the right radial is smaller than the left, the aneurism may be in or near the innominate artery ; if the opposite, it is near or includes the orifice of the left subclavian. In the same way the difference in time may also aid in determining the location. Osler refers to obliteration of the pulse in the abdominal aorta and its branches. In one case he could not feel throbbing in the aorta and the femorals, although the circulation was unimpaired. The aneurism was in the descending portion of the aorta, and its pulsation was seen in the left scapular region. The sac was sufficiently large to act as a reservoir, which filled during the ventricular systole, and from which the blood poured toward the periphery in a continuous stream instead of being intermittent.

Tracheal Tugging. Tracheal tugging may be obtained in one of two ways. By the old method the patient should be sitting or standing, while the observer sits or stands to one side, and faces him. With the hand furthest from the patient steadying the head, the observer gently but firmly grasps the surface of the cricoid cartilage with the thumb and finger of the other hand, while the head is slightly thrown back. The head is then flexed, so that the neck is no longer stretched. The patient is then told to hold his breath completely, and any up-and-down movement of the trachea is immediately transmitted to the observer's fingers. One must not mistake the transmitted pulsation in the cervical vessels for such movement ; and great care should be exercised to see that the breathing is entirely stopped.

In the other method, as proposed and practised by Ewart (*British Medical Journal*, March 19, 1892), the observer stands behind the patient, steadying the latter's head against his body, and the cricoid is firmly held between the tips of the first or middle fingers. The writer, after considerable experience, prefers this second method, on account of delicacy of touch, firmness of grasp, and comfort to the patient.

Diagnosis. The special points of diagnosis are : the etiological factors ; the antecedent pathological conditions, as arterial sclerosis ; the occurrence of pain ; the occurrence of pressure-symptoms ; and the physical signs. These have been sufficiently dwelt upon, and it is not necessary to consider them again. It must not be forgotten that aneurism may be present without diagnostic physical signs, and, on the other hand, the pressure-symptoms may also be in abeyance. If one of the two is present in the male subject past forty, with a previous history of syphilis, gout, alcoholism, or muscular strain, the probability is that an aneurism exists. The pressure-symptoms

always point to some form of intrathoracic disease as the cause of this group of symptoms. Thus, in cancerous disease of the lymphatic glands, or other tumors within the mediastinum, pressure-symptoms exactly simulating aneurism may be present and also the physical signs of a tumor. The tumor, however, rarely projects externally, and still more rarely pulsates. If pulsation is present, it is not of the expansile character seen in aneurism, nor is there as decided a systolic shock when the ear is held against the chest. By the same method we ob-

FIG. 182.

X-ray appearance in aneurism. (PEPPER and LEONARD.)

serve the shock of the heart-sounds, which are notably lessened or absent in tumors from other causes than aneurism. In deep-seated tumors with pressure-symptoms the condition of the arteries, apart from aneurism, is of diagnostic importance. Accentuation of the aortic second sound, with hypertrophy of the heart, points to aneurism. The presence of tracheal tugging is also a valuable diagnostic point in its favor. In tumor, and especially in cancer, there are emaciation and development of a cachexia, which is, as is well known,

most pronounced in cancer of the œsophagus. Cancer of the œsophagus, from its frequent point of election near the left bronchus, often simulates the pressure-symptoms of aneurism.

Aneurism must be distinguished from the pulsation of the aorta which is seen in aortic regurgitation. This pulsation is usually associated with dilatation, the latter causing increased dulness, which may add further to the confusion. Exaggerated pulsation without dilatation may, as Bramwell has recorded, be the cause of dulness and pulsation over the aorta. The subjects are under forty, neurotic, and usually anæmic.

It is not, as a rule, difficult to distinguish between *pulsating empyema* and aneurism. Wilson points out that aneurism bears a definite relation to the central long axis of the chest. The area of dulness of aneurism is circumscribed, and is usually the seat of murmurs or other sounds synchronous with the rhythm of the heart. The signs of pulsating empyema are usually upon the left side and at a distance from the median line. The percussion-dulness is at the base of the chest and quite extensive. Arterial murmurs are not present. The pulsation is influenced by pressure and by respiratory movements.

In *mediastinal cancer* we are aided by the discovery of enlargement of the glands in the axillary or some other situation, or by a history of the growth elsewhere.

Aneurism must not be confounded with *phthisis*. The diseased vessel may occlude a bronchus and cause collapse and bronchial dilatation; hemorrhage may occur; bronchorrhœa and cough always ensue. Fever is not marked, which fact, with tracheal tugging, vascular physical signs, and the absence of tubercle bacilli, points to aneurism.

X-ray Examination. By virtue of the large amount of blood in an aneurism, the tumor is not pervious to the X-rays, and in consequence is readily seen by fluoroscopic examination. Williams and others have been very successful in recognizing an aneurism even when it could not be made out by physical signs. Such examination must be resorted to in all cases. (Fig. 182.)

Diseases of the Mediastinum.

Inflammation of the mediastinum may be limited to the glands or the connective tissue. Moderate inflammation of the glands, lymphadenitis, occurs in bronchitis and pneumonia, particularly if bronchitis is of specific origin, as in measles or influenza. It is said that such inflammation is of common occurrence in whooping-cough, and may be the exciting cause of the paroxysms. DeMussy and Guitéras have found physical signs of enlargement, characterized by dulness in the upper part of the interscapular region, in cases of this disease and of influenza. Other authorities, as Osler, dispute the possibility of this occurrence, or at least of its recognition by physical signs. Tuberculous inflammation of the lymphatic glands of the mediastinum may give rise, however, to local physical signs. Abscess of the glands cannot be distinguished during life.

Tumors of the Mediastinum.

Cancer and sarcoma are the most frequent forms of tumor in this locality. Hare found the proportion in 520 cases to be as follows: 134 of cancer, 98 of sarcoma, 21 of lymphoma, 7 of fibroma, 11 of dermoid cyst, 8 of hydatid cyst, and the remainder of lipoma, gumma, and enchondroma. With the application of more correct histological methods we now know that sarcoma is more common than carcinoma. The tumor is most frequently found in the anterior mediastinum when one region alone is affected. The disease may be either primary or secondary. In sarcoma it is usually primary. Males are chiefly affected, and most often between thirty and forty. The thymus gland, the lymphatic glands, the pleura, or the œsophagus is the source of origin in all cases, the former the most frequent.

The *symptoms* of mediastinal tumor are chiefly due to pressure. *Dyspnœa* is early and constant, and may be laryngeal, or tracheal from pressure on that tube. In some instances encroachment upon the heart or the vessels causes dyspnœa. Again, the dyspnœa may be due to a pleural effusion which accompanies the growths. Cough of a peculiar character occurs. It is laryngeal, and of a dry, brazen quality. Aphonia may arise from pressure upon the recurrent laryngeal nerves. (See Diseases of the Larynx.) If the bloodvessels are pressed upon symptoms of obstruction occur, depending upon the vessel occluded. Œdema of the upper extremities may occur. If the œsophagus is pressed upon, there is difficulty in deglutition. In some instances the sympathetic nerve is pressed upon, causing hyperæmias and pupillary changes.

The *physical signs* (Plate XXXIV., Fig. 2) are those of a tumor in the anterior portion of the chest, frequently in the præcordial area, which may or may not pulsate; dislocation of the heart, not limited to any position; great dulness and resistance; frequently conduction of lung-sounds and heart-sounds to some distance; at times a systolic murmur; increased size and pulsation of the veins; and physical signs from pressure. (See Aneurism.) It must be remembered that *pain* is more common in aneurism, *fever* and *emaciation* in mediastinal growths.

Tumors of the anterior mediastinum present the physical signs, in front, of a prominence more or less marked, often including projection of the sternum; an irregular area of dulness; rarely transmitted pulsation; more frequently transmitted heart-sounds and lung-sounds. It is the form in which phenomena from pressure upon the veins are most marked. Symptoms from arterial pressure (difference in pulse), pressure on the vagus and sympathetic are less frequent. *Dyspnœa* may occur.

Tumors of the middle and posterior mediastinum are characterized by pressure upon the bronchi and structures adjacent thereto, hence we have symptoms from pressure upon the œsophagus, aorta, and the nerves. *Dyspnœa* and *cough* are the most pronounced symptoms, while phenomena from pressure on the vagus, cardiac palpitation, vomiting, etc., are not uncommon. *Emaciation* and a cachexia are more marked

than in tumors in other regions. Pepper and Stengel consider that fever attends growths in this region with greater frequency.

Tumors of pleural origin have symptoms of acute or subacute pleuritis, with or without effusion. The fluid secured by puncture is usually bloody, rarely chylous, and may contain suspicious vacuolated epithelial cells. A mass may be suspected if there is great resistance to the trocar. If the tumor ulcerate into the lung, the sputa may contain characteristic groups of cells, while hemorrhagic oozing may be suspicious.

CHAPTER IV.

DISEASES OF THE MOUTH, FAUCES, PHARYNX, AND ŒSOPHAGUS.

The Mouth.

The mouth is affected by comparatively few diseases, and most of these are the result of infection or of trauma, or, rarely, are trophoneurotic. The cavity forms a good breeding-place for all forms of organisms, and were it not for the secretions and constant cleansing of the mouth by the passage of food and its physiological labors, diseases would be very common. Indeed, it is possible that such diseases do not take place at all unless there is such perversion of the normal secretion as destroys its antiseptic or antimicrobic qualities. We know but little specifically concerning the changes in the secretions. Clinically, we do know, however, that in conditions of poor nutrition, in wasting diseases generally, and probably in connection with the rheumatic diathesis, there is such change in the secretions as permits pathogenic micro-organisms to exercise their influence upon the mucous membrane. The result of their action is seen in various forms of *inflammation*.

Symptomatology. The symptomatology of mouth-affections is the symptomatology of inflammation : *pain, heat, redness,* and *swelling.*

The Data Obtained by Inquiry.

The subjective symptoms are not characterized by great gravity, but they are most annoying.

Pain. This symptom is most aggravating, because it is excited by the many functional acts connected with the mouth. It occurs in all inflammations and ulcerations except those due to syphilis. It is aggravated by food, by movements of the lips, cheeks, or tongue, and by attempts to discharge saliva. The absence of pain is observed in gangrene.

Heat. The patient complains of heat of the mouth in inflammations.

Dryness. This symptom is complained of in fevers, and by those who are compelled to sleep with the mouth open. It may be a condition of itself, as the following shows :

Dry Mouth. *Xerostoma.* Hutchinson first described a condition of the mouth in which dryness was the chief complaint. The secretions are suppressed entirely, the tongue red and dry, the mucous membrane of the cheeks and palate smooth, shining, and dry. Functional movements are very difficult. The majority of the cases are in women in whom the general health is always impaired. Hayden thinks that

the secretion of the salivary and buccal glands is modified as the result of a central nervous disturbance. In xerostoma there is also dryness of the nostrils and eyes, with intolerable itching. In a case which Harris reported both parotid glands were enlarged and firm but painless.

There is some dryness of the mouth in fevers. It is also symptomatic of chronic gastritis, and may occur in diabetes.

The Data Obtained by Observation.

The objective symptoms are determined by inspection and palpation. By these means we observe the color of the parts of the mouth, changes in temperature, as well as in the size and shape (swelling). The teeth, gums, and tongue are also examined.

Color. The normal redness of the mucous membrane may be increased or diminished in intensity. Pallor is associated with anæmia. Increased redness attends inflammation, and with it the temperature is raised. The mucous membrane is yellow in jaundice, bluish in cyanosis. Both of the latter changes are observed to greater advantage under the tongue. The mucous membrane is the seat of pigmentation in Addison's disease and in argyria. In the former, small oval purplish spots are seen. They must not be confounded with the pigmented spots common after stomatitis in negroes. Eruptions occur in the mouth and may precede external eruptions. This is notably so in measles. In this affection the eruption is seen on the hard and soft palate twenty-four hours before the development of the rash. In smallpox and chickenpox the vesicles are seen.

Shape. Swellings are seen usually as the result of disease of structures about the mouth. The floor of the mouth is encroached upon by glands underneath or by swelling of the cellular tissue. Bone diseases and some teeth affections cause swellings. The dental arch must be observed. Narrowing of the arch is due to adenoid disease or to the habit of thumb-sucking in childhood, much more likely the former.

Fœtor. The odor imparted to exhaled air is peculiar in mouth-affections. It may be a simple fœtor or of a metallic or gangrenous odor. Fœtor attends all inflammations; it is more pronounced in ulcerative and mercurial stomatitis. In the latter it may be metallic.

Hemorrhage. Petechiæ in *purpura* hemorrhagica; submucous hemorrhages in *scorbutus* and severe forms of purpura—*morbus maculosus werlhofii*—are common on the cheeks and on the gums. In ulcerative endocarditis hemorrhagic infarcts are seen. In grave anæmias petechiæ are also seen.

Capillary oozing of blood takes place from the mucous membranes in low typhoid states. The accumulated blood collects about the teeth, on the tongue, etc., and in febrile states becomes dry. Dry incrustations are known as *sordes*.

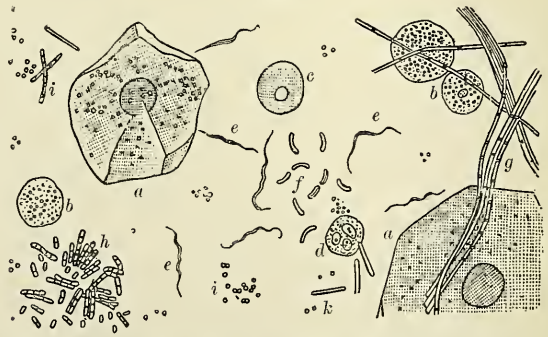
Salivation. Increased flow of saliva occurs in all inflammations unless attended by high fever. It may be constantly discharged by the patient or dribble in a continuous stream. (See Saliva.)

Secretions of the Mouth. The Saliva. The saliva is derived from the parotid, submaxillary, and sublingual glands, and from the mucous

glands within the mouth. The mouth should be washed with a warm alkaline solution and afterward with cold water, in order that the saliva obtained may be perfectly pure for examination. After the washing the glands may be stimulated by the application of dilute acid on a glass rod. The normal amount secreted in twenty-four hours varies from two to three pints. It is of a light bluish color, or colorless. It is somewhat stringy. On standing, two layers form in a conical glass, the upper clear, the lower cloudy. The reaction of saliva is alkaline.

Microscopical Examination. The following formed elements are observed: 1. Salivary corpuscles of the appearance of, but larger and more granular than, a white corpuscle. 2. Epithelium. The squamous variety derived from the mouth is seen. The cells are large in size and of polygonal shape. 3. Fungi. In health the mould and yeast fungi are seldom found. In disease they are present in large numbers; fission-fungi are met with in great numbers, both in health and in disease. In health small and large colonies of micrococci are found along with abundant bacilli. Miller has studied the microorganisms of the mouth carefully and exhaustively (see *The Dental Cosmos*), both by microscopical examination and culture-methods. The following are found to be pathogenetic: (1) The leptothrix buccalis; (2) vibrio buccalis; (3) spirochæte dentium; (4) micrococcus tetragenus; (5) the micrococcus de la rage; (6) the micrococcus of sputum septicæmia; (7) the bacillus of decaying teeth, three varieties of the staphylococcus; (8) the bacillus crassus sputigenus; (9) the bacillus salivarius septicus and bacillus septicus sputigenus.

FIG. 183.



Buccal secretion. (Eye-piece III., obj., Reichert, 1/15, homogeneous immersion; Abbé illumination, open condenser.) Friedländer's and Günther's method. (VON JAKSCH.)

a, epithelial cells; *b*, salivary corpuscles; *c*, fat-drops; *d*, leucocytes; *e*, spirochæte buccalis; *f*, common bacilli of mouth; *g*, leptothrix buccalis; *h*, *i*, *k*, different fungi.

Of course, in the saliva the thrush-fungus, actinomyces, the tubercle bacillus, and the bacillus of diphtheria are found. It must not be forgotten that the diplococcus pneumoniæ or micrococcus lanceolatus, which is the specific cause of pneumonia, is found in the saliva of some persons in health. It is also called the bacillus sputi septicæmici.

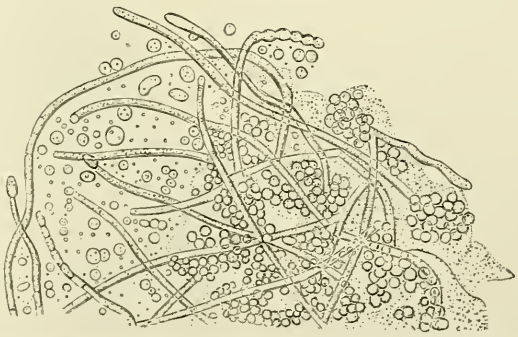
Chemical Examination. The chemical characters of the secretion depend upon the activity of the different glands. The saliva con-

tains a trace of albumin, found by heating; a ferment which changes starch into sugar; mucin; and occasionally sulphocyanide of potassium. In disease, as the quantity is diminished rather than increased, examinations have rarely been made. In *ptyalism* the saliva should be collected after rinsing the mouth frequently, especially after eating. The reaction is found to be alkaline, and the specific gravity low, 1002 to 1006. Albumin is tested for by the usual methods. The sulphocyanides are detected by a solution of chloride of iron. When this is added to the fluid a bright red color appears which does not disappear with heat; a similar color, due to the precipitation of meconic acid, may be obtained by the same test from the saliva in opium-poisoning.

Sugar is tested for by the methods used in the examination of the blood. The diastatic ferment is detected by adding 5 c.cm. of saliva to 50 c.cm. of starch solution and placing the mixture in a warm chamber or a water-bath heated to 40° C. After an hour's time the fluid will show the presence of grape-sugar. Nitrites are detected by adding a little saliva to a mixture of starch paste, iodide of potassium, and dilute sulphuric acid. If the nitrites are present, a blue color results.

Saliva in Disease. In catarrhal stomatitis the secretion is *increased*. It is acid and contains epithelium in excess. In ulcerative stomatitis it is also increased, is of a dark-brown color, fœtid, and alkaline. It contains degenerated epithelium, leucocytes, blood-corpuscles, and many forms of fungi. It is increased in pregnancy, in rabies, and in glosso-labio-laryngeal palsy. I have seen it in excess in the convalescence of typhoid fever. It is increased by the internal use of jaborandi.

Fig. 184.



Oïdium albicans, the vegetable parasite of muguet or thrush. (Reduced from Ch. Robin.)

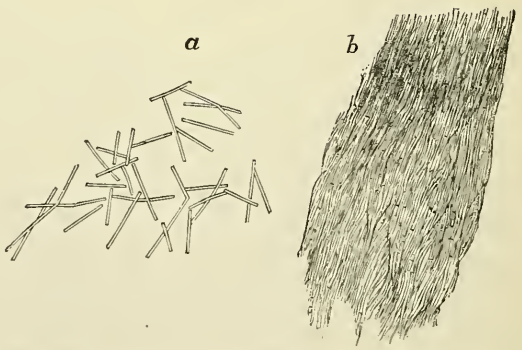
The reaction becomes acid in diabetes, gout, rheumatism, and mercurial poisoning. Urea may be found in cases of nephritis, particularly in uræmia. There is no sugar in diabetes. Fenwick has investigated the changes in the sulphocyanide of potassium in disease. By a scale of colors he was enabled to compare the saliva in which sulphocyanide of potassium had been detected in health with the saliva in various diseases. He believes that the amount of this ingredient is indicative of the degree of functional activity of the organs of nutri-

tion. It is increased in acute inflammation and in the earlier stages of cancer and phthisis; in acute congestion of the liver from stimulants or food excess; and in rheumatism, gout, and the convalescence of typhoid fever. Where the power of the nutritive organs is diminished the sulphocyanide of potassium is lessened, as in late phthisis and cancer, the later stages of chronic diarrhœa and dysentery, chronic catarrhal jaundice, in ascites, and in the passive congestion of the abdominal viscera. Fenwick believes that tedious recovery and frequent relapses will occur if this element is found in excess in acute rheumatism.

Thrush. The fungus peculiar to this disease is found. Saliva is increased; it is usually acid. The disease is characterized by the formation of small patches on the mucous membrane, which in a few days coalesce and form a mass which may cover the entire mouth and extend to the fauces. Before coalescing they are firmly adherent. Subsequently they loosen. On microscopical examination, in addition to epithelial cells, leucocytes, and unorganized elements, the characteristic parasite is seen. It is of ribbon-shape, varying in length, and composed of long segments which often contain highly refractive nuclei at either end. The segments are homogeneous; they vary in length, those nearest the extremities being somewhat shorter. When mounted in glycerin they are readily seen. Spores are also seen.

The Leptothrix Buccalis. The latter is seen in ribbon-like bundles composed of numerous segments; it stains a bluish-red in potassic iodide solution. It is most frequently seen in the tartar of the teeth.

Fig. 185.



Leptothrix buccalis from the gums at edges of teeth. × 350.
a, the filaments separated; *b*, masses of filaments.

The Gums. The gums and the mucous membrane of the mouth are involved in inflammations and ulcerations, and in certain metallic poisonings. The gums swell and grow spongy in inflammations.

The Gingival Line. In cases of tuberculosis a red line at the junction of the gums and the teeth is frequently seen. At one time it was thought to be of diagnostic value. It is seen, however, in other cachectic conditions, as carcinoma, and at times in diabetes.

The Gums in Scurvy. In scurvy the gums are swollen and spongy. They bleed easily, and are usually streaked with blood. Ulcers form along the margin of the teeth. There is not much fœtor of the breath. In mild cases the inflammation may be limited to the gums of four or five teeth. The gums of decayed teeth are usually the seat of the most marked inflammation. Infants may have scurvy as well as adults—especially if fed exclusively on sterilized milk or malt preparations. (See Scurvy-rickets.)

The Gums in Lead-poisoning. The Blue Line. In lead-poisoning a blue line is seen at the margin of the gums. The line is preceded by a row of separate black dots occupying the seat of the papillæ of the mucous membrane. If examined with a magnifying glass, the line is readily seen to be an interrupted one. It does not always extend along the entire margin, but may be limited to a few front teeth in either the upper or lower jaw. In the more advanced cases there is some salivation and a sweetish metallic taste in the mouth and metallic fœtor of the breath.

The Teeth. In all diseases of the gastro-intestinal tract it is important to investigate the state of the teeth. Cases of indigestion are often due to defective mastication, rendered so by decayed teeth. Persistent aural, nasal, and ophthalmic affections may have their primary origin in disease of the teeth. Caries of the teeth may cause headaches or neuralgias, near or remote (see Headache), and may explain many cases of foul breath. Pitting of the surface of the teeth and thinning of the enamel in transverse grooves are held by some to be due to mercury. There is no doubt that infantile stomatitis, independent of mercury, is the cause of these changes. They must be distinguished from the so-called Hutchinson's teeth. In stomatitis the molars are often honeycombed to an extreme degree, the incisors becoming affected next. In addition to pitting and erosion the color may be darker. A transverse furrow crosses all the teeth at the same level.

The Teeth in Gout. Erosion of the teeth takes place in gouty subjects. There are wasting and loss of polish of the labial surface, followed by deep grooves which extend into the body of the teeth. *Pyorrhœa alveolaris* is another expression of gout. There is, first, usually a marginal inflammation of the gums; second, inflammation and necrosis of the pericementum; third, loosening of the teeth and the formation of so-called calculi.

The Teeth of Congenital Syphilis. The upper central incisors of the permanent set are affected. They are dwarfed, narrowed, and short.

Fig. 186.

Notched teeth. Malformation of permanent teeth found in hereditary syphilis. (Mr. Jonathan Hutchinson.)

The middle lobe of the tooth is so atrophied as to leave a single broad vertical notch in the edge of the tooth. A narrow furrow some-

times passes upward from the notch on both anterior and posterior surfaces, nearly to the gum. It is seen from the above that the appearances of the permanent teeth may be an index of the condition of nutrition of the child in infancy.

Teething. During the period of infancy it is well to remember the influence of the eruption of the teeth upon the general constitution. While many prominent authorities believe that the eruption takes place without the occurrence of general or reflex symptoms, equally careful observers, on the other hand, believe that nervous phenomena often attend the process. The latter class of observers attributes the feverishness, insomnia, restlessness, loss of appetite, and gastro-intestinal disturbance to this cause. Convulsions at this period are believed to be due to the pressure of the tooth, which cannot break through the mucous membrane, upon highly sensitive nerves at the root. Even in later life reflex convulsions are held by some to be due to the teeth.

Slowness in the development of the teeth may be due to rhachitis, which should be looked for. The student should be familiar with the periods of development, the number of teeth that appear at each period, and the date of the eruption.

Dates of Eruption of the Teeth.

Milk Teeth.

$$\frac{2\,M\quad 1\,C\quad 4\,I\quad 1\,C\quad 2\,M}{2\,M\quad 1\,C\quad 4\,I\quad 1\,C\quad 2\,M} = 20$$

Eruption of	central incisors about	7th month.[1]
"	lateral incisors "	9th "
"	first molars "	15th "
"	canines "	18th "
"	second molars "	24th "

Permanent Teeth..

$$\frac{3\,M\quad 2\,B\quad 1\,C\quad 4\,I\quad 1\,C\quad 2\,B\quad 3\,M}{3\,M\quad 2\,B\quad 1\,C\quad 4\,I\quad 1\,C\quad 2\,B\quad 3\,M} = 32$$

Eruption of	anterior molars about	7th year.
"	central incisors "	8th "
"	lateral incisors "	9th "
"	anterior bicuspids "	10th "
"	posterior bicuspids "	11th "
"	canines "	11th "
"	second molars "	12th to 14th year.
"	third molars (wisdom teeth) about	18th to 25th "

Stomatitis. This inflammation is not limited to the mouth alone, but extends to structures within the mouth, as the gums, and may invade the tongue. The inflammation is recognized by the subjective and objective signs common to such inflammations. There is pain, and hence the child (for it usually occurs in children) refuses to nurse or take the bottle, or cries when food is given. The pain is accompanied by fœtor of the breath. This occurs in all forms of stomatitis. Its origin, as well as the origin of the pain, is readily determined by inspection.

[1] Lower incisors first.

On inspection we note the usual signs of inflammation. They are rarely general, being, as a rule, localized to small areas, which may rapidly become ulcerated. When general the mucous membrane is red and hot; the color extends to the gums, lips, and tongue. This is seen in the *catarrhal* form; the follicles are also enlarged. The tongue becomes red and smooth, or may be covered with a white coating, through which the prominent red fungiform papillæ project. Accompanying the inflammation there is increased secretion, which dribbles from the mouth, or is constantly discharged by older patients. The red hue of the mucous membrane is attended by swelling. The heat of the mouth is often sufficient to raise the temperature of the exhaled air, so that the breath is hot.

A peculiar form of inflammation of the mouth is seen in gouty subjects. It occurs at intervals. Pain is not so marked, but the heat, redness, and burning are associated with a superficial glossitis and salivation. The saliva is highly acid, and causes a dermatitis on the chin. Other mucous membranes are involved at the same time, as the vagina. An acid mucoid discharge sets up irritation at the vaginal outlet and causes much distress.

Aphthous Stomatitis. Local areas of intense inflammation are sometimes followed by ulceration. Thus in *aphthous stomatitis* small yellowish-white spots appear, at first discrete, but soon dotted over the mucous membrane inside of the cheeks, in the roof of the mouth, along the sides of the gums, and on the tongue. They subsequently break down into shallow ulcers with raised red margins.

Aphthous ulceration is seen in *foot-and-mouth disease.* The local process is characterized by greater swelling, with softening and ulceration of the soft parts, than in other stomatitis. In foot-and-mouth disease there is a history of infection, profuse diarrhœa, followed by constipation, and considerable physical depression.

Ulcerative Stomatitis. The disease occurs in ill-nourished subjects, and is often intercurrent with exhaustive disease, as chronic diarrhœa. It may be seen in epidemic forms in camps and in penal and other institutions, on account of unsanitary conditions. In *ulcerative stomatitis* the inflammation is more pronounced on the gums. They are swollen, red, and covered with ulcers. The gums in which teeth remain are affected, and the ulcers are usually at the gingival border. Gums without teeth are not affected. The ulcers are covered with yellowish material. The flow of saliva is much increased in this affection. It is acid in reaction. The submaxillary glands are enlarged. The fœtor of the breath is very great.

Parasitic Stomatitis. Thrush. In *parasitic stomatitis,* or *thrush,* raised white patches are seen looking like small curds of milk. The patches vary in size, and on the tongue may cover an area as large as a three-cent piece. (See page 690.) The white patches are distinguished from milk-curds because they cannot be removed by the napkin or brush. The parasite has been called the *oïdium albicans* (see Fig. 184); but Forchheimer prefers to group it under the saccharomyces.

Stomatitis Materna. Painful ulcers occur in the mucous membrane of the lips and cheeks in nursing-women. They are solitary, and interfere with mastication.

Gangrenous Stomatitis. The affection appears as a gangrenous inflammation of the gums, mucous membrane, and deeper tissues of the cheek. At first a small, dark red, hard spot is seen, which increases in size, and becomes of a purplish color. The cheek rapidly becomes swollen, tense, and brawny. On the surface of the more indurated portions a bleb forms which soon breaks with rapid ulceration. The ulcer is dark and gangrenous and soon perforates the cheek. It extends to the jaw and is followed by necrosis of that bone. The characteristic odor of gangrene attends the process. While the affections previously mentioned are generally dependent upon poor nutrition, *gangrenous stomatitis* is always secondary to depraved, depressed, or debilitated states of the system. Cases may occur simultaneously in asylums for children in which the hygienic conditions are bad and the food-supply poor.

Mercurial Stomatitis. Mercurial stomatitis, or ptyalism, particularly affects the gums. It also involves the salivary glands. The inflammation is caused by mercury. It may occur from the medical use of the drug, particularly in persons who are unduly susceptible, or are not particular in regard to mouth-cleansing. The inflammation is painful and attended by profuse discharge of saliva, hence the name, *salivation*. The tongue is swollen, marked on the sides by the teeth, and may be protruded with difficulty on account of its size. It is tender to the touch. It is covered with a heavy, creamy coating. The gums are swollen, red, sore, and bleed on the slightest touch. Ulcers along the border occur, may become diffused, and in some instances extend to the jaw. The teeth become loosened. The fœtor of the breath is heavy, offensive, and of a metallic character. The inflammation is usually preceded by a metallic taste in the mouth, and the patient notices pain on mastication, which increases in severity as the inflammation develops. In mild cases it is limited to the gums, in others the tongue and salivary glands and the mucous membrane of the mouth are affected.

Leprosy. This affection frequently invades the mouth. The nodular and ulcerative lesions are seen. It is always associated with the characteristic lesions of the skin. Scraping or sections would show the characteristic micro-organism.

Glanders may invade the mouth from the nasopharyngeal space.

Actinomycosis results from the entrance of the ray-fungus through carious teeth or an abraded mucous membrane. Often there is first disease of the alveolus, as pyorrhœa, or a periosteal abscess; then the jaw is involved. Before this a general stomatitis may be set up.

Ulcers. In addition to the above forms of ulcerative stomatitis, solitary *ulcers* are seen in herpes, secondary to gastric or uterine disturbances, and syphilis. The herpetic ulcers are of frequent occurrence at the menstrual period or during the course of lactation. The tendency to their formation is often hereditary. I have seen them occur at the menstrual period or in pregnancy in the women of three generations. In the secondary stage of syphilis mucous patches are seen as bright red, symmetrical, oval, or crescentic patches or erosions, occurring on the mucous membrane, sometimes on the tongue and

fauces. They are generally covered with a scanty grayish-white secretion, and are not usually painful.

Sublingual Ulcer. This form occurs on the frænum of the tongue. It is seen in whooping-cough, and is due to the rubbing of the tongue against the teeth in the act of coughing.

Scleroderma. This rare tropho-neurosis occasionally invades the mouth. It is characterized by a submucous infiltration of cartilaginous hardness, the surface of which is denuded of epithelium or covered with crusts. The invasion comes from the nostrils or the nasopharynx. Later the infiltration changes to a yellowish-red or a tendinous-like scar.

The Tongue.

Examination of the tongue is made for diagnostic purposes with a greater show of wisdom on the part of the examiner, and greater satisfaction to the patient, but with less satisfactory results from a diagnostic stand-point, than the examination of any other portion of the body. The mucous membrane of the tongue is examined because it is the only mucous membrane of the body, except the oral and faucial, which is open to inspection, and is, therefore, supposed to enable us to judge of the effects of general diseases upon mucous membranes. It is thought to be indicative of disorders of the gastro-intestinal tract because of its relations with it, but recent studies by Hutchinson, Butlin, and other observers have resulted in the promulgation of different views. Both the above-mentioned distinguished gentlemen are surgeons, and look upon the tongue as a local organ. Investigating it as such, they concluded that the changes in the coating, which had been considered to have so much clinical significance, depended largely upon parasitic invasion, and were not due to changes in the epithelium. The parasitic invasion, they hold, is largely dependent upon local conditions, which, it is true, are on their part dependent upon a state of the system. Since the writings of Hutchinson and Butlin, Dickinson returned to the investigation on the lines laid down by older teachers, and has, in a measure, restored the tongue to its original position as a diagnostic feature in an estimation of the state of the general system and in diseases of the gastro-intestinal tract.

We study the tongue to ascertain its color; the character of eruptions if they are present; the occurrence of indentations, excoriations, furrows, or fissures; the occurrence of ulcers and of patches. Plaques, nodes, and nodules are also seen on the tongue. *Inflammation* of the tongue occurs, and it is the seat of *atrophy* and *hypertrophy* and of the various *tumors* in the parasitic diseases. The *movements* of the tongue are also observed, as an indication of the power of muscles which are under centric influence closely related to important centres in the medulla oblongata. Surgical affections of the tongue will not be considered; local affections will only be referred to in connection with general diseases.

Discolorations of the Tongue. Yellowish-white, oblong patches, soft, but slightly raised, are sometimes seen along the sides of the tongue—*xanthelasma.* They are sharply defined, and vary in size from

a split pea to a three-cent piece. Xanthelasma is also situated upon the eyelids and upon the palms of the hands, rarely in other portions of the body. It occurs in jaundice, or in persons who are said to be subject to bilious attacks.

Pigmentations. Dark purple, bluish-black, or black marks are seen on the tongue as well as on the surface of the lips, where they may be brown. They are sharply defined, neither raised nor depressed, and vary in size. Such pigmented spots are seen after glossitis and in Addison's disease. In the latter affection other pigmented areas are found. *Blood-stains* are observed in purpura. Bright red spots the size of a split pea or larger, patches, known as *ecchymoses*, are of frequent occurrence. They are not removed by pressure. Hemorrhagic *infarcts* are sometimes seen on the tip of the tongue.

Black Tongue. This rare condition is of parasitic origin. It has recently been described anew by Cohen. It is also known as *nigrities*.

The affected portion is of a brownish-black or black color, varying in size and usually situated in the middle of the dorsum of the tongue. It looks like an iron-stain, and in some instances the surface is roughened. The papillæ are abnormally enlarged. It usually begins as a small spot, and extends slowly, so that at the end of a month the dorsum is covered. The centre is blacker than the circumference. After the entire dorsum is covered the spot begins to disappear from the circumference toward the centre, and is followed by desquamation. This series of phenomena is repeated and the entire affection subsides slowly. Desquamation may last from a few days to two months. The papillæ of the affected surface, too, look like "a field of corn laid by the wind and rain." The sensations of taste and touch are not altered, but a feeling of dryness is marked. It must be remembered that a black tongue is sometimes the result of deliberate deception.

Inflammation of the Tongue. *Acute glossitis* is a rare affection, more common in adults than in children, and more frequent in men than in women. It occurs more frequently in the summer. The onset is rapid. After a short period of tenderness on mastication the movements of the tongue are stiff and painful, or there are pains in the muscles of the neck and submaxillary region. In a few hours the tongue swells. It rapidly increases, and at the end of fifteen to twenty hours is three times its natural size, protrudes from the mouth, is indented by the teeth, and is almost immovable, feeling heavy, painful, and tender. It is coated with a thick fur on the dorsum. Salivation accompanies these symptoms, speech is impossible, dysphagia extreme, and dyspnœa not unusual. The glands underneath the jaw are swollen. The temperature rises to 101°, rarely above it, even if the case is severe. Death may occur in a few hours from suffocation, or after a longer interval from diffuse suppuration, gangrene, exhausting septic fever, or pneumonia. Gangrene is more frequent than spontaneous resolution. If resolution is to be established, the swelling begins to subside in three or four days. Small ulcers form on the surface of the tongue, and by the end of a week its normal appearance is regained. The fever and distressing symptoms subside with the local swelling. It is said to be due to colds, to bites and stings of animals, to mercury, and to corrosive and

acrid substances. It may occur in fevers. The diagnosis is easy. It must be distinguished from acute œdematous swelling due to salivary calculus or affections of the floor of the mouth. Acute *ranula* sometimes causes considerable swelling of the tongue, simulating acute glossitis. *Hemiglossitis* sometimes occurs. The local symptoms are not so great, because only half of the mouth is occluded. I saw a case in which the inflammation was limited to half the side of the tongue on the posterior surface. It went on to suppuration, but was not attended by serious symptoms, except discomfort in eating. It was preceded by a definite nodule in the substance of the inflamed part. Glossitis from mercurial poisoning has been described in connection with stomatitis.

Chronic superficial inflammation of the tongue may also occur. The surface is smooth and deprived of papillæ over the affected area, which is redder than natural. The margin of the raw patch is sharply defined, but the area has no depth. The epidermis alone is removed. When associated with dyspepsia it covers a considerable area of the surface of the tongue. The tongue may be deprived of papillæ on the anterior part of the dorsum while the fungiform papillæ remain. The tongue is enlarged and the borders marked by the teeth. The surface looks glossy. The tongue feels stiff and uncomfortable. Movement is irksome, irritating foods are painful. Spirits and tobacco cause distress. Indiscretions in diet and slight traumatism quickly produce fresh inflammation. One observer, Hack, has described a form of glossitis hereditary and peculiar to women. He observed a row of long, oval areas, caused by previous inflammation. They commenced in early childhood. The tongue was smooth over remaining large areas, with red excoriations here and there. There was no syphilis.

Sequelæ of glossitis. Indentations occur when the tongue is swollen, as in mercurial and other forms of glossitis. The borders of the tongue are indented by the pressure of the teeth. But in states of debility a flabby tongue with indented borders is often seen. Sometimes the swelling is so great that the pressure of the teeth causes ulceration.

Furrows, or grooves and wrinkles, are seen on the dorsal aspect of the tongue. They are not necessarily tokens of disease; in many persons they are of constant occurrence. *Furrows* vary from a few lines to an inch or more in length. In many this is most striking in the middle line of the tongue. The median furrow is liable to become ulcerated on slight provocation. The edges of the fissures are smooth and without papillæ or fur. Other furrows are directed horizontally and vary in depth. They may be curved and forked. They are more frequent in older persons, especially if the tongue is too large to lie within the circle of the teeth. They are an evidence of past inflammation, or rarely of hypertrophy. They resemble the median furrows as regards smoothness and absence of fur. Inflammatory furrows occur in chronic superficial inflammation, but more commonly after chronic inflammation which has left the tongue enlarged. The furrows are sometimes so abundant that the surface of the tongue looks like the eyelid. The raised areas become sore, due to irritation of a foreign body (food) or a tooth. They are an indirect result of inflammation.

True inflammatory furrows, described as *dissecting glossitis* by Wunderlich, occur. Dissecting glossitis is only a more aggravated form of superficial glossitis. Furrows of this character may be due to syphilis, and dissecting glossitis sometimes has a syphilitic origin. Fissures and clefts are frequently caused by the rubbing and deep indentation of a rough and jagged tooth. The area around the fissure is inflamed and its base indurated. The sides and bottom are ulcerated. It is recognized by its relation with the offending tooth. It may be mistaken for syphilis, another common cause of fissures.

Syphilitic Lesions. It must be remembered that the tongue is always predisposed to inflame and ulcerate in syphilis. In *secondary syphilis* fissures are always found on the borders of the tongue; they are almost certain to occur if the teeth irritate the border. They may be due to the ulceration of a mucous tubercle which is developed upon the border of the tongue. The ulcer is stellate, and gradually deepens until it becomes a foul fissure. Two processes cause the ulceration—the specific infection and the irritation of the teeth. Syphilitic *ulcers* are not very angry, as are non-syphilitic sores and fissures which may occur in persons in poor health. They may be sensitive, however, on account of the involvement of the tongue. The absence of active inflammation, the large number of sores and fissures, and the association with other lesions of the disease upon the tongue, cheeks, and lips point to their syphilitic origin. *Tertiary* syphilitic ulcers are more pronounced and deeper than other forms. They may be as long as two or three inches; they are sinuous and branched. Gummata may occur on the tongue at the same time. The gummata may be circumscribed or linear, and may break down and ulcerate. *Sclerosis* of the tongue, as described by Fournier, follows the healing of these ulcers. It is curious to note that the lymphatic glands are seldom enlarged in association with syphilitic fissures. The fissures must be distinguished from *carcinoma* and *tuberculosis*. In carcinoma there is a distinct tumor, which may become fissured. Tuberculous ulceration is a sign of the presence of tubercle in other organs. The tuberculous fissures are small, at first single; tubercle, however, rarely begins as a fissure, but as tuberculous ulcers on the tip or borders of the tongue. They are stellate or irregularly branched. They are shallow at first, and deepen later, but do not widen in a corresponding manner. The lymphatic glands are always involved. (See Tuberculous Ulcer.)

Ulcers of the Tongue. They may be simple, aphthous, or traumatic. *Simple ulcers* follow long-standing superficial glossitis. They form in the centre of the tongue, or of the inflammatory area. They are due to sloughing, or simple melting away of epithelium. The ulcer is smooth, red, glazed on the surface. The edges are callous and inactive, and the shape irregular. It is sensitive, and may be painful. The signs of chronic glossitis continue with it. *Dyspeptic* or *catarrhal* ulcers occur on the tip, or on the dorsum near the tip. The dorsum of the tongue, from the tip backward, is very red, and filiform papillæ are absent. The ulcers are small and superficial without definite shape or character, except that they are red and irritable. Dyspeptic ulcers may occur from the breaking down of vesicles on the

tongue. They are small, circular, well-defined ulcers, with sharp-cut edges, in size from a pin's head to a split pea, and are the source of considerable pain and much annoyance. They are recurrent. Salivation may attend them. *Aphthous ulcers* are seen in children and adults, and when multiple are attended with the same symptoms as aphthous ulcers of the mouth, with slight fever. Fœtor is characteristic. When single they occur with indigestion, or in women at the menstrual period. The tendency to their formation is inherited. *Traumatic ulcers* from sharp teeth may persist a long time if the general health is bad. When indolent they may be mistaken for syphilitic, tuberculous, or cancerous ulcers. The rapidity of formation, the location opposite a rough tooth, and the absence of other signs of syphilis point to the true nature of the ulcer. *Chancre* can be excluded by the greater hardness and circumscription of the lesion, its seat near the tip, and its association with enlargement of the lymphatic glands. The latter is not present in traumatic ulcer, unless it is acute and angry. Traumatic ulcer is distinguished from tuberculous ulcers by the absence of signs of tubercle in other organs and by the result of an examination of the scrapings of the ulcer; from cancer by the age. In cancer all the glands become affected later.

Excoriations on the surface of the tongue, or rawness, arise from injury, and may also be seen in dyspepsia.

Tuberculous Ulcer. The tuberculous ulcer presents an uneven, pale, flabby surface, covered with a yellowish-gray viscid or coagulated mucus. The edges are sometimes sharp-cut, sometimes bevelled, seldom elevated. They are not usually very red. There is but little surrounding inflammation, and the adjacent portions of the tongue are but slightly swollen. The borders of the ulcer may be sinuous, and the shape oval or ovoid, or elongated. In the neighborhood of an ulcer a number of tiny yellowish gray points may be observed. The ulcer is painful, and attended by salivation. I saw in the Philadelphia Hospital a case of tuberculous ulcer of the tongue, in a young man twenty-five years of age, with pulmonary and intestinal tuberculosis. The dorsum of the tongue was covered with a dozen ulcers, with sharp-cut edges and pale, flabby granulations, without induration or inflammation around them. They were yellowish-gray, and tubercle bacilli were found in the scrapings. Tuberculous ulceration must always be carefully distinguished from syphilitic and cancerous. The associate symptoms are often most reliable. Ulcers due to lupus are also seen upon the tongue.

Patches and Plaques. Space forbids further consideration than the naming of the plaques which are seen on the tongue. First, there is the smoker's patch, on the middle of the dorsum about the point where the tobacco-pipe rests, or where the stream of smoke from the pipe or cigar strikes the tongue. This is a slightly raised area of oval shape. It is not ulcerated, but is smooth and red, or livid. Sometimes it is bluish-white or pearly in appearance. The smoothness is characteristic. White and bluish-white patches or plaques are seen in *leucoma, leucoplakia, ichthyosis, keratosis,* and are also known as opaline plaques. The smoker's patch belongs to the same class, and is proba-

bly an early stage of these affections. It is a whiteness, or white opacity of the surface of the tongue, usually on the dorsum. It is almost always the result of the direct action of irritants. These patches are unknown under twenty years of age, do not commence after sixty, and very rarely attack women. They are not attended by subjective symptoms usually. There may be a sensation of induration and dryness. The course is always chronic.

Wandering Rash. Ringworm, or circular exfoliations—the geographical tongue—occurs most frequently in children. One or more patches on the dorsum of the tongue are observed, smooth and red, but not depressed or elevated. The filiform papillæ have been shed. The patch spreads and becomes a ring, circular or oval. The border is faintly or decidedly yellow, and usually slightly raised and sharply defined. The circles may widen and contract from time to time. No subjective symptoms are noted except itching in a few cases. The cause is not known. The diagnosis is easy. It may continue for months or years.

Mucous patches are multiple lesions of syphilis in the mucous membrane. They have been referred to in the section on Diseases of the Mouth.

Eruptions. Eruptions of variola, measles, and erysipelas are seen on the tongue. Herpes and aphthous ulcers, preceded by vesicles, are met with on the surface of the tongue.

Nodes. Nodules in the tongue are always tuberculous or syphilitic.

Atrophy. Atrophy of the tongue is very unusual. Hemiatrophy may occur as the effect of central or peripheral causes, as softening, hemorrhage, or tumors of the region of the hypoglossal nucleus. Other centres near the nucleus are affected, hence other forms of paralysis are seen, due to the lesions of the medulla. These are seen in progressive muscular atrophy and bulbar paralysis, and in cases of hemiplegia. It is not difficult to recognize it on inspection. The functions of the tongue are not affected.

Hypertrophy. Enlargement of the tongue, or *macroglossia*, is generally congenital, but may occur late in life. The tongue enlarges, and is accompanied by pressure symptoms due to such enlargement. Hypertrophy of the tongue is sometimes seen in idiots and cretins. The hypertrophy is more frequently the result of lymphatic obstruction, on account of which there is lymph-stasis. The diagnosis is easy. Inflammatory hypertrophy occurs in stomatitis, and syphilitic hypertrophy occurs with gummata.

Cysts. Various cysts occur in the tongue. Mucous cysts and blood-cysts are the most common. The cysticercus cellulosæ and the echinococcus occur rarely. Ranula is a cyst underneath the tongue that causes suffering from mechanical obstruction. It is easy of recognition.

Parasitic Disease. Thrush is the most common. Other infections of the mouth extend to the tongue in most instances.

The Tongue in General and Remote Disease. The Coating. With a view to estimate the condition of the system in general by the appearances of the tongue, excluding all local conditions, the

following characteristics are observed: First, the color; second, the fur; third, the degree of moisture; and, fourth, the movements. The student should bear in mind that changes in the condition of the tongue are frequently of local origin; that dryness, for instance, may be due to the open mouth, or that a coating may be unusually marked because the tongue had not been used in mastication. Often coating is seen on one side of the tongue. This has been referred to as due to disease of the nerves of one side. It is just as likely to be due to an absence of mastication on that side of the mouth, the bolus of food being kept on the other side because of pain, diseased teeth, or other local cause.

Clinical experience has shown that certain conditions in the tongue are associated with certain general conditions which render the appearance somewhat diagnostic. The term diagnostic must be qualified, because the changes are so often local, or are modified by conditions independent of the general system. For convenience, the classification of Dickinson as to the appearance of the tongue in disease may be utilized. In the Lumleian lectures this eminent authority described the average healthy tongue based on extensive observations. Departures from the normal were arranged and afterward classified. It resulted in the formation of eleven classes:

1. The Stippled or Dotted Tongue. The tongue is moist and dotted with little white points, due to an excess of white epithelium on the papillæ. It is usually seen in persons in poor health without fever. It is not, therefore, a febrile tongue, nor one indicative of grave constitutional disease. It is seen in cases of chronic disease, usually one in which there are no grave symptoms.

2. The Dry Stippled Tongue. This is found in mildly acute diseases, or in cases in which the constitutional disturbance is more marked.

3. The Stippled and Coated Tongue. The patients in whom this is found are very frequently the subjects of acute and constitutional affections. Fever is more frequently present with this variety of fur.

4. The Coated Tongue. There is excess of white epithelium on the papillæ, and the coat is continuous. The intervals between the papillæ are more commonly filled up with epithelium and accidental matters than in the preceding types. It is seen in acute and febrile diseases, and whether moist or dry, in pneumonia, pleurisy, and typhoid fever. It is associated with a far greater degree of prostration and pyrexia, while the saliva is absent in the larger proportion of cases.

5. The Strawberry-tongue. The tongue is coated and injected; the fungiform papillæ shine through the coat, particularly at the tip and edges. It is the tongue of scarlet fever, but may often be seen in any acute febrile disorder. In scarlet fever, however, it appears by the second or third day—most marked after the second. Pyrexia is more common in this class than in the preceding.

6. The Plaster-tongue. A thick, uniform coat, edges abrupt and striking, covers the tongue. The papillæ are elongated and the intervals crowded with accumulations, among which are bacteria; it is the tongue of acute febrile disease. Fever was marked in a number

of cases Dickinson studied, and prostration was a common attendant. Saliva was deficient.

It is thus seen that, beginning with the healthy tongue, Dickinson described a series of groups, in each succeeding one the coating becoming more marked, with or without moisture. The clinical association that he found is a common experience. Each successive group was attended by more fever, greater exhaustion, and less saliva than the preceding group, and in each the tongue became more and more furred.

7. The Furred or Shaggy Tongue. When moist the papillæ are greatly elongated, composed mostly of horny epithelium. It has the same appearance as if the tongue were dry. The moist, furred tongue is not so common as the other. It is most commonly seen in old age and in constipation. The dry, furred, or shaggy tongue may succeed the dotted tongue or the coated tongue in the course of advancing disease. It is the result of disease and want of moisture. The saliva is deficient; it indicates that there has been fever, and that possibly but little food was taken.

8. The Incrusted, Dry Brown Tongue. Over the surface of the tongue there is a dry, thick, felted coat, which is continuous and dips down between the papillæ. The coat is largely made up of parasitic material. In the course of fevers it is the outcome of a preceding condition, the coated tongue, and is indicative of the typhoid state. It occurs in the fevers with high temperature, but may be seen in conditions of low temperature, as from cancer, phthisis, albuminuria, chronic nervous diseases. There is much depression or prostration associated with it, and there is absence of saliva. If the patients with a dry brown tongue recover, it retrogresses to the furred or incrusted tongue, which in turn becomes bare gradually, at first in small layers; the latter is thin, usually dry, but is more moist than the dry brown tongue. As the incrustation disappears it may become bare, red, and dry.

9. The red dry tongue indicates a more serious condition usually than the dry and brown. It is the tongue of chronic wasting diseases. It occurs in phthisis in the later stages, and, as the raw-beef tongue, is associated with dysentery and also with liver abscess. There may be fever associated with the cases. It is in a measure the tongue of chronic diarrhœa. The tongue is shrunken, red, polished, and smooth. The papillæ have disappeared and the epithelium is stripped off in patches. It may be associated with aphthæ. If the patient is to improve, the redness fades, the papillæ become softer, and the moisture returns.

10. Red and Membranous; otherwise as (9) the red denuded tongue.

11. Cyanosis, or Venous Congestion of the Tongue. The tongue is of a bluish or purplish color, the surface is smooth and wet, and the papillæ are almost indistinguishable. It is not confined to organic heart disease or cyanosis. It is of quite frequent occurrence in albuminuria. With the venous congestion in the albuminuric cases there is always a superabundance of deep epithelium. When the surafce is examined it looks as if the papillæ were fused together and overlaid by a moderate coat.

CLASSIFICATION OF TONGUES.

To the naked eye.	*Microscopically.*
1. Healthy, moist.	White epithelium in small amount on papillæ, not continuous or superabundant.
2. Stippled, moist, dotted with white.	Excess of white epithelium on papillæ, not extending between them.
2 (D).[1] Stippled, dry.	Ditto.
3. Stippled + coated; moist. Coat continuous in parts.	White epithelium on papillæ in excess, with partial filling of intervals.
4. Coated white; moist. Coat continuous.	Excess of white epithelium in papillæ. Intervals more or less filled up with epithelium and accidental matter.
4 (D). Coated white, dry. Coat continuous.	Ditto.
5. Strawberry, coated + injected, especially showing in fungiform papillæ.	Like the coated or plastered, but with more injection.
6. White, plastered, thick, uniform coat; edges abrupt and striking.	More elongation of papillæ than with coated tongue, more filling of intervals with superficial accumulation.
7. Furred or shaggy, moist. Greatly elongated papillæ.	Extravagantly long papillæ, mostly of horny epithelium.
7 (D). Furred or shaggy, dry.	Ditto.
8. Incrusted, dry, brown; thick, felted dry coat over papillæ.	Continuous crust on and between papillæ, largely of parasitic matters.
9. Furred or incrusted, becoming bare. Generally dry.	Crust breaking away, together with more or less of normal surface.
10. Red, denuded. Absence of normal covering.	General absence of all epithelium excepting the Malpighian layer; sometimes of that also.
11. Red, smooth, dry, membranous covering.	Level membrane replacing epithelial processes.
12. Cyanosed.	Injected; hypernucleated; excess of deep epithelium.

MOISTURE OF THE TONGUE. The moisture is due to the saliva, any deficiency of which causes dryness of the tongue. It is natural, therefore, to conclude that any changes in the moisture of the tongue are due to altered secretion of the salivary glands. This is almost always deficient when fever is present, and hence the tongue is dry.

[1] The letter D is used to imply dryness. Thus, to Class 2 a certain description is attached. Class 2 D presents the same characteristics with the addition of dryness.

At the same time, it must be remembered that this failure of secretion of the salivary glands does not depend upon gastro-intestinal disturbance.

Dryness of the tongue, it must not be forgotten, may be due to increase of evaporation from keeping the mouth open, as well as to diminution of the salivary secretion. All states, therefore, in which the mouth is open will lead to dryness of the tongue. Again, in chronic fever, dryness of the tongue is a constant characteristic. Dryness is due to the effects of the temperature upon the secretions in general, but it is not the effect of high temperature, curiously, but rather a temperature which has persisted for a considerable length of time. Thus, in pneumonia, with a temperature of 105°, the tongue may be moist; whereas, in typhoid fever, with a temperature of 103°, the tongue is dry. General dehydration of the body causes dryness of the tongue, even without local diminution of secretion. This dehydration is seen in diarrhœa, in which disease simple or uncomplicated dryness of the tongue is the common symptom. It is curious to observe that in cholera the tongue remains moist even until death; whereas, if the patient is about to improve and the discharges cease, reaction and fever setting in, the tongue begins to dry and becomes quite brown. Local causes may explain this. The watery vomit may keep the tongue moist, and the temperature of the body may contribute to the change. Next after diarrhœa we have excessive discharge of urine as a frequent cause of dryness. Hence, in diabetes in all forms extreme dryness of the tongue is seen. The osmotic action of the sugar in the blood is the cause of a reaction in diabetes mellitus, just as it is in cases of dehydration of the lens in cataract. The final cause of dryness of the tongue is prostration. Asthenia in all forms continuing over a moderate period of time, as a week or ten days, causes lingual dryness.

The Effects of Food. These must be studied before deciding upon the clinical significance of changes in the tongue. The immediate results of taking of food influence the coating and the degree of moisture. The act of eating cleanses the tongue. In disease, therefore, in which this act is not performed, it is natural that we observe more fur on the surface, and in conditions in which diet is limited to fluids the effect is marked. In cases of liquid diet the tongue is likely to remain furred. It is particularly seen in patients who are kept upon a milk-diet exclusively.

The Tongue in Relation to Diseases of the Alimentary Canal. So much has been written on this subject that it is well to give the experience of Dickinson briefly. He has not been able to discern any relationship between any state of the tongue and dyspepsia, or ulcer of the stomach, apart from that which might occur from loss of appetite or restriction in the amount of food. With regard to the bowels, some forms of constipation are often connected with changes in the tongue, but such connection is not constant. The author rather thinks it to have been a coincidence, and cannot even point to the diagnostic significance of the tongue in obstruction. The state of the tongue in the latter condition is dependent not upon the intestinal

lesion but upon the constitutional disturbance. A dry tongue is well known to occur in acute obstruction, due to deficiency of salivary secretion. In chronic obstruction, unless, however, there is constitutional disturbance, the tongue will not change. In diarrhœa all conditions of dryness, furring, and incrustation are observed. The absence of saliva, dehydration, and pyrexia help the desiccation. In diarrhœa and dysentery, therefore, the change in the appearance of the tongue is more marked than in any other disease.

Other Diseases. As regards the relation of the tongue to other individual diseases but little can be said. Of more direct association, we have the cyanotic tongue in heart disease; the dry tongue in chronic albuminuria and diabetes mellitus; the strawberry-tongue of scarlet fever; and the dry brown tongue of typhoid fever. Of course, the so-called typhoid tongue represents but one stage of typhoid fever. Throughout the disease it may present all varieties in direct succession, from the stippled, the coated, the plastered, the furred, to the incrusted. In lobar pneumonia the same changes occur as the disease advances. In bronchitis the lower degrees of coating are presented, while in rheumatism the variety is considerable. In conclusion, it may be stated that the tongue seldom points to solitary organs or isolated disorders, but is a gauge of the effects of disease upon the system.

The Tongue in Prognosis and Treatment. Clinical observers agree with Dickinson, that the condition of the tongue is due very largely to the four states with which he has associated it—dehydration, exhaustion, pyrexia, and local conditions about the mouth. As these conditions modify the state of the tongue, it is evident that the first sign of improvement, as return of moisture, denotes a diminution in temperature. Its appearance is, therefore, of good prognostic omen. The degree of fever, the state of the nervous system, the maintenance or abeyance of secretions, and the failure of vitality, are indicated by the condition of the tongue. The return of moisture, the removal of fur, the subsidence of tremor, at once indicate that the patient is getting better. The persistence and increase of these signs show that the disease is getting the better of the patient. As to indications for treatment, the dryness, furring, and incrustation are connected with the want of saliva. The processes by which this want is brought about differ. They have previously been referred to, and the indications for treatment are obvious. One can infer from the state of the saliva the condition of the intestinal canal, a matter of the highest importance practically. There is no doubt that, except possibly in diabetes, when there is diminished saliva, there is also diminished gastro-intestinal secretion. Such diminution is followed by loss of appetite and impairment of digestion. The indication is at once to administer material that is digested with the least difficulty. Hence, liquid food and stimulants are to be used. The dry and bare tongue is of serious prognostic omen in all conditions. While it may be due to want of saliva alone, it also occurs as a part of the failure of nutrition in hectic fever, suppuration, and other conditions. It is an indication for the use of tonics, stimulants, and liquid and highly nutritious food. The weak pulse does not more surely tell of an asthenic tendency than the red, dry, and polished tongue.

Movements of the Tongue When the patient is asked to put out his tongue it is done without other movement than that required for its ejection. Interference with its motility occurs in disease, when the projection is attended by abnormal movement. It may be tremulous, as in alcoholism or in simple weakness alone. It may be slow or impeded in the various stages of paralyses. It is tremulous and the seat of fibrillar contractions in general paralysis. It cannot be projected at all in glosso-labial paralysis; it can be projected, but with difficulty, and may have to be aided by the finger, in general paralysis and diphtheritic paralysis, progressive muscular atrophy, and hemiplegia, because the paralysis is only partial. The tongue points to the paralyzed side of the body in hemiplegia when the face is involved.

Angina Ludovici. Angina Ludovici is characterized by slight inflammatory congestion of the throat out of proportion to the symptoms of the inflammation in the external structures. Woodeny induration of the connective tissue, which will not pit on pressure; spreading of this induration, which is circumscribed, so that it is bound sharply by unaffected cellular tissue, is characteristic. The induration may extend from the rami of the jaws to the face. With this there is a hard swelling in the *tongue* and along the lower jaw, causing thickening of the floor of the mouth. This is observed by palpation with the finger in the mouth. The glands are not affected. For a long time the nature of this affection was not known. It is now believed to be due to actinomyces. (See Parker, *Lancet*, 1879, and Anderson, *Transactions of the Medico-Chirurgical Society*, 1891.)

The Fauces and Pharynx

The passageway between the mouth and the respiratory passages is lined with mucous membrane, which is subject to diseases to which they are liable. The symptoms thereof are similar to the symptoms of mucous membrane inflammation elsewhere. The large muscles of the pharynx which aid in deglutition are subject to affections which belong to muscular tissue generally, hence *rheumatic inflammation* and loss of power of muscle, or *paralysis* occurs. *Paralysis of the pharynx* has not the same practical importance in diagnosis of central lesions as paralysis of other structures, such as parts of the larynx. This is due to the fact that the nerve-supply of the pharynx is derived from a nerve (glosso-pharyngeal) which supplies other structures, paralysis of which is more evident than pharyngeal paralysis, more readily ascertained, and which causes more pronounced symptoms. (See Cerebral Nerves.) From its exposed situation the pharynx is particularly liable to *infection* from micro-organisms. The infection may extend from the mouth, or from the nares above, or the micro-organisms may affect it primarily.

The fauces and pharynx may be the seat of morbid processes which occur secondarily to diseases in other portions of the body with a moderate degree of frequency. Inflammations of the mucous membrane of the pharynx are of rheumatic or gouty origin in a large number of cases. Indeed, gouty inflammation of the pharynx seems to be more common than gouty inflammations of mucous membranes in other

situations. The large majority of subacute or chronic pharyngeal inflammations are secondary to dyspepsia. They also occur from extension of the disease from cavities related to the pharynx.

Affections of the tonsils are usually more common in rheumatic states, and bear some relationship to the rheumatic diathesis. Inflammation of the tonsils may follow acute rheumatism or may alternate with it. A patient who is predisposed to rheumatism may at one season have tonsillar inflammation, at another rheumatism. The writer has seen tonsillitis immediately followed by rheumatism, and then the latter replaced by the former.

Apart from what has just been said, diseases of the pharynx bear but little, if any, diagnostic relationship to disease elsewhere. While there may be cyanosis of the mucous membrane, or tuberculous ulceration, or other changes which we have noted, the signs of the primary disease are so much more marked that we need not rely upon the appearance of the pharynx or symptoms of pharyngeal disease for diagnostic purposes. The only general affection which may be diagnosticated from the appearance of the pharynx alone is measles. In obscure cases of sudden fever, with nasal catarrh, the appearance of the eruption in the situation just indicated may lead to the recognition of measles when the external eruption is not apparent. For the purposes of the therapeutist it should be borne in mind that symptoms referable to the pharynx are very frequently due to disease in the nares, particularly in that portion of the pharynx which is not open to direct inspection—the nasopharynx.

The *general symptoms* of pharyngeal disease are not marked, except in diphtheria, in erysipelas, in retropharyngeal abscess, and in affections of the tonsils. In the latter the general symptoms appear to be out of proportion to the local process. The high fever, the intense headache and backache, and rapid pulse, seem to point to a process which in extent and severity should far surpass that which occurs in the tonsils.

As a passageway or channel, affections of the pharynx are liable to obstruct it, causing symptoms of *occlusion*. As a channel for the passage of air, obstruction in the pharynx will lead to *dyspnœa*. In addition to its function as a simple channel, the pharynx is concerned in the act of deglutition. When, therefore, there is obstruction of the pharynx, *deglutition* is made difficult, or may even become impossible.

Attention cannot be too strongly directed to the investigation of the *nasopharynx* in children who are poorly developed physically and mentally, and who present appearances that, to the practised eye, are most familiar. The experienced observer will at once judge, and judge correctly, that this combination of symptoms is due to disease in the nasopharynx. Reference must be made to the remarks on adenoid vegetations of the nasopharynx, but it is proper to state here the relationship and the importance of investigating the structures in the class of cases just indicated.

The Data Obtained by Inquiry.

Pain. In affections of the fauces and pharynx pain is one of the most common subjective symptoms. It is due to the fact that the

functional acts of the pharynx require movement of all the structures. When they are the seat of inflammation, or ulceration, the movement excites pain. It is, therefore, a symptom of great severity in inflammation of the tonsils and pharynx, of rheumatism of the muscular structure of the pharynx, and of tuberculous and cancerous ulceration. Pain in the pharynx is a frequent accompaniment of post-nasal inflammations, although the pharynx itself is not affected.

Dryness. Dryness of the fauces, with a tickling sensation and a more or less constant desire to hawk, occurs in pharyngitis. Hawking, however, is not a symptom of disease of the pharynx alone. It may also be due to disease in the posterior nares.

The Odor of the Breath. In follicular tonsillitis the breath has a peculiar odor. This is more marked in the milder forms of inflammation, with retention of the secretion of the glands. The odor is intense and fœtid. In cancer and syphilis there is also fœtor of the breath. The fœtor may be of diagnostic significance in distinguishing cancer from tuberculosis.

Dysphagia. The symptom varies in degree from slight difficulty in swallowing to complete prevention of the act. Any disease which occludes the passageway causes dysphagia; pain is also a cause. It is, therefore, present in all painful affections of the pharynx. *Dyspnœa* is seen in tumors, in inflammation of the tonsils, in the rare form of erysipelas of the pharynx, and in retropharyngeal abscess. It occurs from occlusion of the passages, and is more marked in retropharyngeal abscess and erysipelas than in other conditions. In certain forms of abscess of the tonsils it may be very extreme.

Spasm of the pharynx is a subjective symptom complained of in some cases of pharyngitis. The degree of spasm or the amount of choking sensation is largely dependent upon the neurotic constitution of the individual. It may be extreme when only a moderate amount of inflammation is present. It is seen in the most aggravated form in hydrophobia.

The Data Obtained by Observation.

Examination of the Fauces. Method. For this purpose examination is made by the unaided eye, illuminating the throat as in the examination of the larynx. The difficulties of examination arise from the tongue and the uvula. The mouth should be opened as wide as is consistent with comfort and in an unrestrained manner. The tongue is pressed out of the way by the use of a tongue-depressor. In many cases, however, even with the tongue-depressor, the tongue muscles will contract and the organ bunch up in the mouth. Moderate, quiet, full breathing, gently opening the mouth as the deeper inspirations are made, causes the tongue to relax and lie in the bottom of the mouth, and at the same time elevates the uvula. At the time of a full breath the part may be inspected throughout. Sometimes the fauces can be examined if the tongue is protruded and held with a soft napkin between the finger and thumb by the patient. In the fauces the tonsils and uvula are to be observed, following out the routine method of ascer-

taining all facts. Attention is then paid to the posterior wall of the pharynx, with the same object in view.

Inspection. In examining the fauces and pharynx observation is made of the *color* of the parts, the appearance of the mucous membrane and its glands, the *appearance* and *position* of the uvula, the size of the tonsils, the character of the secretions on the pharynx, and the presence or absence of swellings and abnormal exudations.

Color. The color of the mucous membrane is generally dark red. In the acute forms of pharyngitis the color is bright red. In cases of heart disease, when there is cyanosis, the veins are congested and the surface dusky. In obstruction of the superior vena cava by tumor there is a cyanotic hue of the surface of the pharynx.

Appearance of Surface. The capillary vessels may pulsate in aortic regurgitation. Bleeding-points may be seen over the surface of the pharynx, the discharges of blood from which may simulate pulmonary hemorrhage. The blood may be swallowed and then vomited, and hence gastric hemorrhage is simulated. When the hemorrhage occurs at night it is seen on the pillow as yellowish stains. It is often due to adenoid vegetations in the nasopharynx. In chronic pharyngitis the membrane is dry, the glands are prominent, and the secretion viscid.

On examination of the posterior wall of the healthy pharynx little elevations due to glands are seen upon its surface, and moderate-sized vessels are seen coursing through the mucous membrane.

Eruptions. Eruptions may be observed in the pharynx in some of the specific fevers. Thus, in measles, the appearance of the rash on the pharynx and on the soft palate may be observed before the development of the rash on the skin. The eruption of scarlatina is also seen in the pharynx, and the papules and pustules of variola are frequently observed in that situation.

Ulceration. *Follicular Ulceration.* Small superficial ulcers corresponding to the follicles may be seen over the posterior wall of the pharynx. They occur in chronic catarrh, and are due to inflammation of the follicles. In addition, ulcers secondary to infectious processes are sometimes seen, as in *typhoid fever*. In *syphilis*, in the secondary stage, small, shallow ulcers are seen on the posterior wall of the pharynx. They do not cause pain. Mucous patches are observed at the same time, not only on the pharynx, but also in the mouth. In the tertiary stage deep ulcers, followed by scars, are seen on the posterior wall of the pharynx. Although the absence of pain renders it probable that they are of syphilitic origin, nevertheless the history of infection and of the primary lesion, and the evidence of the disease in other structures, ought to be secured before a diagnosis is fully established. In the tertiary forms it may be necessary to resort to the therapeutic test. (See The Infections—Syphilis.)

Tuberculous ulcers are irregular in shape, and the floor grayish. They are seen in tuberculosis in its later stages. They are the source of extreme pain. There is usually ulceration in the larynx at the same time, and, in extremely rare cases, tuberculous ulceration of the tonsils. In tuberculous ulceration, after the application of cocaine, a portion may be scraped off and examined microscopically for tubercle bacilli.

Cancer of the pharynx is rare, and is usually secondary, the disease having spread from other situations.

Exudations. On the pharynx the exudation may be due to diphtheria, to pseudodiphtheria, or to thrush. The method of distinguishing the various forms will be considered in the articles on the respective affections. In diphtheria the membrane is made up of fibrin arranged in a network, in the meshes of which epithelium, blood-corpuscles and pus-corpuscles and micro-organisms are found. When removed, hemorrhagic abrasions and raw purulent inflammatory areas remain. Two forms of bacilli are found in the membrane—the pseudodiphtheritic bacillus and the true, or Klebs-Löffler bacillus. (See Bacteriology.) The Löffler bacillus is best detected by cultivations. After the membrane is removed and washed in a 2 per cent. solution of boric acid, it is cultivated in blood-serum. The pseudodiphtheritic bacillus likewise grows, but its appearances are different.

Anæsthesia. Some of the results of inspection may be confirmed by means of the probe, and alterations in the sensibility of the pharynx may be detected. Sensations may be absent in the whole posterior wall of the pharynx. Loss of sensation may occur in hysteria, in bulbar paralysis, and in diphtheritic paralysis. On the other hand, there may be an apparent *hyperæsthesia*. In some individuals the pharynx is particularly sensitive to the presence of foreign bodies, such as inflammatory exudates, and may resent their presence by sudden coughing and retching. Inflammations increase the hyperæsthesia of the pharynx. The condition is sometimes observed in hysteria.

The Uvula. In health it hangs midway from the palate. It varies in shape from congenital causes, and may be elongated, on account of disease. This takes place particularly if there has been hawking or coughing, on account of chronic nasal catarrh. When elongated it is pointed and may extend almost to the base of the tongue. The uvula may be swollen and œdematous. The œdema is usually associated with subcutaneous œdema in acute Bright's disease. It may occur in debility. In both conditions it may become so enlarged as to interfere with swallowing and breathing. In some cases of pharyngitis the uvula is the seat of intense inflammation and great œdema. In addition to the constant cough which it causes there may be dyspnœa and repeated attacks of choking.

Hemorrhagic infarcts may take place in the uvula. In two instances under the writer's care the intense infarction led to sloughing, and in one the uvula was swallowed.

The Cervical Glands. The pharynx is in such intimate relation with the large lymphatic glands in the neck that diseases of the former are frequently attended by enlargement of the latter. The glands at the angle of the jaw are increased in size. The glands extending along the vessels of the neck may also be enlarged. In cases, therefore, of enlargement of the glands in this situation, it is absolutely essential to examine the fauces and pharynx.

The Tonsils. The tonsils are situated at the sides of the pharynx, between the anterior and posterior folds of the palate. They are small bodies, not larger than a filbert in the adult. Their entire

surface can be seen by ordinary inspection. If enlarged, the posterior surface cannot be seen, although a larger view may be obtained by causing the patient to gag or retch, during which they are brought forward to the light. They are pathologically of much importance. They are made up of glandular structure arranged in follicles and held together by connective tissue. The crypts of the follicles open on the surface, and in disease are visible. The diseases of the tonsils have nothing to do with their function as far as known. The tissue and gland follicles are liable to inflammations, which may be bacterial or may be the result of rheumatism. The tonsils become enlarged; the swelling takes place rapidly in the acute forms. They may be simply enlarged and the covering membrane intensely red. In other forms of inflammation the surface may be dotted over with white points, due to exudation from the follicles; these may be covered with a white or grayish membrane, which is removed with difficulty, leaving an abraded surface beneath. Repeated attacks of inflammation cause chronic enlargement of the tonsils. They are enlarged sometimes to a great degree, filling almost entirely the lumen of the fauces. The surface is irregular, and may be scarred. The mouths of the follicles may be dilated. By virtue of their position, enlarged tonsils from any cause are a source of *dyspnœa* and *dysphagia*. The tonsils may be the seat of sarcoma and tuberculosis.

Ulcers. Tuberculous ulceration is rare. In a patient, a lad of sixteen years, under the writer's care, the large tonsils were of a honeycombed appearance, on account of the grayish, irregular ulceration. Deglutition was absolutely impossible, on account of pain, and the young man died of starvation.

Exudations on the tonsils are due to inflammation of the follicles, to diphtheria, to the pseudodiphtheritic inflammation which attends scarlatina, or which arises secondarily to other infectious debilitating diseases, and to thrush.

Leptothrix of the Tonsils. In healthy persons the plugs which block the tonsillar crypts are found to be made up of cells and segmented fungi. The latter stain bluish-red with iodo-potassic iodide solution. Sometimes the micro-organisms extend beyond the follicles, covering the surface of the tonsils with patches of various size. They are thus seen in follicular tonsillitis.

Tonsillitis. Acute inflammation of the tonsils may affect the follicles, to which form the term *follicular tonsillitis* is applied, or it may be limited to the mucous membrane, when it is known as *catarrhal* or *erythematous tonsillitis*. If with the catarrhal inflammations vesicles appear on the surface of the mucous membrane, the term *herpetic tonsillitis* is used. When the inflammation extends to the stroma of the glands it goes on to suppuration. It is characteristic of all forms of acute tonsillitis to recur frequently in the same subject. The relationship to rheumatism has been spoken of. This relationship applies to both the acute and the suppurative forms. The various forms of tonsillitis occur at any age, although it is least common under ten years of age; the suppurative form occurs most frequently in adolescence. Tonsillitis occurs in both sexes. It may follow exposure to

wet and cold, although patients who are subject to the attacks bear exposure, unless they are at the same time unduly fatigued. The *follicular* form of tonsillitis is apparently associated with bad drainage or other unhygienic conditions, which makes it possible that noxious emanations act as an exciting cause. Several persons of the same family may be affected at one time, so that it is often difficult to distinguish the cases from diphtheria. The disease, however, is not contagious. Persons brought in contact with the family, but who do not reside in the same house, escape the disease. This applies as well to children, who would, if the cases were diphtheritic, be most liable to become infected. The disease occurs more commonly in the spring than in any other season of the year, more especially in cold and wet seasons.

Symptoms. In *follicular tonsillitis*, with or without a rigor, but always with chilly sensations, the temperature rises rapidly to a great height. The subjective sensation of fever is very quickly noticeable to the patient, and is generally more pronounced than in other affections. With the chill and during the rise of temperature there are some frontal headache and severe pain in the back and limbs. The pain in the back is most excruciating. In a short time the patient complains of pain in the throat. Swallowing is difficult, and there is a sense of fulness. The throat is dry and burning. On examination the tonsils are found to be swollen, and a yellowish-white exudation is seen on the crypts. In twenty-four hours the points may coalesce to form a patch. The glands expand slightly, and may extend only slightly beyond the arches, or, in younger subjects, one-quarter of the way into the lumen of the fauces. Sometimes one gland is affected before the other. The difficulty in deglutition increases and the voice becomes nasal. There is usually some enlargement of the cervical glands. The general symptoms continue for forty-eight hours, the temperature remains at 105°, and the pulse is very rapid. After the first twenty-four hours the pain in the back lessens. The tongue is coated and the breath heavy. The urine is loaded with urates. At the end of the fifth day the fever, which subsides gradually, has disappeared. The local symptoms, however, may remain longer; that is, the tonsils are still enlarged and the exudation disappears slowly. Sometimes the prostration and general symptoms are very severe, so that after the fever has subsided convalescence may be very slow.

Albuminuria, due in all probability to the fever, frequently occurs; in some cases, undoubtedly, acute nephritis attends the attack and retards the convalescence. In a case under the writer's care the patient first had acute rheumatism; this was replaced by a severe attack of tonsillitis, during which albumin, blood, and granular casts were found in the urine. The swelling of the tonsils subsided in due course, but the Bright's disease continued for a long period, finally ending, however, in complete recovery.

In *herpetic tonsillitis* the severe pain and intense general symptoms are out of proportion to the local lesion.

In *suppurative tonsillitis* the constitutional disturbance is also very great. The temperature rises high, 104° to 105°, and the pulse is

very rapid, from 110 to 130 in the adult. The inflammation usually begins in one tonsil, and the other may be involved later. The tonsils at first are enlarged and firm and very red. There is swelling of the surrounding tissues. In twenty-four hours deglutition becomes almost impossible, and there is salivation. At the end of forty-eight hours the patient presents a striking appearance. The glands of the neck are enlarged, the patient is unable to open his mouth, the voice is nasal or almost suppressed; there is dribbling of saliva from the mouth. The face may have a dusky hue in spite of the capillary congestion due to the fever. There is constant desire to discharge saliva and accumulated secretions from the back part of the mouth. The patient cannot lie down. The pain is extreme, and is aggravated by swallowing. It is sometimes of a throbbing character, and often shoots to the ears. Indeed, earache may be the chief complaint. The patient does not take food, and exhaustion soon ensues. During the twenty-four hours before rupture takes place the previously reddened face becomes blanched from exhaustion. The fever is continuous during this time, with great rapidity of the pulse. The patient may be delirious. Sometimes the delirium is marked and the patient resists efforts to keep him in bed.

The suffering is out of proportion to the danger of the case. About the fourth or fifth day suppuration is over, and if the finger can be inserted into the mouth between the almost closed teeth, fluctuation is detected. In cases in which the mouth is opened a little more freely, in addition to the swelling of the tonsils below the arches, marked swelling and projection forward of the half-arches may be seen. The fluctuation may be detected through the anterior fold of the palate, and, if lancing is to be performed, the pus can only be reached through this structure. In short, a peritonsillitis takes place. After spontaneous rupture, which usually takes place into the mouth, instant relief is experienced. Rupture may take place into the pharynx and cause suffocation from entrance of pus into the larynx. In rare cases it has opened into the carotid artery, causing instant death from hemorrhage.

Diagnosis. The diagnostic features of acute tonsillitis are the sudden high fever, severe backache and headache, pain in the throat, and albuminuria. The characteristic appearance of the face, the salivation and pain, with suppressed voice and difficult deglutition, should distinguish it from trismus or *tetanus*. In both the jaws are closed. It must not be confounded with *smallpox*, which it resembles during the first twenty-four hours.

Cases of follicular tonsillitis are frequently mistaken for *diphtheria*. The follicular inflammation in tonsillitis is limited to the gland, on which patches of a yellowish-gray color, easily removed without leaving bleeding surfaces, are seen. In diphtheria the membrane is of an ashy-gray color, not in points or small patches, or separated by red tonsillar tissue; it extends to the pillars of the fauces, and may appear on the uvula. There are, nevertheless, many cases which are doubtful, when a bacteriological diagnosis must be resorted to. (See Bacteriological Examination.) A history of exposure sometimes helps us to arrive at a

conclusion. The cases that particularly increase our anxiety are those of adults who are subject to attacks of follicular tonsillitis. In the grave and extensive forms of diphtheria with asthenic symptoms (septicæmia) the diagnosis is not difficult.

Enlargement of the Tonsils. Chronic Tonsillitis. The tonsils may be enlarged, on account of repeated attacks of acute inflammation or from chronic inflammation. They do not appear to cause serious symptoms unless associated with adenoid vegetations in the nasopharynx. They may interfere with hearing, however, and with breathing, and cause snoring at night. Fœtor of the breath may be noted, particularly if the secretion lodges in the crypts. The latter may be recognized by its characteristic yellowish color and by its odor on removal. The enlarged tonsils are irregular in contour.

Foreign bodies in the tonsils are not of common occurrence. They give rise to local symptoms, as the sensation of the presence of a mass causing repeated efforts at swallowing. If calculi are present the patient may complain of a rough sensation. The calculi follow frequent attacks of quinsy. *Hydatids* are sometimes located in the tonsils.

Adenoid Vegetations of the Nasopharynx. Adenoid vegetations cause more or less obstruction in the nasopharynx. The symptoms may be classed as primary and secondary. The former are local, and due to the foreign substance, *per se;* the latter are local and general. The former are catarrhal; the latter the result of stenosis.

Local Symptoms. In a large number of cases there is *discharge* from the nose. This may be mucopurulent, or be associated with crusts. If the discharge is not constant, the child is subject to coryza, with its customary discharge, on the slightest provocation. With or without the chronic purulent nasal discharge mucus and blood may be passed at night and found on the pillow in the morning.

The *hearing* is frequently impaired. There may be simply dulness of hearing, or it may amount to marked deafness, either because of pressure of the adenoid vegetations, or extension of secondary inflammation to the Eustachian tubes. The senses of *taste* and *smell* are often much impaired. There is increase in the secretion of pharyngeal mucus, which in older persons causes difficult expectoration.

Rhinoscopic Examination. The roof of the pharynx is covered with rounded or villous projections, often concealing the posterior nares. Rarely the villi may be seen projecting below the soft palate. In children the examination is difficult, and hence digital exploration must be used under an anæsthetic. The finger readily detects the masses, which sometimes are soft, at other times tough and of fibrous or cartilaginous consistency.

The Appearance. The *expression of the face* is characteristic. It is dull and stupid, and may be drawn. (Fig. 187.) The *mouth* is kept open in breathing. The lips are dry, and may be cracked. They are thickened. The palatal arch is high and narrowed.

The *nostrils* are flattened laterally. Rarely they may be depressed. In one instance, which the writer saw with Dr. Harrison Allen, the exterior of the nose suggested inherited syphilis, all the more because

of our knowledge of the possible presence of the disease. There were no other evidences of hereditary syphilis in the child or in any member of the family.

The Voice. It is thick and muffled, becoming indistinct upon the occurrence of slight cold.

The Chest. While there is a general lack of physical development, the appearance of the chest is most striking. The cases have been frequently mistaken for rickets, however; in this country adenoid vegetations are a common cause of chest-deformity, whereas in England and on the continent rickets is the most frequent cause. The ribs are prominent in front, the sternum is angulated forward at the manubrio-gladiolar junction and grooved at the gladiolar-xiphoid junction. A saucer-shaped depression is found at the lower costal cartilages. The ribs behind are closely compressed, so that the intercostal spaces at the lower part of the chest are obliterated. The chicken-breast appearance is most striking, with the depression in the lower portions of the chest. The diaphragm may be drawn in during inspiration in the middle and lateral thoracic regions.

FIG. 187.

Appearance in adenoid disease. (DAWSON-WILLIAMS.)

In addition to the "chicken" or "pigeon-breast" the more advanced deformity known as the "funnel-breast" or *trichterbrust* is seen. In children who suffer from asthma and bronchitis, the chest becomes emphysematous.

MENTAL AND NERVOUS SYMPTOMS. Headache, listlessness, and indisposition for mental exertion are marked. The patients are usually backward in their studies and are unable to fix their attention for any length of time upon any subject. The child is forgetful and cannot study without effort. *Aprosexia* is the term applied to this condition.

Choreiform *spasm* of the face occurs in connection with it. *Enuresis* is a frequent associate symptom. The child is subject to frequent attacks of *indigestion.* I have seen the following occur in many cases: Prior to operation the child had an abnormally poor appetite and was subject to frequent attacks of indigestion, characterized by vomiting, with fever. After the operation the appetite improved and continued good, and the attacks of indigestion disappeared entirely. The cases had been under observation before and after the operation for a number of years.

The indigestion seems to have been due to the fact that, owing to the obstruction, the child would have to eat rapidly, in order to keep the lumen of the mouth free for breathing purposes. The rapid eating, of course, prevented proper mouth-digestion, and hence the occurrence of gastric catarrh.

Symptoms from Embarrassed Respiration. In addition to mouth-breathing, the patient snores at night, and sleep is always disturbed. The respirations are irregular, with a pause between, followed by noisy inspirations. The difficulty of breathing is the cause of restlessness, and the child will often wake up in the night with dyspnœa. *Night-restlessness*, with *dyspnœa* and irregular respirations, should point, therefore, to obstruction in the nasopharynx.

Diagnosis is based upon the facies, which is very characteristic, and the physical examination. In children, digital examination is necessary. The finger can readily detect small, flat bodies or grape-like masses in the nasopharynx.

The student cannot become too familiar with the symptoms and signs of adenoid disease of the nasopharynx. There is no doubt that in our large cities this local affection is of more common occurrence and more disastrous in its results than any other that we have to deal with in children. It may be said that in children in poor health, anæmic, with impaired digestion, and lack of muscular and physical development, if the causes are not due to impure air and improper diet, or to improper sanitation generally, it is almost certain that there is disease of the nasopharynx. The writer has seen a very large number of cases in recent years in his practice, and has had the satisfaction of seeing the entire picture of the child change after proper operations. It may be said in passing that this change does not take place at once, but after three to twelve months the child will be fully restored in physique, if during that time attention is paid to proper exercise and the development of the chest. Notwithstanding all this, however, the natural shape of the chest and appearance of the face are only resumed gradually.

Inflammations of the Pharynx. Inflammation of the pharynx, *acute pharyngitis*, or sore-throat, follows cold or exposure, particularly after the patients have been physically depressed. The acute inflammation may be associated with rheumatism or gout. The inflammation often involves the tonsils as well as the pharynx. The symptoms are pain on swallowing, with dryness and a constant desire to hawk and cough, on account of the tickling sensation. There may be slight laryngitis and inflammation of the Eustachian tubes, with deafness. Stiffness of the neck and enlargement of the cervical glands attend the local inflammation. The general symptoms are not marked. The attack is ushered in by chilliness and slight fever. On examination the mucous membrane is seen to be congested, dry, and glistening, and covered in spots with sticky secretions. The uvula may be very much swollen. When the submucous tissues are involved the parts are more swollen and there is greater dyspnœa. The dysphagia is more marked, although the pain is not any greater. The fever is higher. The larynx is always involved, causing aphonia.

Phlegmonous Inflammation. A diffused inflammation of this character occurs. The writer saw one case with dyspnœa, nervous symptoms, and high temperature, simulating severe pneumonia. Pneumonia was thought to be present because there were congestion and œdema of the lungs. It occurred during the prevalence of the recent epidemic of influenza. The disease began in the pharynx; the tissues were swollen and infiltrated. The early symptoms were pharyngeal. The dysphagia was extreme, and there was an abundant mucopurulent expectoration, which did not contain pneumococci. Death took place on the ninth day from exhaustion. The autopsy showed a high degree of congestion of the lungs, and phlegmonous inflammation of the pharynx, larynx, and trachea. While, therefore, the recognition of an acute phlegmonous inflammation is not difficult, it must not be forgotten that it is a grave disease, which may present such marked pulmonary and systemic symptoms as to lead to the suspicion of pneumonia.

Angina Ludovici is an inflammation of the cellular tissue of the floor of the mouth and neck. It is probably a form of actinomycosis. The swelling is most marked below the jaw of one side. The symptoms are very intense and both local and general. There are general septic symptoms from the outset. With the swelling there are œdema and board-like induration. Redness and the rapid formation of an abscess occur rarely. The throat is not affected. Death takes place from reflex suffocation or in coma. (See The Mouth.)

Rheumatic pharyngitis is of short duration, without objective symptoms. Pain is intense, deglutition difficult. The usual concomitants of rheumatism are present. It frequently gives place to torticollis, lumbago, or rheumatism in some other situation.

Chronic pharyngitis follows acute attacks, and is a frequent accompaniment of nasal catarrh. It is common in smokers and alcoholic subjects; the use of the voice in loud tones, as by clergymen, auctioneers, etc., is also a cause. It is a frequent attendant upon indigestion, due probably to the eructations. The objective signs are relaxation of the mucous membrane, with dilatation of the veins. The membrane is covered with a thick secretion, which is dry and glistening. In the granular form the wall of the pharynx is covered with millet-seed projections and is congested. Tough mucus is seen in small areas.

Retropharyngeal Abscess. The inflammation may begin in the submucous connective tissue, and a retropharyngeal abscess form. There are high fever and dysphagia, with stiffness of the neck and enlarged glands. On examination a projection into the pharynx can be seen or distinctly felt on the posterior wall. The disease may be difficult of recognition in infants, in whom it is not possible to get a good view of the pharynx. On the other hand, it may be simulated by disease of the cervical vertebræ, in which there may be stiffness, difficulty in deglutition, and possibly a tumor. It must not be forgotten that retropharyngeal abscess may result from caries of the cervical vertebræ. In children the abscess is attended with dyspnœa and alteration in the voice, so that laryngeal disease may be suspected. I

recall a case of retropharyngeal abscess in which the dyspnœa was so severe as to suggest croup; in fact, preparations for tracheotomy were made, when sudden rupture of the abscess revealed the nature of the disease. Fortunately the child had been kept in the upright position, so that pus was discharged into the mouth, or suffocation would have ensued.

Inflammation of the Parotid Gland. First, specific inflammation or parotitis (see Mumps); second, symptomatic parotitis occurs in typhoid fever, pneumonia, pyæmia, and septicæmia. The process is intense, characterized by swelling, redness, and heat over the parotid gland. There are pain and difficulty of mastication; suppuration rapidly ensues in the septic form. It is thought to be an unfavorable symptom, but I have seen two cases in typhoid fever get well. In a case of septicæmia it did not advance to suppuration. Stephen Paget has described a symptomatic inflammation in disease of the abdomen and pelvis. He collected 101 cases, 50 of which were due to injury, disease, or temporary derangement of the genital organs, as by slight blows, or in females to the introduction of a pessary. It may occur before the menstrual period or during pregnancy. Septicæmia or pyæmia does not attend the process—indeed, many of the cases are afebrile. In 78 cases, 45 suppurated and 33 resolved without suppuration.

Gowers describes a case of parotitis which occurred in the course of fatal peripheral neuritis.

The Œsophagus.

The œsophagus is open to all affections which arise in mucous membranes, although its histological structure, its position, and its functions largely protect it from involvement in disease. Should morbid processes arise, the symptoms expressive of these processes are the common symptoms of disease of the mucous membrane. But the œsophagus is a closed tube, the function of which is to convey food from the pharynx to the stomach. It is subject to all the affections common to channels. Any disease of the tube interferes with its function, made evident by the symptom common to all disorders of the œsophagus—*dysphagia*. As this symptom occupies a position of such prominence in the symptomatology of disease of this tube, it is evident that the diagnosis of disease resolves itself into the differentiation of all forms of difficulty of deglutition.

Before beginning the discussion along the lines indicated, the subjective and objective symptoms of disease of the œsophagus must be considered.

The Subjective Symptoms. *Pain* is a common symptom of disease of the œsophagus. In acute inflammation it is extreme, and is complained of in the neck, between the shoulders, and along the vertebræ for a short distance. Its character depends upon the cause. Severe burning pain, often agonizing, is due to inflammation caused by hot or caustic fluids. Absence of pain after the ingestion of such substances, or its disappearance in a short time, points to extreme corrosive action and gangrene. Pain attends and is a part of the symptom

—*dysphagia* (*q. v.*). *Cough* attends such diseases of the œsophagus as exert pressure upon the bronchus, as carcinoma.

The Objective Symptoms. *Stiffness* of the neck is seen in acute inflammation of the œsophagus and in peri-œsophageal abscess; it may also occur in traumatism. The *expectoration* in diseases of the œsophagus is characteristic. It is usually a glairy mucus, often frothy or viscid. It is not coughed up, but after welling into the pharynx is hawked up. It is abundant in acute and chronic inflammation and in cancer.

Hemorrhage from the Œsophagus. Hemorrhage from the œsophagus occurs from varicosity of the veins at the lower portion of the gullet. It may occur in old people, from senile disease of the liver, kidney, and spleen, or at any age in cirrhosis of the liver. In hemorrhage from the œsophagus the blood is usually bright in color, has not been acted on by an acid, as in hæmatemesis, and is, therefore, alkaline in reaction, and is not discharged by vomiting, although vomiting may occur after the blood is poured out. In a grave case of purpura under the care of the writer hemorrhage took place from the lower end of the œsophagus. Small bleedings from the œsophagus are usually indicative of cancer, especially if, in addition to the hemorrhage, there are present the symptoms of occlusion. Hemorrhage is also seen in foreign bodies: (1) from trauma; (2) from ulceration. *Emaciation* is the most characteristic general symptom of œsophageal disease. It is, of course, more striking in cancer, but occurs to a moderate degree in all forms of stricture. *Fœtor of the breath* attends dilatation of the œsophagus.

Emphysema of the subcutaneous connective tissue should always lead to investigation of the œsophagus. Usually it is found to have been preceded by pronounced symptoms of disease of the œsophagus. In rare cases ulceration of the œsophagus may progress without symptoms, and extend into the air-passages. The passage of air through the fistulous communication causes subcutaneous emphysema. It is of frequent occurrence when foreign bodies lodge in the gullet.

Physical Examination. Examination of the œsophagus is made by inspection and auscultation, and by means of palpation with or without a bougie.

Inspection can be made only with an endoscope.

Auscultation of the œsophagus, while the patient is swallowing fluids, sometimes confirms the results obtained by instrumental palpation as to the seat of an obstruction. A gurgling sound is audible to the left of the spine as the fluid passes the obstruction.

Palpation. The œsophagus behind the trachea in the neck may be palpated when it is enlarged, as in abscess. Palpation yields the most positive results.

It must not be forgotten that the normal constriction of the œsophagus is situated nearly opposite the fourth dorsal vertebra, ten inches from the teeth. The bougie is used to determine the cause of difficulty in swallowing. If the cause is due to paralysis or to spasm of the œsophagus the bougie can usually be passed with ease. If, on the other hand, it is due to organic disease, an obstruction will be

found. In organic disease this is generally in the upper half of the œsophagus. If near the pharynx, the obstruction is due to cicatricial stricture. If the obstruction is encountered nine inches from the teeth or about the position of the bronchus, it is usually due to cancer. The bougie should not under any circumstances be passed if there are grounds for believing there is an aneurism. Fatal rupture has followed its passage under such circumstances.

Method. The patient should be seated with the head thrown back sufficiently far to make the passage from the pharynx to the œsophagus almost continuous. The operator may stand behind or in front of the patient. The bougie, held like a pen, should be passed through the pharynx, guided by the fingers, close to its posterior wall. But little force should be used. It should be passed slowly, when the gagging will soon be overcome. The bougie should be warmed and oiled before it is introduced. The handles should be flexible, the bulb olive-shaped.

Obstruction of the Œsophagus. *Dysphagia* is a symptom common to all diseases of the œsophagus. It may vary from simple pain-dysphagia to complete obstruction of the tube. Dysphagia from obstruction of the œsophagus is due (1) to disease outside of the canal (external pressure), (2) to disease of the canal itself, and (3) to the presence of a foreign body in the canal. In the consideration of this symptom, therefore, these conditions must be studied.

1. External Pressure. The œsophagus at different parts of its course is in intimate relationship with the trachea, the thyroid gland, the carotid artery, the left bronchus, the bronchial glands, the arch of the aorta, and the descending aorta. Disease of these structures attended by enlargement may, therefore, cause difficulty in swallowing. It is not likely that difficulty of deglutition from disease of the trachea, thyroid gland, or carotid arteries will be overlooked. If the trachea is affected, dyspnœa will be a prominent symptom; if the thyroid gland, dyspnœa will be associated with dysphagia, and the enlarged gland will be visible from the outside. Disease of the vertebræ is not likely to cause obstruction of the œsophagus, for it would not press that organ against any other solid structure. Disease of other structures, however, may cause difficulty of deglutition by pressing the œsophagus against the vertebræ. Within the thorax, disease of the mediastinal glands, aneurism of the arch, or descending portion of the aorta, an enlarged left auricle, a pericardial effusion or disease of the left bronchus might cause constriction of the œsophagus. The *mediastinal glands* are enlarged from tuberculosis, carcinoma, sarcoma, or syphilitic disease. The occurrence of physical signs of a mediastinal tumor, with a history of syphilis or the general symptoms of tuberculosis, sarcoma, or carcinoma, would point to the presence of these affections. In *aneurism* of the aorta, in its arch or transverse portion, the physical signs and subjective symptoms of aneurism—with accentuation of the aortic second sound and the presence of atheroma—would lend color to the view that the obstruction was of this nature. In both instances just mentioned the obstruction rarely goes to the extent of preventing the passage of liquids. In enlargement of the *left auri-*

cle and in *pericardial effusion* the degree of difficulty may amount simply to a temporary sense of obstruction or pain about the point where food passes these structures. If the early physical signs are associated with an enlarged auricle, with mitral stenosis, or with pericardial effusion, the diagnosis of the causal condition is easy. It is particularly important, in considering difficulty of deglutition from external pressure, to remember that the œsophagus is in close relation with the bronchus on the left side, at about the fourth dorsal vertebra —ten inches from the teeth—in case it is desirable to investigate the obstruction with a probe. Obstruction from aneurism of the descending portion of the arch of the aorta is also located at the upper portion of the œsophagus, nine inches from the incisor teeth.

2. Organic Disease. Difficulty of deglutition, due to disease of the œsophagus itself, occurs in acute inflammation, in chronic inflammation, and in stricture, which is always the result of traumatic inflammation, syphilis, or cancer.

Acute inflammation is recognized by severe pain on swallowing. It is associated with the sensation of a foreign body in the lower portion of the throat. There may be tenderness on pressure along the course of the pharynx. The pain is aggravated by speaking. The pain may extend along the vertebral column to the cardiac end of the stomach, and is usually of a burning or raw character. When the inflammation is due to traumatism, as the swallowing of acids or other caustics, the mouth and pharynx show the effects of the inflammation, and, in addition, there is agonizing, burning pain at the root of the neck and between the shoulders. The inflammation is usually attended by erosion of the mucous membrane, and hence not only frothy mucus of a glairy character is expectorated, but also blood and shreds of membrane. The effect of the corrosive poisoning on the general system is marked. There is great prostration. Because of the accompanying gastritis there is intense thirst. Acute inflammation of the œsophagus may end in ulceration or in resolution. The traumatic inflammation is followed by chronic inflammation, which ultimately results in stricture.

Chronic inflammation is attended by pain in the act of swallowing; liquids are swallowed readily, but solids with great difficulty. Viscid mucus is expectorated, usually in large amounts.

Abscess of the Œsophagus. The acute inflammation may terminate in abscess. The abscess usually develops slowly, with pain on swallowing and on movements of the neck. When the abscess is high up in the gullet it may present on the exterior of the neck. If it is situated outside of the œsophagus, and is secondary to disease of the vertebræ, it is slow and chronic in its course; fever and rigors attend its development.

Stricture of the œsophagus due to the healing of ulcers, following traumatic inflammation, is recognized, first, by the gradual development of the symptoms, by the painless nature of the obstruction in the large majority of cases, and by its seat. It is readily found by the use of a bougie; the patient can sometimes localize the area in the upper portion of the œsophagus. The difficulty of deglutition continues over such a long period of time that the nutrition is but slowly interfered

with, but gradual emaciation with coincident anæmia develops eventually.

Carcinoma of the Œsophagus. In cancer of the œsophagus dysphagia is the most prominent symptom. It comes on gradually. The patient expectorates a considerable quantity of frothy mucus, often containing blood, and revealing, on careful examination, cancerous tissue at times. Pain is not generally very severe. Cough is usually present, due to pressure of the cancerous mass on the recurrent laryngeal or pneumogastric nerve. Sometimes the cancer develops in the anterior wall, and ulcerates into the trachea or bronchus. When this complication takes place the cough is violent. Dyspnœa from pressure is likely to occur. Perforation of the œsophagus into the air-passages is followed by pulmonary abscess or gangrene, or the sudden appearance of dyspnœa, and shortly the onset of aspiration pneumonia. When ulceration causes a pulmonary œsophageal fistula the condition may simulate that of phthisis.

The difficulty of deglutition due to cancer must be distinguished from that of traumatic or syphilitic stricture and from spasmodic stricture and paralysis of the œsophagus. The history of the case aids in the recognition of traumatic or syphilitic stricture, while the ready passage of a bougie indicates that the difficulty is spasm or paralysis. Cancer usually occurs late in life and is attended with rapid emaciation. Its complications, more common than in other obstructions, are attended with fever and rapid prostration. Cancer may be distinguished from disease outside of the œsophagus by the condition of the stomach beyond the point of stricture. If there is cancer, atrophy is more likely to take place, the change in size being recognized by a tube or by inflating the stomach with air or fluids.

Sarcoma of the œsophagus is very rare. It occurs most frequently in males and presents symptoms like those of carcinoma.

3. Foreign Body. Stricture or difficulty of deglutition from the presence of foreign bodies is usually recognized with ease. The difficulty of deglutition is due both to the foreign body and to the spasm excited by the mass. In consequence of the latter regurgitation of food takes place. In the first place, there is a history of the swallowing of a foreign material. Sudden pain succeeds the act, while there are great anxiety and distress, particularly if the body is a large, hard mass. Not only is there difficulty in deglutition, but also dyspnœa. The latter is due to pressure, but is aggravated by the nervous state. When the foreign body is small the dysphagia is moderate in degree and the reflex irritation slight, although nausea and vomiting may be common. If it cannot be removed, ulceration and abscess result, the further course of which depends upon the seat of the obstructing material. Pain, hemorrhage, subcutaneous emphysema, and the emission of air are symptoms which follow. The exact location of the foreign body may be ascertained by the use of the Röntgen rays, as in the remarkable case of White's.

Harrison Allen[1], in his exhaustive essay, calls attention to several

[1] "Foreign Bodies in the Œsophagus." Allen: New York Medical Journal, August 17, 1895.

features. Many of the symptoms are primary and some are secondary. The former are due to the trauma and the presence of the foreign body; the latter to the secondary ulceration. This softening and ulceration of the walls may take place rapidly. Allen does not think that pain or the occurrence of convulsions is of much significance, but that emphysema, the excessive secretion of mucus, and the emission of air are important signs. Anxiety he considers of very common occurrence and very suggestive. The excessive secretion of ropy mucus, salivation included, is, in Allen's judgment, pathognomonic of disease in the pharyngo-larynx or in the œsophagus, at or above the level of the left bronchus. This secretion may be an early indication of cancer of the œsophagus. It may occur in aneurism.

Dilatation of the Œsophagus. Primary dilatation of the œsophagus is an extremely rare affection. The chief symptom is the regurgitation of food, which is neutral or alkaline, and may be returned some time after the act of swallowing. The patient sometimes complains of a sensation of distention along the course of the œsophagus, with heat and burning. The odor of the breath is fœtid. If the œsophagus is not deflected, a bougie can be passed through its course.

If the dilatation is secondary, the amount of dysphagia depends upon the obstruction. Food, however, is not returned immediately. After remaining an indefinite time, not longer than two hours, it is regurgitated unchanged. Bougies, of course, do not pass. In sacculated dilatation, which usually takes place in the posterior wall near the pharynx, a bougie may sometimes pass, and at other times may be caught in the sac. The sac may be enlarged, so as to retain a considerable amount of food, which is regurgitated some time after it is swallowed. A sacculated diverticulum, from traction on the outside of the œsophagus, may occur when there is glandular disease of the neck, with adhesions to the œsophagus.

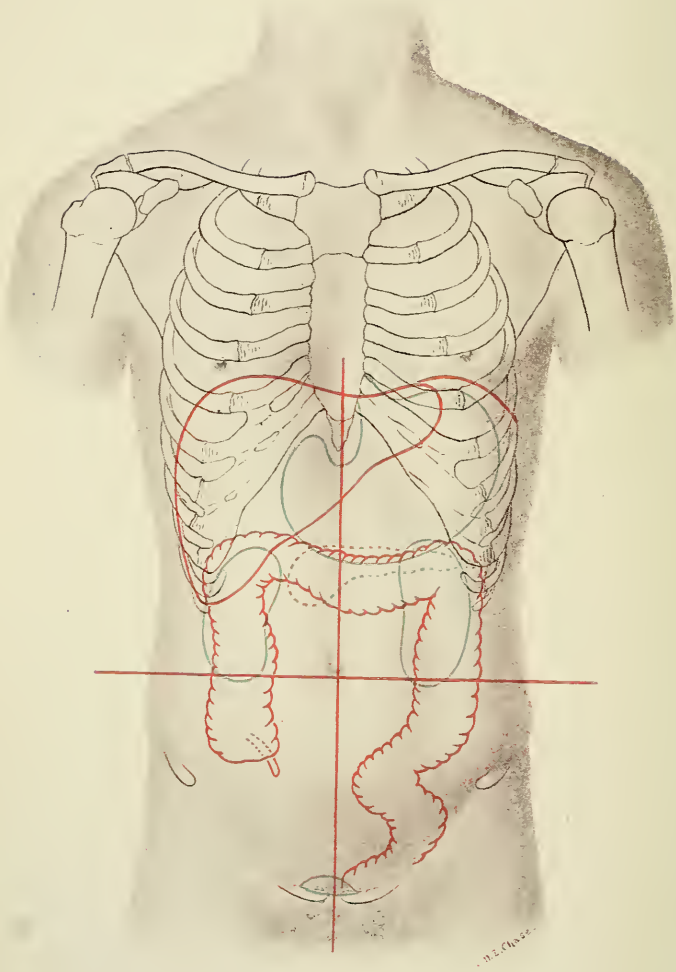
Functional Affections of the Œsophagus. The functional affections are quite as common as organic disease. They are of longer duration, but are unattended by the same grave effects upon the general system. *Spasm* is one of the most frequent affections. It may be so intense as to lead to temporary stricture. It usually occurs in women. The attack comes on suddenly during the act of swallowing food. The food is at once regurgitated. After the subsidence of the perturbation, swallowing can be accomplished, if it is done slowly. It usually occurs in hysteria. The patient may have had some slight accident in the performance of the ordinary act of deglutition, out of which grew the idea that swallowing cannot be accomplished. In consequence, the further acts are performed with trepidation, and slight emotional disturbance at the table may cause a recurrence of the sudden spasm.

Unfortunately calling attention to the act of swallowing always has the effect of embarrassing the patient, and the taking of a meal under unusual circumstances is sure to be attended by complete dysphagia. Sometimes the idea is conceived that certain forms of food alone cannot be swallowed. It is usually thought that solid food gives the distress. Mitchell says that the dysphagia occurs early in cases of

hysteria; unless relieved, the hysterical symptoms are likely to be transferred to the stomach. I saw a female patient who, after an ordinary choking attack, for several years could not swallow food in the presence of strangers, or after the slightest emotional disturbance, or if hurried. The spasm disappeared after treatment with bougies.

In *paralysis* difficulty of deglutition is the main symptom. The course of œsophageal paralysis depends upon its cause. The larynx is usually affected at the same time, so that laryngeal symptoms are present. Paralysis generally comes on very gradually. It may be due to cerebral hemorrhage, tumor, bulbar paralysis, or to general paralysis of the insane. The bougie passes easily, and does not cause irritation. In paralysis there is no regurgitation of food.

PLATE XXXV.



Quadrants of the Abdomen. Position of the Viscera.

Liver and colon—red lines. Stomach, kidneys and bladder—solid green lines. Pancreas—dotted green lines.

CHAPTER V.

DISEASES OF THE STOMACH, INTESTINES, AND PERITONEUM.

The abdomen is divided arbitrarily into regions, to enable us to locate the various organs in health and in disease. Simplicity is essential, and a method of delimitation that is commonly used in the subdivision of other regions should be adopted, for the sake of uniformity of description and to assist the memory of the learner. For these reasons Ballance's method of dividing the surface is the best. This author includes the abdomen within a circle which has the umbilicus as its centre. The circle is divided into quadrants by diameters drawn at right angles, corresponding to the median and transverse umbilical lines. The portions to the right of the middle lines are the right upper and lower quadrants, respectively; the portion to the left, the left upper and lower quadrants. (See Plate XXXV.)

With the abdomen thus divided, the umbilicus and fixed bony structures in the periphery of the circle serve as points from which measurements are made to indicate the exact position of the structure. The circle may be further divided by other radii. To locate a tumor in the right lower quadrant, for instance, the umbilicus, pubic bone, and anterior spine of the ilium may be used as points from which to measure the distance. Measurements may also be made along the radii extending from the umbilicus to fixed points. The following illustrates a useful method: A tumor is situated in the right lower quadrant; the centre of the tumor is two inches below a point on the transverse umbilical line, three inches from the centre; it is also three inches to the right of a point on the median line, two inches from the umbilicus. The size of the tumor can be defined by measurements from its own centre. Organs bisected by the median line, as the bladder and uterus, can be described as situated in the median line, so many inches to the right and left, as the case may be, and so many inches from the pubis.

The right upper quadrant includes the right lobe of the liver, the gall-bladder, the hepatic flexure of the colon, and part of the transverse colon, a portion of the pancreas, the pyloric orifice near the median line, and, deeper, the upper half of the kidney; the left upper quadrant, the left lobe of the liver, the stomach, part of the transverse colon and the splenic flexure, the pancreas, the upper portion of the kidney and the spleen; the right lower quadrant, the cæcum, the ascending colon, appendix vermiformis, right tube and ovary, a portion of the bladder and uterus, and, above, the lower part of the kidney at the end of full inspiration; the left lower quadrant, the corresponding tube, ovary, and portions of the bladder and uterus, the descending colon, and the sigmoid flexure, but not likely the lower part of the kidney, as it is one-half inch or more higher than the right (Holden). About

the centre and extending to the periphery on all sides are the small and large intestines.

The Data Obtained by Inquiry. The Subjective Symptoms of Abdominal Disease.

This class of symptoms will be discussed in the articles devoted to affections of the particular organs of the abdomen, because the symptoms are usually directly referred by the patient to the affected organs. They are local sensations of heat, fulness, or distention, of burning, of pain, of weight, or of undue motion. Local sensations of *weight, fulness*, or *distention* are due to enlargements or to displacements of organs (liver, kidneys), or to tumors. *Heat* or *burning* is described in inflammatory tumors, as pyosalpinx. It is often difficult for the sufferer to define the location of *pain* in the abdomen and describe its features. Moreover, the pain is frequently due to disease of the walls of the abdomen, which may increase the confusion. Pain must be investigated by an examination of each structure in close proximity to the part complained of. The state of the function of each organ must also be inquired into.

Pain Confined to the Abdominal Walls. The skin, the nerves, the muscles and fascia, the connective tissue, may be the seat of pain. If the skin is affected, the pain is usually localized and of moderate degree of severity. There is superficial tenderness. There are evidences of inflammation, as erythema or ulcers. Pain due to affections of the nerves is seen in simple neuralgia and herpes zoster. Herpes zoster is recognized by the localized neuralgic character of the pain in the distribution of superficial nerves and the peculiar eruption which follows. Neuralgias are recognized by the well-known points of tenderness, the intermittent character of the pain, and the association with anæmia; neuritis may be present, with the usual objective signs.

Rheumatism. The muscles and fascia may be the seat of rheumatism, causing severe pain. The muscles are tender. Movement always increases the pain, and sighing, laughing, or coughing aggravates it. The pain may be diffuse and severe, causing it to be confounded with peritonitis. The presence of rheumatism in other muscles, of moderate fever without gastro-intestinal disturbance, of uric acid and urates in excess, due to the rheumatic diathesis, point to the true condition.

Referred Pain. A common cause of pain in the abdomen is disease of the vertebræ, with pressure upon the peripheral nerves at their emergence from the spinal column. The pain is situated in the median line, either below the ensiform cartilage or around the navel; it is an intermittent pain. Aneurism of the abdominal aorta, with pressure upon and erosion of the vertebræ, causes the same kind of pain.

Pain within the Abdomen. The *seat* of the pain, if general or local, will be considered in discussing the special organs and their diseases. In general, it may be said that the seat of the pain is a fair index of disease of some structure in the part indicated. When the pain is general it points to rheumatism or to peritonitis.

Character of Pain. Attacks of severe pain in the abdomen may be sudden in onset, or the culmination of slight sensations of discomfort

progressively increasing in severity. The pain may be of brief duration or may continue over a long period of time. Sudden acute pain points to inflammation, to perforation of some one of the hollow viscera, to gastralgia, to enteralgia, flatulent distention of the stomach or of the intestines, or to occlusion of channels, of which the abdomen contains so many. Attacks of sudden pain are spoken of as *colic;* the onset is sudden; the pain is paroxysmal; each spasm of pain may be attended by vomiting, rapid pulse, cold extremities, cold sweat, and more or less collapse, except in lead-colic. Such pain is seen in intestinal colic, hepatic colic, renal colic, and in uterine and vesical colic.

Sudden acute pain occurs in *perforation* of some one of the hollow viscera, indicated by the history and location of the disease of the part affected and the character of the symptoms attending the pain. Thus, in a case of gastric ulcer, sudden pain indicates possible perforation, which may take place in the course of the disease. Chronic pain points to ulcer, to chronic processes, or to gastric or intestinal neurosis.

The Data Obtained by Observation.

The Objective Symptoms. It must be remembered that objective symptoms of abdominal change are not alone due to disease of the abdominal contents, but also to disease elsewhere. Thus the abdomen may be enlarged from the ascites of cardiac or renal disease, contracted in tuberculous meningitis.

Disease or paralysis of the diaphragm alters the appearance of the upper half of the abdomen and its movements in respiration. Fluctuating changes in size occur in hysteria and gastric neurasthenia, and permanent change in tuberculous meningitis.

Inspection. We note the appearance of the abdominal walls, the movements of the abdomen, its general shape and size, local enlargements.

The Abdominal Walls. A glance suffices to tell of the thickness of the abdominal walls. *Thin* walls are due to absence of adipose tissue and of muscular structure associated with general atrophy (see Emaciation), on the one hand, or sometimes in consequence of intra-abdominal pressure. Frequent pregnancies, previous ascites or antecedent growths (ovarian tumor) lead to atrophy of the muscles; the recti separate and hernia-like protrusion of abdominal contents results. Furthermore, a conical projection of the lower median portion of the abdomen is brought about, especially if ascites is present. Such projections are often confusing when tapping is to be resorted to. *Thick* walls are due to œdema or to increase in fat.

The Color. The abdomen, in general, partakes of the hue of the skin. It is darker around the umbilicus. In Addison's disease a distinct areola often forms. The median line, from the umbilicus to the pubis, darkens in pregnancy—the "brown line." It is sometimes seen in men. The skin of the abdomen is the seat of specific eruptions, as in typhoid fever, and of sudamina. The walls may be pale and glistening in œdema.

Markings. In first pregnancies and great ascites, less frequently in obesity and tumors, *striæ* are produced in the parts of the skin where

the tension has been greatest. In pregnancy they form sinuous lines upon the lower lateral portions of the abdominal wall and upon the upper inner portions of the thighs. When first developed they are reddish, but subsequently become, by a process of fading, more glistening and white than the rest of the skin. They are also known as "water lines," and *lineæ albicantes*. Rarely they are seen after typhoid fever.

The *umbilicus* may project from hernia or may be prominent in ascites. The veins about the umbilicus are often enlarged in cirrhosis even to such an extent as to produce a large soft tumor, the *caput Medusæ*. Not infrequently the walls around the umbilicus are infiltrated with carcinoma, occurring secondarily to gastric carcinoma. In tuberculous peritonitis, as pointed out by Henry, this infiltration, more inflammatory, however, is seen. Removal of such nodules for microscopical study often establishes a correct diagnosis of the internal disease.

Glands. Sometimes isolated lymphatic glands are seen in the abdominal wall. They may be utilized by a microscopical examination to confirm any suspicion of malignant disease.

The Veins. Enlargement of the *superficial veins* is a common accompaniment of cirrhosis of the liver, adhesive pyelophlebitis, and of any cause which obstructs the free circulation in the inferior vena cava. In order to complete the collateral circulation they may anastomose with the mammary veins above or the epigastric veins below. The *caput Medusæ* has already been described.

The Movements. (See the Lungs—Dyspnœa.) The *movements* of the abdomen are of respiratory, vascular, gastric, and intestinal origin. Much is learned by carefully observing them.

Respiratory Movements. The upper half of the abdomen swells or rises synchronously with inspiration. In enlargement of the abdomen and in tumors within the upper half the movement is restricted. In paralysis of the diaphragm it falls in with inspiration, reversing the normal movement. If such paralysis is limited to one side, as in large pleural effusions, the inspiratory collapse is unilateral. In laryngeal and tracheal obstruction, inspiratory retraction is noteworthy and its extent significant of the amount of obstruction. Respiratory movement causes the liver to rise and fall. In persons with thin walls, its shadow can be seen to descend with inspiration, the extent indicating the degree of *respiratory expansion*, the size and position of the liver. Such information is of great value. A tumor connected with the liver and an enlarged gall-bladder will move synchronously with respiration. Other growths are fixed, unless adherent to the liver. Rarely an exception is seen in movable right kidney.

Vascular Movements. They are noted in the median line and usually in the upper half of the abdomen. In moderately thin subjects the aorta may be the cause of such pulsation. (See Epigastric Pulsation.) If the pulsation is wide and extends to the right or left of the median line, an aneurism may be suspected, or the impulse may be transmitted to a growth overlying the aorta, as a carcinoma of the stomach. Aneurism of the cœliac axis will give rise to a movement near the umbilicus and to the right or left of the median line. Pulsation of the

liver, of vascular origin, and hence rhythmical with cardiac pulsation is seen in the hepatic area in right-sided dilatation of the heart.

Gastric and Intestinal Movements. Peristaltic movement, either of the stomach, the large or the small intestine, may be seen through the abdominal walls. In gastric dilatation and gastroptosis the waves may be seen in rhythmical succession, from left to right, in the centre of the abdomen. Their general course may be from the left upper to the lower right quadrant. If of the large intestine, the waves are confined to the course of this canal; if in the small intestine, to the region around the umbilicus. It is due to obstruction of the pylorus, if gastric, or of the lumen of the bowels if intestinal. The application of a cold napkin will excite the movements.

The Shape. In general enlargement the shape is uniform. In large accumulations of fat, in women with relaxed abdominal walls, the abdomen may be pendulous. In ascites the tissue over the umbilicus may protrude, changing the uniform appearance. Abdominal enlargements due to ascites, in women whose abdominal walls have previously been relaxed, sometimes assume a peculiar cone-shape; the base corresponding to the plane of the abdomen, the apex rising below the umbilicus. This is particularly the case if the patient has had to assume the semi-erect position for some time. It is often difficult to decide where to tap in such cases. In local enlargements the surface is often irregular, the prominences corresponding to the seat of the enlargement. The shape changes in hysterical distention. In enlargement due to wasting disease of the viscera, as cancer of the retroperitoneal glands, the abdomen retracts in the later stage of the disease, causing undue prominence of the viscera affected.

General Enlargement of the Abdomen. The abdomen differs very much in size in different persons, depending not only upon the thickness of the fat in the abdominal walls and omentum, but upon the calibre of the intestines themselves, which are apt to be much distended in those accustomed to eat large meals. In general, the belly is more protuberant in infants and children than in adults. Enlargement occurs in obesity, and it is often difficult to tell whether the excessive deposit of fat in the abdominal walls and omentum accounts for the whole enlargement or only serves to mask the presence of a tumor. Enlargement of the belly is only a part, though frequently the most pronounced evidence of obesity; whereas, in enlargements of the abdomen from tumors and *ascites*, there is usually a marked contrast between the size of the abdomen and that of the rest of the body.

Ascites.

In enlargement from *ascites*, when the patient is lying upon his back, the front of the abdomen is flattened, while the flanks bulge. If he turns upon his side, the flank which is uppermost becomes hollowed out and the front of the belly is prominent. This is the appearance in moderately large effusions which have existed long enough to stretch the lateral abdominal muscles. When the effusion is enormous all parts of the belly are distended, and the abdomen is barrel-shaped; no change of shape occurs upon change of posture.

Ascites is the accumulation of fluid in the peritoneal cavity. The causes may be local or general. Its local origin may be, first, simple, cancerous, or tuberculous inflammation of the peritoneum; second, portal obstruction from disease of the liver, as cirrhosis, or disease of the portal veins, either from compression or inflammation. Tumors of the abdomen are often attended by ascites, particularly solid tumors of the ovary. The *general* causes of ascites are those which give rise to dropsy.

Physical Signs. (Plate XXXVI.) *Inspection.* The abdomen is uniformily enlarged. The surface is usually smooth. The skin is tense if the effusion is large, and *lineæ albicantes* may be seen. The navel may project. If the ascites is due to liver disease or disease of the portal vein, the superficial veins enlarge, although the enlargement is sometimes seen when any effusion continues a long period of time.

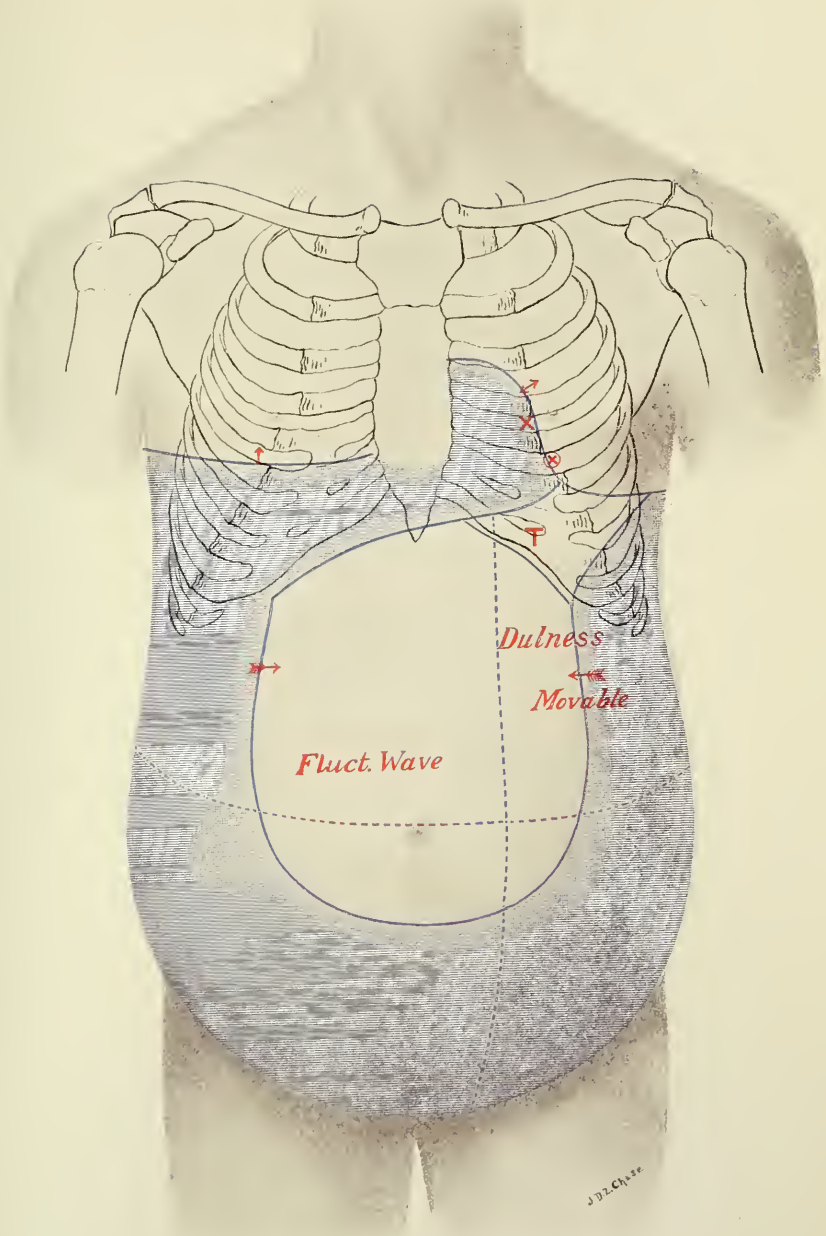
Palpation. On palpation fluctuation can usually be detected. Care must be taken not to confound the wave of the abdominal walls, produced by percussion, with the wave of true fluctuation; the former must be cut off by the hand of an assistant placed vertically in the median line. The left hand should be applied firmly against one side of the abdomen, while with the right percussion or tapping is gently performed at the opposite point. The points selected should be at about the level of the fluid. At first the hand should be placed on the flank, and if the fluctuation is not revealed, then with each successive percussion it should be brought forward toward the median line. Sometimes light percussion will yield the sign, at others more firm percussion must be employed. The faintest tap may be sufficient. In order to ascertain the position of solid organs in ascites, *dipping* is employed. This consists in suddenly pressing the tips of the fingers over the organ sought for. The fluid is thus displaced and the edge or surface of the organ readily felt.

When the abdomen is *percussed* in the usual manner there is dulness over the fluid. As the fluid gravitates to dependent portions the dulness is found in these parts. Sometimes the colon gives rise to tympany in the flanks, as pointed out by Tyson. When the patient is lying down, it is in the flanks, and may extend around the lower portion of the abdomen. If the patient stands up, the dulness may reach to the umbilicus in the median line and to the same level in the mid-clavicular line.

Aspiration In ascites it is important to ascertain the nature of the fluid. This can only be done by aspiration. If the fluid is serous, it has the characteristics belonging to that fluid. Hemorrhagic effusions usually occur in cancer and tuberculosis, although both of these diseases may occur with clear serum. In ruptured tubal pregnancy the effusion is hemorrhagic. In rare cases a chylous, milky fluid is found in disease of the lymphatics. In one instance this occurred from perforation of the thoracic duct. Chylous ascites may, however, be due to an excessive milk-diet. In other instances it is due to filaria. The patient on a mik-diet is often lipæmic, in consequence of which effusions are made turbid.

The *subjective symptoms* are those due to the cause of the ascites and to mechanical pressure.

PLATE XXXVI.



Ascites.

Blue shading shows level of dulness in recumbent posture. Dotted lines indicate change of level of fluid in other postures.

Ascites must be distinguished from enlargement of the abdomen due to ovarian tumor, enlargement due to pregnancy, and enlargement due to an overdistended bladder. In ovarian tumor the development at

FIG. 188.

Ascites. Upper limits of dulness indicated by the dotted line. Umbilicus prominent.

first takes place to the right or left of the median line. When large the signs may be in the central region of the abdomen. The flanks, however, are always tympanitic on percussion. On vaginal ex-

FIG. 189.

Ascites from sarcoma of ovary. Dislocation of liver and spleen. × is apex beat, not lifted because of fallen abdominal organs.

amination the local disease may be ascertained. A distended bladder should always be thought of, and catheterization performed in doubtful cases. Cysts of the pancreas may be mistaken for ascites, and

large hydatid cysts connected with the liver may simulate an accumulation of fluid in the peritoneal cavity. The history and the appearance of the fluid on aspiration point to the diagnosis.

Enlargement from *accumulation of gas* within the bowels is general, and may attain a very high degree, giving the abdomen a uniform arched appearance resembling a barrel. The diaphragm may be pressed upward so far as to interfere seriously with respiration and heart-action. In debilitated children the enlargement due to flatulency is associated with flaccid abdominal walls, causing lateral and central enlargement. Moderate degrees of distention from gas in the intestines may be the result of eating certain articles of food, such as turnips or beans. Excessive accumulations are met with in typhoid fever; peritonitis, operative and non-operative; and in stenosis of the colon or rectum from any cause. They are also common in hysteria.

In the last month or two of *pregnancy* enlargement of the abdomen is general, especially in a woman who has previously borne children.

General enlargement of the abdomen may be due also to *fecal accumulation*, *cancer* of the *peritoneum*, to *hydatid cyst*, and to *cancer* of the *bowel*.

It has been observed in children in *dilatation of the colon*. The dilatation may take place temporarily in constipation with obstruction. In rare cases it may become permanent. In such the distention of the abdomen is enormous. It often begins in childhood and continues through adult life. Congenital obstruction, the eating of oatmeal and similar food, with attendant constipation are causes. The bowels are constipated. The constipation may continue for several weeks, during which period there is increasing dulness in the tract of the colon, with

FIG. 190.

Case of dilatation of colon. (GRIFFITH.)

fecal tumors distinguished by palpation. This condition is relieved by diarrhœa, which may continue for two or three days, during which enormous amounts of feces are passed. It may be preceded by vomiting of a fecal character. After the bowels are open the distention continues, the dulness being replaced by tympany. The abdomen was

uniformly enlarged in Hughes' and Osler's cases. Coils of the intestine, with waves of peristalsis, were seen through the thin abdominal walls. Formad's patient was an adult. It must be remembered, as described on page 729, intestinal peristalsis is observed in constriction of the bowels. The motion of the intestine above the seat of stricture is wave-like or worm-like, and the bowel itself dilated.

From a consideration of the recorded cases of so-called *idiopathic dilatation of the colon*, Treves believes that, although enormous dilatation of the large intestine may undoubtedly occur in adults independently of mechanical obstruction, in children it is probably due to congenital defects in the terminal part of the bowel.

Enlargement of the abdomen simulating ascites may be due to retroperitoneal and peritoneal *lipomata*. Fluctuation even may be detected, but repeated puncture fails to secure fluid; the negative aspiration should always suggest lipoma. This is all the more likely if the enlarged abdomen is due to a slowly growing tumor, which is probably more visible on one side than the other, but which causes little if any general disturbance except progressive emaciation, dyspnœa, and sometimes œdema of the legs. The tumor is usually crossed by a portion of the intestine.

Other causes of abdominal enlargement are diseases of the *liver* and *gall-bladder*. When these are enlarged a local swelling may be detected in the right upper quadrant; but when they attain very large dimensions, as happens not infrequently in cancer, amyloid disease, and hydatid liver, inspection may be able to detect only general enlargement, with small prominences corresponding with cancerous nodules or small cysts.

Splenic enlargements, which attain the greatest size, are from leukæmia or chronic malarial poisoning, and are usually visible only as general enlargements of the belly. There may, however, be greater prominence over the lower left ribs and in the left upper quadrant posteriorly.

In diseases of the *kidney* producing great enlargement there is usually visible a prominence in the lateral and lumbar region of the side corresponding with the kidney involved, unless there is considerable emaciation; anteriorly the enlargement, if any be visible, usually appears to be general.

Enlargements of the abdomen which begin in the lower quadrants are usually of pelvic origin. The most common are those due to *pregnancy, retroperitoneal sarcoma, cysts* of the ovary or parovarium, *fibroids* and *fibro-cysts* of the uterus, and *abscesses* or *effusions* (chronic peritonitis). A greatly *distended bladder* may cause confusion; it is a good rule to be sure that the bladder is empty, by having a catheter passed before proceeding further with the examination.

Local Enlargement or Tumors of the Abdomen. In the space below the xiphoid cartilage and between the ribs (epigastrium) local enlargements may be due to a distended or dilated stomach or to a tumor of the pylorus, which is almost always cancerous. Prominence in this region is seen in large eaters. But enlargement in this region is sometimes due to cysts, sclerosis or cancer of the *pancreas*,

to *aneurisms*, to cancer of the large intestine or tumor of the left lobe of the liver. It is in this region or to the left of the median line and nearer the umbilicus that the effusions into the lesser peritoneal cavity are found.

A rigid rectus muscle is capable of simulating a tumor. Likewise, in hysterical subjects, rigid abdominal muscles, with tympanites, give rise to a swelling known as "phantom tumor." Such swellings are less constant in shape and character than genuine tumors, and although dull on percussion appear more superficial; they sometimes disappear under friction with the hand, and certainly under full anæsthesia; the stigmata of hysteria are present.

Enlargements in the *right upper quadrant* (right hypochondrium) are most frequently due to diseases of the *liver* (*q. v.*) and to affections of the gall-bladder. Less frequently, a much enlarged *kidney* or a hydronephrosis causes swelling in this region. The differential diagnosis is made by the history of the case and by noting the direction in which the tumor has grown, by examination of the urine, and by the relation which the ascending colon bears to the tumor; kidney tumors carry it in front of them as they grow; hence, their dulness is obscured by the superficial tympany of the colon.

Primary malignant disease of the suprarenal bodies—a rare affection—is often attended by a tumor in the upper abdomen (Rolleston and Marks, *American Journal of the Medical Sciences*, 1898.) The clinical picture is not one of Addison's disease even when both the organs are invaded. Some of the symptoms occur partially, as pigmentation, vomiting, asthenia, pain in the back. The growth extends forward, and resembles in many respects renal tumor. It also, however, may resemble tumors of the liver, enlarged gall-bladder, or pancreatic cyst.

Enlargement in the *right lower quadrant* (right iliac region) is most frequently due to affections of the cæcum and appendix, to tumors of the ovary, and to pelvic abscesses.

The diseases of the *cæcum* and *appendix* causing enlargement in the right iliac fossa are fecal accumulation, typhlitis, fecal abscess, perityphlitic abscess, carcinoma, and stricture of the ileo-cæcal valve.

The diseases of the *ovaries* and *tubes* causing enlargement in this region are ovarian tumors, cysts of the broad ligament, pelvic abscess (usually tubal in origin), and extra-uterine pregnancy.

Other affections which need to be considered are *tubercular peritonitis*, acute and chronic, and enlarged or movable kidney.

Enlargement in the *left upper quadrant* (left hypochondriac region) is due to dilatation or carcinoma of the stomach; enlargement of the spleen, movable kidney, or tumors of the kidneys, and effusion in the lesser peritoneal cavity. Enlargement in the *left lower quadrant* (left iliac region) is due to tumors (cancerous) of the sigmoid flexure and to the tumor due to volvulus, and to the same causes of enlargement of the right side which are possible on the left.

Enlargement about the *centre of the abdomen* (umbilical region) may be due to umbilical hernia, to a floating kidney, spleen, or liver, or to tubercular disease of the omentum or mesenteric glands. It is seen

in retroperitoneal sarcoma. It is seen in cases of dilatation after a full meal. This region is frequently enlarged, in conjunction with a more prominent swelling extending from the sternum, in cancer of the stomach; and from the ribs on the right in cancer of the liver or gall-bladder, or other diseases of these viscera; from the ribs on the left, in effusions into the lesser peritoneal cavity, disease of the pancreas or the spleen.

Undue *projection* of the *vertebræ* must not be mistaken for tumors.

Enlargement above the pubis (hypogastric region) is due most frequently to enlargement of the uterus from pregnancy, fibroid tumors, or fibro-cysts, or to distention of the bladder; but it is also common in gastric dilatation and *gastroptosis;* flattening of the upper half is then seen, and the lesser curvature is then made out.

Enlargement in the lateral regions and behind (*lumbar region*) may occur in malignant tumors of the kidney, in hydronephrosis and pyonephrosis, in perinephritic abscess, and in renal cysts of large size. Usually renal enlargements of any kind are not observed behind, however. It may also, in the left side, be due to perigastric subdiaphragmatic abscess and to enlargement and displacement of the spleen. On the right side the cause may be enlargement of the liver, or a hydatid cyst, or a retroperitoneal sarcoma.

Diminution in Size. The abdomen is diminished in size in wasting diseases, or such as result in insufficient food being taken. This class comprises cancer of the œsophagus and stomach, chronic lead-poisoning, anorexia nervosa, and chronic diarrhœa and tuberculosis of childhood. In the second stage of tubercular meningitis in children there is retraction of the abdomen. The wasting of the subcutaneous and the omental fat and atrophy of the abdominal organs cause the abdomen to be concave or *scaphoid.*

Palpation and Percussion of the Abdomen. Palpation and percussion in diseases of the abdomen may be discussed together.

Position of Patient. Generally the best position is the recumbent one, because it admits of examination without too great exposure, and because in that position the abdominal muscles are partly relaxed. When the muscles need to be still further relaxed the patient should lie upon the back, with the head and thorax partly elevated and the knees drawn up. In certain obscure tumors much can be learned by having the patient rest on the hands and knees, or assume a knee-chest position. By this means we can determine if the pulsation is due to aneurism or to a tumor. The latter falls away from the vessels, and hence pulsation is lessened thereby in the knee-chest position. A tumor surrounded by coils of intestine may thus become more palpable. A good plan to secure relaxation for palpation of the liver and spleen is to have the patient sit on a chair with the body leaning forward; then flex the thighs, supporting the feet on a stool or the rung of another chair.

Method. The examining hand should be warm, as the application of a cold hand throws the abdominal muscles into involuntary contraction. By grasping the abdominal walls between the thumb and fingers their thickness and the relative proportion of *fat* can be estimated.

So, too, the presence or absence of *œdema* of the skin can be readily detected. This œdema is general, but is especially marked in the lateral and posterior portions of the abdomen. Relaxed abdominal walls occur after dropsy and pregnancy. Redundant skin remains in folds when pinched up. This is particularly so in abdominal cancer.

When it is desired to explore deeply the patient should be instructed to breathe with the mouth open, and the examining hand pressed firmly in during respiration, and held there, if need be, during several long respirations. The palm of the hand should be laid upon the surface ; after the muscles are relaxed the flexed fingers may be used to palpate. The same procedure is adopted when we desire to get the percussion-note of a body lying deep in the abdomen : the finger is pressed firmly and deeply in, and then percussed. In this way any superficial resonance due to overlying intestine is largely eliminated.

When palpating to determine the lower edge of the liver or spleen the palmar surface of the fingers is pressed into the abdomen at different levels from below upward until the edge is felt. The edge of the right lobe of the liver in its normal position extends to the margin of the ribs. It may be detected by pressing the fingers in as described and having the patient take a long breath.

By *palpation* the information obtained by inspection is confirmed ; the character of the abdominal walls and of swellings is determined ; the precise location of pain is ascertained ; the condition at the hernial rings and the movability of tumors are investigated. The condition of the integument should first be determined. Passing the hand gently over it is sufficient to decide whether it is normal, smooth and elastic, or harsh and dry. Any marked *unevenness*, such as is produced by umbilical and inguinal hernia, by striæ, or by large tumors of the pylorus, or cancerous nodules, and hydatid cysts of the liver, can readily be detected. The degree of *tension* of the abdominal walls is easily appreciated. It is increased, of course, in all forms of great enlargement, but not equally ; some persons are so sensitive to touch that any attempt at palpation throws the abdominal muscles into such rigid contraction that examination is impossible. *Rigidity* of the abdominal walls may be the only sign of acute peritonitis. It is common in local peritonitis. The recti muscles contract quickly on hurried palpation. Local contractions point to inflammation underneath. In tuberculous peritonitis we see distention with board-like rigidity or preternatural hardness. The term *carreau* is used by the French for this condition. *Peritoneal friction* may be detected most frequently over the liver and in chronic peritonitis.

Palpation and Percussion of the Lower Quadrants. On the right side, the groups of affections connected with the cæcum and appendix, the uterine appendages, and the peritoneum, which cause enlargement in this region, have been mentioned already under local inspection of the abdomen. Palpation and percussion, however, are the methods which afford the most exact information of their physical characteristics, and, with the clinical history, enable us to distinguish one from the other.

Diseases of the Appendix and Cæcum. The information supplied by palpation and percussion in perforation of the appendix will depend upon the rapidity with which perforation has supervened and upon the stage at which the examination is made.

Generally speaking, after the sudden onset of pain in the right iliac fossa, in a person previously in good health, there is tenderness on palpation in that region. This tenderness is first localized, but may spread with great rapidity over the whole abdomen. Or the tenderness may at first be general, and subsequently become localized over the appendix. Subsequently, the tension in the part is increased, the overlying abdominal muscles are rigid (spasm) and firm, and the percussion-resonance impaired. Examination with the finger in the rectum may discover a tense, swollen appendix, or a tumor in the pelvis.

But the disease may be fulminating in character, perforation being followed by the rapid development of peritonitis, with collapse, so that when the patient is seen there will be no more tenderness over one part of the abdomen than over another.

Again, the appendix may be subject to repeated attacks of inflammation without perforation, but with the development of local peritonitis. There is increased thickness in the region of the cæcum, tenderness, diminished resonance, and increased resistance to the percussed finger. Sometimes an enlarged and hardened appendix can be made out by palpation, both during an attack and in the intervals.

In still other cases, of slower development, a distinct perityphlitic abscess develops. In addition to local pain and tenderness a swelling appears above Poupart's ligament. The skin over it becomes brawny and pits on pressure with the finger-tips. The tumor is dull on percussion, and on palpation obscure deep-seated fluctuation may be secured. A fluctuating tumor may also be made out by rectal examination with the finger.

In *fecal impaction of the cæcum* a tumor forms, following the course of the cæcum, and directed upward from Poupart's ligament. It is usually oblong and rounded, and may appear uneven or lumpy on closer palpation ; it is not tender unless the cæcum itself becomes inflamed. It has a doughy consistency. *Fecal* tumors give rise to some distention of the abdomen. To distinguish these tumors from solid growths, Gersuny calls attention to the "adhesive symptom." If strong pressure is slowly made with the finger tips on the tumor, and then the pressure be withdrawn gradually and the hand removed from the abdomen, a peculiar sensation due to the separation of the intestinal mucous membrane from the fecal matter is transmitted to the hand. If the feces are dry and hard, the sensation may not be observed until an oil enema is used. When the feces are soft naturally or artificially, the tissues remain depressed and only gradually separate from the mass and return to their normal position. Slowness of the separation of the abdominal walls from the tumor is also characteristic of the fecal accumulation. The diagnosis is made by the situation and character of the tumor, and the absence of pain, tenderness, and constitutional symptoms, and by its disappearance under the influence of purgatives.

If the impaction causes a localized colitis, or so-called *typhlitis*, the tumor is tense, tender, and painful, dull on percussion, the dulness being sharply limited by the boundaries of the cæcum.

Appendicitis.

This is by far the most important affection of the intestinal tract. It is of frequent occurrence compared with intestinal obstruction, and, if recognized, is amenable to relief in a very large percentage of the cases; whereas intestinal obstruction is more frequently fatal. We see twenty-five cases, at least, of appendicitis in all its forms to one case of any form of obstruction. Its importance, therefore, is readily recognized. Appendicitis occurs most frequently in the young—in the large proportion of cases under thirty. I have seen it as early as two years of age, although from the fifteenth to the thirtieth year it is more frequent than at any other period. The symptoms vary, but clinically may be divided into those of appendicitis without perforation and appendicitis with perforation. Appendicitis without perforation is characterized by relapses, and is known also as *recurring appendicitis.*

Appendicitis without Perforation. Cases of catarrhal appendicitis probably occur, although I am not prepared to say that catarrhal inflammation of the appendix gives rise to marked local symptoms, for in cases on the post-mortem table in which the lesions of catarrh were found there had not been any symptoms during life, due either to intestinal catarrh or to any symptoms pointing to appendicitis in any form. Moreover, many cases in which the attacks of appendicitis had at first been slight, finally developed into appendicitis with perforation. In the milder cases, if operative measures are resorted to during the intervals between the attacks, the appendix is always found to contain a fluid loaded with micro-organisms which are capable of causing purulent inflammation, as the staphylococcus or streptococcus. Clinically, therefore, all forms of appendicitis should be considered infectious, with, on the one hand, escape of the contents into the bowel, and natural relief of the symptoms; or, on the other, complete obstruction with perforation. After removal of the appendix in cases of recurring appendicitis, I have always found pus or a mucopurulent material which was charged with streptococci or staphylococci, as well as the bacillus coli communis, natural to the intestinal canal in this region.

Symptoms of the Attack. After exposure to cold rarely, frequently after an indiscretion in diet, the patient is seized with pain, referred to the right lower quadrant of the abdomen. It is paroxysmal in character, increasing in intensity, and may be complained of as colicky. The pain is usually such as to require the patient to take to bed and attempt to secure relief by local applications. The severity of the pain may be so slight that the patient pays but little attention to it. He may even go about his business during the time and seek professional advice at the office of a physician. Such cases as these are attributed to ordinary cholera morbus or intestinal indigestion. The attack may be only moderately severe, particularly if there is

diarrhœa. With the onset of the pain vomiting usually occurs. The bowels may be open or they may be confined. Vomiting may not occur if there is diarrhœa. It is usually attended by some nausea, although this is not marked. The vomiting is complete, there is no retching. It occurs at intervals, between which there is comparative comfort. The contents of the stomach are ejected, and then mucus. If the patients are to get well, vomiting does not return unless excited by food. If peritonitis supervenes, vomiting returns in the course of two or three days. If in bed, the patient lies on his back with his right leg flexed.

Even with a mild degree of pain the skin is hot and temperature slightly raised. In the cases in which the pain is more severe the general reaction is greater. The temperature rises rapidly to 102° to 103°. The skin is hot and dry, the face flushed. The pulse in a young adult rises to 90 and 95. It is full and strong. On account of the pain there is some restlessness. In some cases the patient complains more of the fever than of the pain after its first severity has subsided. The tongue is coated ; appetite is lost.

On *physical examination* the area which was the seat of pain is found to be tender. When examined with the tip of the finger pressing firmly, a point of more marked tenderness can usually be found on a line midway between the anterior superior spine of the ilium and the umbilicus. It is known as McBurney's point, and is most characteristic. It indicates the site of the diseased appendix. The swollen tender appendix may occasionally be palpable. On *inspection* the affected area is slightly or may be considerably enlarged. Comparison must be made with the opposite side. It will be seen that the usual depression in front of the anterior spine, or the cavity toward the loin, is not so deep as on the opposite side. In front the surface may be even with the plane of the ilium. On *palpation*, in addition to tenderness and pain at the point previously indicated, fulness and enlargement can be distinguished. There is resistance to pressure and more or less rigidity of the abdominal muscles. On careful *measurement* the semi-circumference will be found in most instances to be larger than the semi-circumference of the opposite side. When bimanual palpation is performed, the left hand being placed in the loin behind and the right over the abdominal surface, resistance, induration, and rigidity can more easily be detected. On *percussion* there is change in the note compared with that of the opposite side, and change in the percussion-note during the course of the disease. This is particularly the case if the symptoms go on to perforation. On careful deep percussion a dull tympanitic tone is elicited, or a distinct area of dulness can be mapped out, but in some instances the distended cæcum yields tympany, which is greater than on the opposite side.

The *pain* is usually referred to the region above mentioned. The pain may be in the lower quadrant on the *left* side instead of the right. It is seen in those cases in which the appendix normally dips into the pelvis. It may also be referred to the bladder or genitals, and be attended with vesical tenesmus and frequent micturition. The char-

acter of the pain and the bladder symptoms are such as to simulate an attack of renal colic, with the passage of sand. On account of the locality of the pain it may be attributed to the Fallopian tube or ovary, and thought to be due either to pain on account of disease of these organs or to dysmenorrhœa. It is not likely to be mistaken for the pain of dysmenorrhœa if the patient is subject to pain at the usual monthly period. If, however, the physiological and the pathological affection should take place at the same time, or the latter occur about the time of the monthly period, a mistake in diagnosis may occur, particularly as increased abdominal pain may cause a uterine discharge. The occurrence of fever would exclude dysmenorrhœa in cases in which this symptom was present. The pain and leg-flexion simulate hip-joint disease.

After the first twenty-four hours, during which the above-mentioned symptoms described take place, the fever continues. There is anorexia, but vomiting occurs only at long intervals if at all. The local symptoms continue, although modified usually by methods of treatment which are applied. Both general and local symptoms frequently subside after a free movement of the bowels, which occasionally takes place spontaneously. In other cases constipation continues a week or ten days, and even over a longer period.

After five or six days at the furthest the fever subsides, the local distention lessens, the paroxysms of pain disappear, and convalescence ensues. There may, however, be localized tenderness for a considerable period of time, and diarrhœa, or at least two or three evacuations each day, for a week or more. In rare instances peritonitis supervenes without the occurrence of perforation. Its onset under these circumstances is gradual, but the symptoms are like those of peritonitis under other circumstances. Infection takes place directly through the appendix.

When the fever continues, with mild diarrhœa, intestinal pain, and flatulency, the case may be mistaken for *typhoid fever*. The temperature is, however, more remittent in character in appendicitis, and the diarrhœa is not characteristic of typhoid fever. The eruption of typhoid fever does not occur, the spleen is not enlarged, and the symptoms of the typhoid state do not ensue. The diazo-reaction, the bacteriological examination of the stools, and the serum test, may aid in forming a conclusion.

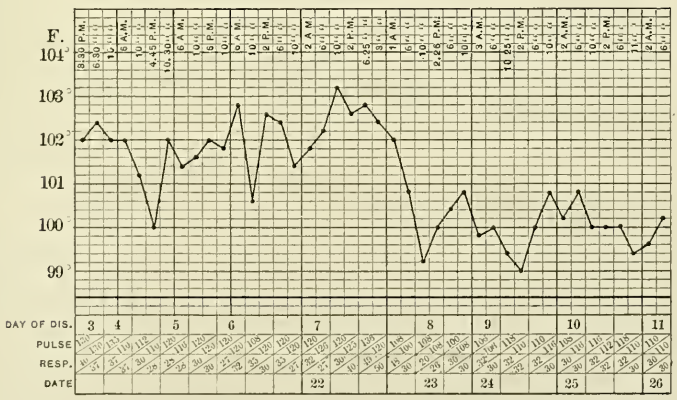
Recurrent Appendicitis. Frequent attacks of mild appendicitis occur; they may occur as frequently as every three months, or the interval may be as long as a year. The attacks are similar to the attacks just described, although the duration is shorter. The local symptoms in some instances are more marked, because there has been a localized peritonitis previously. The induration is greater, and dulness more marked. In some instances the attacks are comparatively mild, continuing but twenty-four hours, and are described as attacks of colic. Often they have been treated by the patient himself, by household remedies alone. The patient spends a night in agony, with cramps, but the next day follows his usual habits. It is possible that there has been no fever with the attacks, but in all cases of

recurrent appendicitis which I have seen, fever, although often slight, has been a constant accompaniment.

Appendicitis with Perforation. Before perforation takes place the patient may have had symptoms of the mildest form of appendicitis for two or three days, or they may have extended over a long period of time, without any symptoms except colicky pain. As observations are not made, the presence of fever cannot in such a case be utilized as a diagnostic feature. The perforation may take place early in the course of an acute attack, and result in *localized peritonitis* and *abscess*, or in *diffuse peritonitis*. If the latter, after the characteristic symptoms of appendicitis the symptoms of intense peritonitis set in. The abdomen rapidly becomes distended, the characteristic vomiting ensues, and collapse develops. Perforation under these circumstances has occurred within the first twenty-four or at most within forty-eight hours. Local inflammation about the appendix does not take place, and the local signs of an inflammatory tumor are not present, although tenderness at the special point can be elicited.

Abscess. If the perforation is more gradual, and there has been time for the occurrence of local inflammation about the appendix, by which pus is prevented from infecting the general peritoneum, or if perforation takes place behind, in the connective tissue which surrounds the mass, in which situation there is always inflammation, the local signs of abscess or inflammatory tumor occur. There is swelling of the affected side; the normal outline is effaced. The area is indurated, and the early pronounced rigidity gradually gives way to a boggy sensation, with the appearance of œdema of the skin. This can be elicited by pressure over parts that are hard and resisting, as the spine of the

Fig. 191.



Acute appendicitis, with perforation and abscess. Female, æt 8. Operation on seventh day.

ilium. Fluctuation can often be detected by bimanual palpation. Dulness is found, although in some instances it may be very slight, there being scarcely an appreciable change in pitch. Both light and deep percussion must be performed, and compared with the results of

percussion in the opposite region. Palpatory percussion may alone indicate the departure from normal. Examination per rectum may yield much information. An induration may be felt about the brim of the pelvis or the rectal fossa, which fluctuates and may eventually soften. With the finger in the rectum, and pressure above, better results may be obtained. If the symptoms of peritonitis do not arise, or rapid infection of the system take place, the signs of abscess become more and more marked. The surface becomes reddened, and pointing may take place toward the groin or opposite the spine of the ilium. Sometimes the swelling increases in the direction of the loin, and the abscess may point in that situation.

As the abscess develops the general symptoms change. They now become the symptoms of *suppuration*. The fever is remitting or intermitting. There may be chills. Sweats are common, and there are loss of appetite and diarrhœa. Until recently it was customary to see abscess develop in some other situation, or symptoms occur from burrowing of the pus in various directions. It may extend upward along the back of the colon, underneath the diaphragm, and thence to the pleura and lung, and be expectorated. The abscess may open into the rectum or into the bladder. If the local inflammation is virulent, even if peritonitis has not taken place, the symptoms of *septicæmia* may rapidly ensue. This sometimes occurs quite early in the disease. There may be vomiting and septic diarrhœa, and slight delirium at night. An excessively rapid and feeble pulse is seen ; in one instance it was irregular. Extreme prostration ensues, followed by symptoms of the typhoid state.

Gangrenous appendicitis is most treacherous. The early symptoms are like an acute attack ; all symptoms then subside. Unless the temperature is taken or the physical examination is very painstaking, the patient is allowed to get up. The course may be afebrile. In a few days or a week an abscess forms about the slough, and then the usual phenomena of suppuration set in ; or perforation may occur.

It is clear that in cases of appendicitis we must attempt to recognize : (1) The inflammation before perforation has taken place ; (2) the occurrence of perforation ; (3) the occurrence of peritonitis due to either of the two conditions ; (4) the occurrence of abscess (paratyphlitis and perityphlitis) ; and (5) the occurrence of septicæmia.

Typhlitis is an inflammation of the cæcum due to accumulation of fecal or foreign substances. The inflammation may remain as a localized enteritis, or may be followed by ulceration. In the majority of cases the ulceration is due to pressure by the contained foreign material or feces. The inflammation occurs in early life usually. The patients have been subject to constipation. The attack may follow some error in diet. There are pain in the right iliac fossa, constipation, and nausea. Moderate fever develops. On examination there is fulness in the right iliac region, and the right thigh may be flexed, the cæcal region is tender to pressure, and a doughy, sausage-shaped tumor may be outlined. The more severe symptoms last two or three days. Local tenderness may continue a week or even longer. The tumor gradually disappears. If ulceration takes place, inflammation

about the cæcum ensues. An abscess forms gradually in the flank. Perityphlitis is the term applied to this secondary abscess, although, as the term has been confused with paratyphlitis, it had better not be used in this connection.

Abscess about the head of the cæcum is due (1) to appendicitis, of which sufficient mention has been made; (2) to perforation of the cæcum, on account of typhlitis; (3) to perforation, on account of cancer of the intestine; (4) abscess secondary to kidney disease, perinephritic abscess; (5) to abscess secondary to disease of the vertebræ. The physical signs are those of abscess due to perforation of the appendix. The symptoms are the local symptoms of abscess and the general symptoms of suppuration.

Fecal abscess, arising from ulceration of the colon, may be suspected, according to Fenwick, when there is a localized abdominal swelling, immovable in respiration or by a moderate amount of pressure with the fingers, the size and shape being altered when diarrhœa occurs, and when percussion over the tumor gives a tympanitic or a more forcible stroke, a dull sound, or when an emphysematous sensation is communicated to the fingers.

Pericæcal abscess follows the stercoral typhlitis which occurs as the result of cancer in the course of the large intestine. The history of the case points to the true nature of the disease. Abscess may occur behind the cæcum in cases of caries of the vertebræ and in some rare instances of empyema in which the pus has dissected downward.

Appendicitis must be distinguished from *perinephritic abscess* and the abscess which follows perforation of the intestine or cæcum at this point. Perinephritis can scarcely be distinguished unless there has been a previous history of renal calculus and pronounced evidence of disease of that organ preceding the formation of the abscess.

Hip-joint disease must be distinguished from appendicitis. The leg is flexed, the patient complains of pain about the region of the hip; unless careful observation has been made in the beginning of the attack, the early march of appendicitis may not be recognized. The two are confounded after abscess-formation. The flexed leg of appendicitis can be extended under ether, and examination then shows the joint to be free from disease.

Fenwick says that *acute tubercular peritonitis* may be confounded with perforation of the appendix. In both there may be pain and tenderness in the hypogastrium, dulness on percussion, and fever. In tubercular peritonitis the onset is more gradual, the pain and tenderness more general, and there is no distinct tumor or increased tension in the hypogastrium. If there is dulness on percussion, the line generally varies with the position of the patient. Diarrhœa is urgent, and there are, in most cases, some signs of consolidation of the lungs. The absence of tumor in the right iliac region and in front of the rectum is the chief point of distinction; for when perforation occurs in phthisical subjects there is generally very slight pain, and severe diarrhœa is often the only prominent symptom. The appendicitis itself may be of tuberculous origin, as in several cases reported by the writer.

Returning to palpation and percussion of the lower quadrants, we find in *intussusception* a tumor, often detected in the right lower quadrant or to the right of the navel. It is generally distinct, of the shape of the bowel, not very tender, and harder than the tumor of appendicular inflammation. The diagnosis from the latter is made by the difference in the character of the tumor, by the pain being colicky and recurring in paroxysms, by vomiting and constipation being more marked, and by the tenesmus and passage of blood and mucus from the bowel. The last-named symptom and the tumor, with a constant desire to defecate, are the most characteristic features of intussusception. A tumor may be detected within the rectum by digital exploration, if the intussusception is low down. There may be distinct hemorrhage, or the passage of the invaginated portion of the bowel per rectum. Intussusception is the most frequent cause of intestinal obstruction in infants and young children. It occurs nearly twice as often in males as in females. Stercoraceous vomiting is not so common as in other forms of acute obstruction of the bowel.

In *pelvic abscess* a swelling sometimes makes its appearance on the right side, above Poupart's ligament. It is, perhaps, situated more toward the median line than perityphlitic abscess, and it is less defined than the tumor of typhlitis; but the diagnosis from these affections must be made by the history, which is usually that of an antecedent salpingitis or of previous abortion or miscarriage. Vaginal examination discovers that palpation of the uterus causes pain; that the uterus is fixed, instead of being freely movable, and that the pelvis is blocked up by an exudate on the affected side.

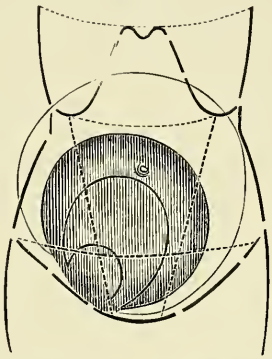
In *pelvic hæmatocele* a tumor may form in the lower half of one of the lower quadrants. It is distinguished from appendicitis, perityphlitic abscess, and pelvic abscess by the absence of fever and constitutional signs of suppuration; from perityphlitic and pelvic abscess by its sudden onset, probably at a menstrual period; by the less degree of tenderness, and by the anæmia and collapse which follow its appearance. It is almost invariably the result of a ruptured *extra-uterine pregnancy*. Hence, it may be preceded by the passage of decidua and the objective signs of pregnancy. It is distinguished from pelvic abscess by its occurrence in a woman without antecedent tubal or uterine disease, and by the less degree of tenderness of the uterus and relative absence of fixation.

In *stricture of the ileo-cæcal valve* due to cancer there is frequently a tumor in the right lower quadrant, between the umbilicus and anterior superior spinous process of the ilium, or between the latter and the ribs. The diagnosis is made by noting the fact that the tumor has developed gradually, that the patient has suffered with colicky pain, vomiting, and constipation, possibly preceded by diarrhœa, and that peristaltic movements of the intestines can readily be seen through the abdominal walls. The abdomen at the site of the tumor is somewhat distended. The tumor itself is irregular and tender, and is dull on percussion.

The disease is very rare, and is said by Fenwick to be more common in women from twenty to forty years of age.

In *tumors of the right ovary* there is at first a gradual enlargement in the right groin, unaccompanied by pain, fever, or impairment of health until the tumor has attained considerable size. They are usually cystic, and fluctuation can be obtained. The tumor is dull, and by bimanual examination, with the fingers of one hand in the vagina, the tumor can be traced into the broad ligament, and the displacement of the uterus which it occasions made out. The cystic ovarian tumors grow from the starting-point in the direction of an axis diagonally toward the median line. There is dulness in front of the abdomen and a clear percussion-note or tympany in the flanks. (Fig. 192.)

FIG. 192.



Position of an *ovarian tumor* of the right side, in various stages of enlargement. The shading indicates the *percussion-dulness in ovarian dropsy of moderate extent;* the umbilical region is dull from the presence of fluid, and the flanks remain clear. The outer circle shows a further extent which the dulness may reach in ovarian dropsy. (BRIGHT.)

PALPATION AND PERCUSSION IN THE LEFT LOWER QUADRANT. Enlargements in this region are due most frequently in women to *ovarian tumors, pelvic abscess, pelvic hæmatocele, and fibroids of the uterus,* the diagnostic points of which have been referred to already under palpation and percussion of the *right* iliac region. In addition to the affections named, enlargements are occasionally met with from fecal accumulations in the flexure of the colon, *cancer of the descending colon, tubercular peritonitis,* and enlargements or displacements of the *spleen* and *kidney* (*q. v.*). *Fecal abscess* also may occur here, and the tumor of *intussusception* may be detected on the left side.

PALPATION AND PERCUSSION ABOVE THE PUBIS. Enlargements in this region may be due to *fibroid tumors of the womb.* They occur most frequently in sterile women, and are accompanied usually by hemorrhage. Bimanual examination of the uterus will reveal an unevenness of surface of the womb if the tumor is external, and passage of the sound will detect any growth projecting into the cavity of the womb.

The enlargement may be due to *a distended bladder.* It is a good rule always to be sure that this viscus is empty before beginning an examination.

In *acute tubercular peritonitis* a swelling may develop in this region. It appears gradually, is diffused and free from tenderness, but is pre-

ceded by pain and fever. There is no palpable tumor, but the percussion-note is dull and the tension is increased. Moreover, the level of dulness is apt to vary with change of posture. The general health is markedly affected, loss of flesh is rapid, and diarrhœa and sweats are common. A focus of disease may be discovered in the lungs.

Palpation and Percussion of the Region below the Sternum. Enlargement in this region is most frequently due to affections of the *stomach* (*q. v.*). It is not uncommon, however, to find here a cancerous nodule projecting from the surface of the *liver*, or a hydatid cyst of the same organ. The diagnosis must be made by determining, with the aid of palpation and percussion, whether the tumor is continuous with the liver, the effect of respiration upon it, and its apparent depth from the surface, tenderness, fluctuation, etc., and by a study of the subjective symptoms pointing to disease of the stomach or liver. (See Diseases of the Liver.)

Much more rarely enlargement here may be from tumor of the *pancreas*, such as cyst, abscess, or cancer. According to the studies of Fitz, the former is marked by deep-seated colicky pain occurring in paroxysms, by discharges from the bowels of matter resembling saliva, by the detection of fat in the stools and sugar in the urine, by salivation, and by the occurrence of jaundice.

Cancer of the pancreas is recognized by the detection of a painful tumor in the epigastrium. The pain is not aggravated by the taking of food, but is said to be increased by the erect posture. The bowels are constipated, and the stools may or may not be fatty. Emaciation is progressive, as in all cancerous affections, and in the last stages there may be occasional vomiting and persistent jaundice.

Palpation and percussion of the upper right quadrant is limited largely to an investigation of changes in the liver and gall-bladder, and is discussed in the section devoted to them.

Palpation and Percussion of the Upper Left Quadrant. Enlargement in this region is generally due to disease of the *spleen* (*q. v.*). It may be due to *fecal accumulation* in the left transverse and descending colon. This condition is recognized by the painlessness and doughy consistence of the tumor, and by careful inquiry as to the condition of the bowels. Constipation will, of course, exist, but both patient and physician may be misled by apparent diarrhœa, or even dysentery; there will be fluid or semi-fluid dejections mingled with scybala, and sometimes mucus and blood.

An interesting cause of swelling in this region, and in the lumbar region, is *perigastric*, or *subdiaphragmatic abscess*, a collection of pus walled in by the stomach, spleen, diaphragm, colon and the abdominal walls.

The most common cause is the irritation of a gastric ulcer which has nearly or quite perforated, and has formed adhesions with surrounding viscera. This was the cause in forty-one out of fifty-two cases analyzed by Fenwick, while in six it was associated with cancer and in four with abscess commencing externally. Pain in the epigastrium or abdomen was the chief subject of complaint, and in most of the cases there was dyspepsia, sometimes vomiting. It is singular

that hæmatemesis was mentioned in only two cases. Fenwick thinks that in every case of perigastric abscess, except in persons affected with phthisis, cancer, or some other chronic exhausting malady, the first formation of the abscess will be accompanied by either collapse and signs of general peritonitis, or by sudden and severe pain in the epigastrium, attended with indications of local peritonitis.

Fever is a prominent symptom, but physical signs are absent. A tumor, according to the same author, is rarely distinguishable except when the cause is cancer. It is dull, but afterward tympanitic on percussion, and not movable on inspiration or external pressure. The tension of the abdominal muscles prevents successful palpation. There may be arching outward of the ribs. The displacement of surrounding viscera will depend upon the size of the abscess and the extent of adhesions. The diaphragm is pushed upward, and dulness may extend as high up as the angle of the scapula, in which case a pleural effusion is simulated. Breathing is embarrassed by the upward pressure upon the lung and heart. Sometimes when gas is formed in connection with the abscess, amphoric sounds on auscultation and percussion are heard both in the abdomen and over the thorax. To this condition the name *pyo-pneumothorax subphrenicus* has been applied. The abdomen then becomes tense, tender, prominent, and tympanitic on percussion. (See p. 578.) It must be distinguished from left *pneumothorax*. Air in the pleural cavity pushes the left wing of the diaphragm down, and hence increases the area of percussion-dulness and the palpability of the left lobe of the liver and spleen. In subdiaphragmatic abscess with gas, the liver and spleen are not palpable, nor can their area be limited by percussion. The heart is dislocated in pneumothorax, and its area tympanitic on percussion, while the impulse is seen in the epigastrium or to the right of the sternum. In subphrenic pneumothorax the heart is elevated, and the impulse seen in the nipple-line. At the same time there is tympany in the lower half of the cardiac area of dulness. *Pyo-pneumothorax subphrenicus* must not be mistaken for dilatation of the stomach.

Palpation and Percussion of the Loins. Enlargements in these regions may be due to affections of the *kidney* (*q. v.*). They may, however, be due to enlargement or displacement of the *spleen* and *liver* (*q. v.*), or to tumors of the retroperitoneal glands. On the left side the possibility of *perigastric abscess* must be borne in mind, as sometimes the dulness and increased tension of the tumor extend as far down as the lumbar region.

Palpation and Percussion about the Centre of the Abdomen. *Umbilical hernia, cancers of the stomach, liver, and intestine, sarcoma of the retroperitoneal glands, hydatid cysts of the liver,* and *tumors of the gall-bladder,* together with *floating kidney, spleen,* and *liver,* all at times cause tumors which may be felt in this region. They must be distinguished from each other by methods already referred to under the organs named. The general principle upon which to proceed is to endeavor, by palpation and percussion, to discover the organ to which the tumor belongs. To this end careful inquiry should be made as to the time the tumor has been known to exist; its effect, if any,

upon the general health ; its effect upon the function of the possible organs affected, and particularly as to the presence or absence of vomiting, constipation, diarrhœa, or jaundice.

Tumor in the region about the umbilicus may be from *tubercular disease of the mesenteric glands* (*tabes mesenterica*). It occurs nearly always in children, and presents the physical signs and symptoms of tubercular peritonitis, with the addition that enlarged mesenteric glands may sometimes be felt. Children grow pale and anæmic, waste away, have apparently causeless diarrhœa, the passages being foul and the food undigested. The abdomen is large, but appears larger when compared with the emaciated body. It is tender, its walls are thickened and less elastic than normal. Signs of tubercular disease in other organs may be detected.

Facts gathered in this way, carefully analyzed, and then studied with reference to the physical properties of the tumor (hard or soft, fluctuating, doughy, or not), will generally suffice for a probable diagnosis. A positive diagnosis often cannot be made at the first examination, and sometimes is possible only after watching the progress of the case for a considerable time.

Enteroptosis.

It is by inspection, palpation, percussion, and auscultation that we discover the anatomical cause for the symptom-group about to be described. Attention to this affection may only be called by the subjective symptoms.

This disease or physical condition, called sometimes Glénard's disease, after the physician who first called attention, in 1885, to its existence, has received, of late, much study. It is characterized by the falling down or descent of a number of the abdominal organs. This occurs on account of relaxation of the supporting ligaments, the number of which Glénard puts at six. This relaxation is largely due to a flabbiness and hence lack of support of the abdominal wall ; or to strain from undue physical exertion ; or to the abuse of cathartics ; or possibly to injury. It is far more common in females who have borne children. It may be the result of feeble muscle-tone, following prolonged illness. The degree of descent, and hence the severity of the symptoms, may vary from slight displacement of one or two organs to that of the large intestine, the stomach, the liver, the spleen, and the right kidney (sometimes both). In moderate cases but two of the ligaments are relaxed—the ligamentum cŏlico-hepaticum and the ligamentum gastro-colicum ; in the more severe all are affected.

Symptoms. The *objective* symptoms are due to the slight displacement, and are either purely physical or arise from the alteration of the function of the stomach and the intestines.

The *subjective* symptoms are due to the same cause. The displacement gives rise to local symptoms of *weight, heaviness*, and abdominal distress, amounting in some instances to *pain*, especially when in the upright position, and to protracted and pronounced *neurasthenia*. Later, we have the subjective symptoms of *dyspepsia, gastritis, gastric*

dilatation, and *intestinal atony*, while the neurasthenic symptoms grow more aggravated.

The earliest objective symptoms are: (1) Pulsation of the abdominal aorta; (2) a linear tumor or band about midway between the xiphoid cartilage and the umbilicus, extending transversely from four to six inches in length; (3) gastroptosis, or descent of the stomach; (4) movable right kidney. Later, the liver may fall from one to four inches, the spleen become palpable, and the left kidney movable. The transverse tumor above mentioned was held by Glénard to be the thickened transverse colon. Ewald, however, seems to have demonstrated that it is the pancreas. The displacement of the viscera is recognized by the methods previously detailed for physical examination of the various organs. The patient must always be examined in the erect as well as in the recumbent position. Care must be taken to distinguish gastric dilatation from gastric descent. This can be done by careful percussion after inflation with air, by gastric diaphany, by measurement with a sound, and with fluids. Glénard laid much stress upon the *splashing sound*. This may or may not be present; it may be of gastric or intestinal origin, usually the former. It does not depend upon the displacement as much as upon the occurrence of gastric dilatation. It occurs in other affections.

An objective sign of diagnostic value, attention to which has been called by Treves, is the relief the patient experiences when the lower half of the abdomen is supported by a belt or by the hands of the patient or surgeon, when in the upright position.

The objective signs of gastric origin depend upon functional or organic disease of that organ. We may have, on the one hand, only the perverted gastric secretion and digestion that go with gastric neuroses; on the other hand, we may have the perverted gastric secretion of gastritis, gastric atrophy, or dilatation, and the evidences of diminished digestive, motor, and absorptive power of these affections.

The *subjective* symptoms also depend upon the functional or organic changes in the stomach and intestines, upon the displacement of the organs, with or without the above, or upon the associate physical muscular condition of the individual and the state of the nervous system.

Glénard divided the progress of the subjective symptoms into three periods:

In the *first* there is gastric atony, when the patient experiences weight and burning after eating; a short period of wakefulness about two o'clock A.M.; a loose stool in the morning; loss of strength.

In the *second* period the patient cannot eat fats and starches, and the subjective symptoms arise late in the period of digestion. A dragging sensation or a feeling of emptiness occurs about three hours after meals. The patient awakens at two o'clock A.M., and remains awake for two or three hours. Constipation, at times alternating with diarrhœa, is present. There is continued loss of strength, and a tired feeling is complained of on rising.

In the *third* period the symptoms of neurasthenia are most pronounced. The patient is emaciated, and complains of a constant weight and of cramps in the stomach. Constipation is obstinate, and the stools

are scybalous and mucous. The patient is much prostrated and suffers from sleeplessness. The constipation and the intestinal distress are aggravated by aperients. Enemata must be resorted to, to relieve the symptoms. Intestinal catarrh or membranous enteritis is very likely to follow.

Pain throughout the abdomen, especially when walking about or in the erect posture, is frequently complained of. Some authorities speak of tenderness on pressure over the solar plexus and of tender points along the vertebra.

The disease is overlooked and the symptoms are attributed to neurasthenia. It is often difficult to estimate which of the two preponderates.

Diseases of the Peritoneum. Peritonitis.

Inflammation of the peritoneum may be acute or chronic. It may be general or localized. Acute inflammation is rarely primary; it may occur in the later stages of chronic Bright's disease, or other dyscrasia, as a terminal infection. If it follows exposure to cold, or trauma, it is called traumatic peritonitis. It is due in the large majority of cases to extension from organs which the peritoneum covers, or to perforation of one of the abdominal organs. In the first instance it may follow inflammation of any portion of the gastro-intestinal tract, of the pelvic viscera, and suppurative inflammation of the spleen and liver and of the pancreas.

Peritonitis an Infection. In all instances the primary inflammation in the organs mentioned is due to some micro-organism, as the staphylococcus, the streptococcus, or the bacillus coli communis, and the peritoneal inflammation to subsequent extension of the infection. In peritonitis from perforation the element of infection is also the most important part in the process, as in ulcer of the stomach or bowels. In inflammation of the gall-bladder perforation may take place, with resulting peritonitis. Abscess in the liver, spleen, or kidneys, bursting into the peritoneum, also leads to general peritonitis. The most common forms, however, are due to appendicitis or disease of the Fallopian tubes. Acute peritonitis may also occur in tuberculosis and in other systemic infections by direct infection.

Symptoms. The onset of acute peritonitis depends in a measure upon the cause. When there is perforation the onset is sudden. Chilly feelings or a rigor occur, with intense pain in the abdomen. The pain is at first localized, but rapidly becomes general. It is constant, increases in exacerbations, and is very intense, aggravated by movements and by pressure. The patient lies on the back with the legs drawn up. The dorsal decubitus is assumed, in order that the tension of the abdominal muscles may be relieved. The location of the pain depends upon the seat of primary infection; this is usually in the right or left lower quadrant, more marked about the tubes or the appendix. In perforation of an ulcer of the stomach the pain may be located in the back, or in the chest or shoulders.

Physical Examination. On palpation the abdomen is extremely sensitive. The patient is unable to bear the weight of clothing or ex-

ternal applications. The abdomen gradually becomes distended, and is tympanitic on percussion. The distention may become so great as to push up the diaphragm and interfere with the respirations, so that they are shallow, and may dislocate the heart, so that the apex-beat is seen in the fourth interspace. The splenic dulness may be obliterated entirely and the liver-dulness reduced. It is said that in some instances this may be obliterated, although recent observations show that such obliteration only occurs in the anterior portion of the abdomen. Liver-dulness persists in the axillary region, though diminished in extent. This obliteration could only take place in perforative peritonitis. Osler points out that in pneumoperitoneum from perforation the anterior hepatic dulness may be obliterated, although dulness in the lateral region continues, on account of the effusion of fluid. If a patient with gas in the peritoneum is turned on the left side, a clear note is heard beneath the seventh and eighth ribs (hepatic region). The abdominal muscles are more or less rigidly contracted. Spasm of the muscle over the seat of primary inflammation takes place at once, and is a valuable indication of the origin of the infection. In some cases, usually when the inflammation is due to the streptococcus, there is not much distention of the abdomen, or it may be flattened entirely with board-like rigidity. In these instances pain is not so marked, and tenderness may not be complained of.

The *respirations* are hurried and the superior thoracic type of breathing is seen, because the action of the diaphragm is painful. The act of speaking or coughing increases the pain, and the patients are unable to take a full breath without suffering. With the occurrence of pain and local signs *vomiting* usually sets in. It is painful and at first is complete, the contents of the stomach being ejected and then a yellowish bile-stained fluid; later, the vomit becomes greenish in color. Complete vomiting is replaced by simple regurgitation of fluid, so that on the slightest motion of the patient, or on taking a small amount of fluid, the characteristic greenish-colored fluid is regurgitated without action of the diaphragm. This may be almost continuous for from twenty-four to forty-eight hours. The *tongue* is at first furred, but later becomes dry, and often is cracked and red. The *bowels* are constipated. They may be loose at first, but constipation is characteristic. The intestines are paralyzed from overdistention and from œdema of the walls due to inflammation.

The general symptoms are marked. After the chill the temperature rises to 104° or 105°. In septic cases it continues at this point, or may rise to a greater height. If cases progress rapidly, a temperature of 105° or 106° on the second or third day is not uncommon. In other cases after the initial rise the subsequent elevation is not so great, but there is not much difference between morning and evening temperature unless there is an abscess.

The *urine* is scanty; micturition may be frequent and painful, particularly if the inflammation began in the pelvic organs. The urine usually contains a large amount of indican in the suppurative form.

The appearance of the patient at the height of the disease is characteristic. The expression is anxious, the face is pinched, the eyes

sunken. Vomiting causes wasting. The collapse is marked, with the characteristic facies previously described. (See Expression.) The pulse is rapid and feeble and soon becomes thready, ranging from 110 to 150. In the first stages it may be small and hard. Attention has been called frequently to the peculiar wiry pulse of the early stage of peritonitis.

In severe cases death may take place in from thirty-six to forty-eight hours. Usually a fatal termination does not take place for five or six days, and may be delayed longer. The vomiting persists, collapse with falling temperature ensues, the pulse becomes rapid and thready. Throughout the entire attack, unless symptoms of septicæmia are marked, the mind is clear. The patient dies of paralysis of the heart. Septicæmic symptoms are indicated by a dusky color of the face, rapid and irregular pulse, slight delirium, dry, brown tongue, and other evidences of the typhoid state.

If the cases are prolonged, some effusion may take place into the peritoneal cavity. Dulness is noted in the flank, and if it is possible to move the patient, it alters with the position. If recovery takes place, particularly in tuberculous cases, the affection may become circumscribed and be indicated by dulness, which is not movable.

Diagnosis. It is essential in making a diagnosis to ascertain, if possible, the primary source of the infection or inflammation. To determine this we inquire the age, sex, and history of previous disease of the patient. In young male adults appendicitis is first to be thought of; in females inflammation of the pelvic organs. In chlorotic subjects, if the pain is high up, a history of ulcer of the stomach must be inquired for. Later in life, particularly if there has been jaundice, the possible history of frequent attacks of gallstones and of hepatic disturbances must be ascertained. All forms of intestinal obstruction must be sought for. Frequently, however, a definite cause cannot be ascertained. If it occurs in the course of typhoid fever, it is usually due to perforation, but the occurrence of pain may not be complained of, on account of the mental state of the patient. Under other circumstances the symptoms cannot be overlooked.

Acute peritonitis must be distinguished from *entero-colitis.* The distinction is not usually difficult if attention is paid to the development of the case. The pain is not so severe in entero-colitis; it is more colicky in character. The general tenderness is not so great as in peritonitis, and the distention does not interfere with respiration to such a marked degree. Diarrhœa is more common in entero-colitis; collapse, if present, is not so pronounced.

Acute hemorrhagic pancreatitis may simulate peritonitis in the sudden intensity of pain and the occurrence of shock.

The diagnosis from *obstruction of the bowel* is difficult in the absence of a distinct history, but in peritonitis we do not have stercoraceous vomiting until late. The tympanites and the pain are more general. Peritonitis frequently accompanies or is due to obstruction. A tumor, if present, may point to the true nature of the case, and, if there is any discharge from the rectum, invagination may be the exciting cause.

Peritonitis is simulated by a condition to which the name *hysterical peritonitis* has been applied. It occurs in hysterical subjects, and every feature of the true form is imitated. The mode of onset, the decubitus, the difficulty in micturition, and the local distention and tenderness of the abdomen are characteristic of both. In a few cases which we have seen the vomiting is not of the nature of true peritonitis, either in the mode of ejection or the character of the fluid. It must not be forgotten that even the temperature may be elevated and collapse take place in the hysterical form. In the cases which I have seen the abdominal facies does not develop, while, on the other hand, the facies of hysteria, with the self-interest which the patient exhibits, and the precision with which symptoms are narrated, coupled with emotional or other manifestations of hysteria, point to the true nature of the affection. Other symptoms of hysteria may arise. The case is judged by the history of these associated manifestations and the permanent stigmata of the disease. There is always a positive absence of cause and of disease in any of the abdominal viscera. Sometimes, in these cases, if the attention of the patient is diverted, the tenderness on pressure may not be complained of. I am not familiar with the results of examination of the urine in this form of peritonitis. Indican should not necessarily be increased, as we find it to be in acute suppurative peritonitis.

Rheumatism of the Abdominal Walls. There is absence of a history of sudden acute pain followed by general pain. The fever is not so great. The respirations are not interfered with, the pulse is not so rapid, and symptoms of collapse do not supervene. A rheumatic pharyngitis or inflammation of muscles in some other portion of the body may occur simultaneously.

Local Circumscribed Peritonitis. The causes of localized peritonitis are those of general peritonitis—that is, extension of inflammation from neighboring viscera, or perforation of the viscera. In the latter instance the inflammation does not become general, if rapid local inflammation shuts off the perforated area from the general cavity of the peritoneum. Local peritonitis of mild degree, and local or circumscribed peritonitis with suppuration, are therefore found in the regions previously indicated, from which a general peritonitis may develop. The inflammation, however, if retained by a limiting wall, may, after suppuration has taken place, gradually extend and the pus burrow in various directions. In such cases of localized peritonitis as may exist in the upper half of the abdomen, a *sub-diaphragmatic abscess* may form, or an abscess containing air and pus, known as pyopneumothorax subphrenicus. If the inflammation is secondary to disease of the pancreas, it may be limited to the lesser peritoneum and cause the physical signs of effusion in this cavity. (See Disease of the Pancreas.) Sub-diaphragmatic abscess is not limited to the lesser peritoneum. It can only be recognized by the history of the previous disease, which may cause perforation, and by the general symptoms of abscess. If the abscess is on the left side, there is an extension of dulness upward toward the scapula, the lower limit of the lungs in health ceasing at the eighth or ninth interspace. There may also be

dulness in the axillary region. If the abscess is on the right side, it may simulate enlargement of the liver, and be characterized by marked increase in dulness anteriorly, laterally, or posteriorly. Localized peritonitis in the lower half of the abdomen is due to disease of the vermiform appendix, or to disease of the Fallopian tubes. The localized signs are, first, those of pain and tenderness; second, the development of tumor.

Chronic Peritonitis. The symptoms of diffuse peritonitis, chronic in course, may follow the acute, or may occur in the course of tuberculosis. The intestines and peritoneum are matted together. General pain and tenderness, with a prolonged period of ill health, attend the diffuse form. (See Tuberculous Peritonitis.) In the chronic forms, if there is considerable fibrous proliferation, even though not cancerous or tuberculous, the abdomen becomes retracted, the muscles rigid, the note over the abdomen modified or dull tympanitic. The modification may be detected in the upper half of the abdomen particularly, and especially over the liver. Sometimes a fremitus can be felt. The patients are under weight and without strength. The pain may continue a long time. It finally results, at least clinically, in such compensation that the patient is able to continue his usual occupation. Localized bands form, and may cause local sensations of a dragging character, or pain with drawing or pulling sensations; but, save the local symptoms, these are not serious, unless it should happen, as has been seen in intestinal obstruction, that coils of intestine are twisted about the bands or caught in them, thus leading to obstruction.

Cancer of the Peritoneum.

It usually occurs in the aged, and follows cancer in other organs, as the stomach, liver, or uterus. Occasionally it is primary. The omentum is indurated, and forms a mass which lies transversely across the abdomen in the upper zone. Ascites usually develops, and the exudation is bloody. The disease occurs more frequently in women than in men. With the development of ascites there is emaciation. The surface of the indurated omentum is irregular. It may be painful on pressure. A tumor of the same physical character is seen in tuberculous peritonitis, and I have seen several such tumors in the aged without apparent cause, unless from proliferative peritonitis. (See Tumor.) Progressive emaciation, chronic ascites without cause, and a localized tumor without the occurrence of fever point to the probable nature of the case. Sometimes pain is the most pronounced symptom. If these symptoms are present with signs of cancer in some other organs, as the stomach, rectum, or uterus, there is probably primary cancer of the peritoneum.

Retroperitoneal sarcoma, or Lobstein's cancer, is central or lateral, deep-seated, and usually fixed. It is accompanied by the general symptoms of cancer and by ascites. The growth is very large. It can be detected above the sacrum by rectal examination. The intestines are in front of the growth, causing an unusual sensation to the hand, as in

Burrow's case, like a hydatid fremitus. Lockwood's cases were believed to be solid ovarian tumors.

Sarcoma of the glands and the tissues in the above mentioned space is, according to J. Dutton Steele, slightly more common in males than in females; more common in the first decade or after the fiftieth year. Its duration is about nine months. Of the sixty-five cases collected by Steele 39 per cent. were spindle-celled sarcoma, 34 per cent. round-celled sarcoma, 14 per cent. lympho-sarcoma, and 13 per cent. were mixed cases. The growth originates in the lymph glands or in fibrous connective tissue about the kidney, the spinal column, the pelvis, or the sheaths of the bloodvessels. The onset is insidious. The first symptoms are the presence of a tumor, or the effects of pressure upon the vessels, nerves, or viscera of the abdominal cavity; they depend upon the site of the tumor. Varicocele is of frequent occurrence. It is often impossible to distinguish it from malignant disease of the kidney or of the suprarenal bodies. The diagnostic features are (*a*) the rapid growth; (*b*) the position of the colon, which is pushed in front of it, particularly if the tumor is lateral; (*c*) the pressure symptoms; (*d*) the tumor may move with respiration or independently, and may fluctuate.

Tuberculosis of the Peritoneum.

The tuberculous process in the peritoneum may be either acute or chronic. In rare instances it may continue without any symptoms, either local or general.

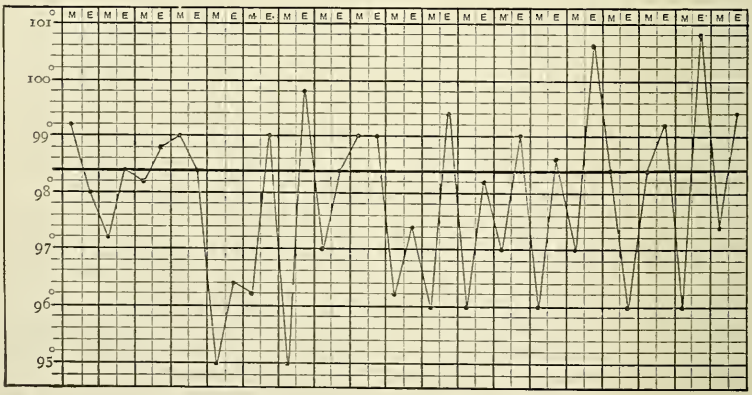
Acute tuberculous peritonitis may exactly simulate suppurative peritonitis, although usually the course is more prolonged and the fluctuations of temperature less pronounced. In other respects, it cannot be distinguished from acute general peritonitis, save by the absence of the causes of the latter. A history of exposure to tuberculous infection, or the presence of tuberculosis in some other portion of the body, may be of service in determining the nature of the case. Often there occurs in a short time associate tuberculosis of other serous membranes, so that tuberculous pleurisy or tuberculous pericarditis will supervene, an associate process which does not take place in ordinary peritonitis. There is diarrhœa in most cases—at least it has been present in the few instances which I have seen of this form of tuberculosis. Nevertheless, the diagnosis is sometimes impossible. Henry has called renewed attention to the occurrence of inflammation about the navel as a sign of tuberculous peritonitis. He believes the periumbilical erythema is pathognomonic of the affection.

Acute tuberculosis of the peritoneum may precisely simulate appendicitis in, first, the local symptoms and signs; and, second, the subsequent infection of the peritoneum. In acute tuberculous appendicitis, however, the signs of a tumor are not so marked as in true appendicitis. Nevertheless, in one instance, Keen operated upon a patient of mine, a healthy laborer in a rolling-mill, who had the classical symptoms of appendicitis. At the operation the appendix was found to be perforated and hanging in a local abscess. A fecal fistula ensued which did not heal, and within two months the patient died of general

tuberculosis. The appendix was the seat of primary tuberculous ulceration. In a second instance the appendicitis arose in the course of tuberculosis.

In a third instance the patient, aged forty-five years, was admitted to my wards in the Philadelphia Hospital, with high fever and pain in the abdomen, at first more marked along the margin of the liver. By the end of twenty-four hours it became more decided in the right lower quadrant of the abdomen ; tenderness at McBurney's point was distinct ; the area was enlarged and dull on percussion, the surface slightly œdematous. Fluctuation could not be detected. Extension of the leg was painful. Rapid general peritonitis ensued, during which the surgeon saw him, but declined to operate until the subsidence of the attack. When the attack subsided the local signs of tumor were not present. The fever persisted irregularly for a short time, while the more acute peritoneal symptoms subsided ; then the right pleura became infected, and cough ensued with expectoration of mucopurulent fluid. It did not contain tubercle bacilli, however. Subsequently the left pleura and the pericardium became involved. During the entire course of the disease there were diarrhœa, most pronounced sweats, rapid emaciation, and exhaustion. Death took place at the end of five weeks, and at the autopsy general serous tuberculosis was found.

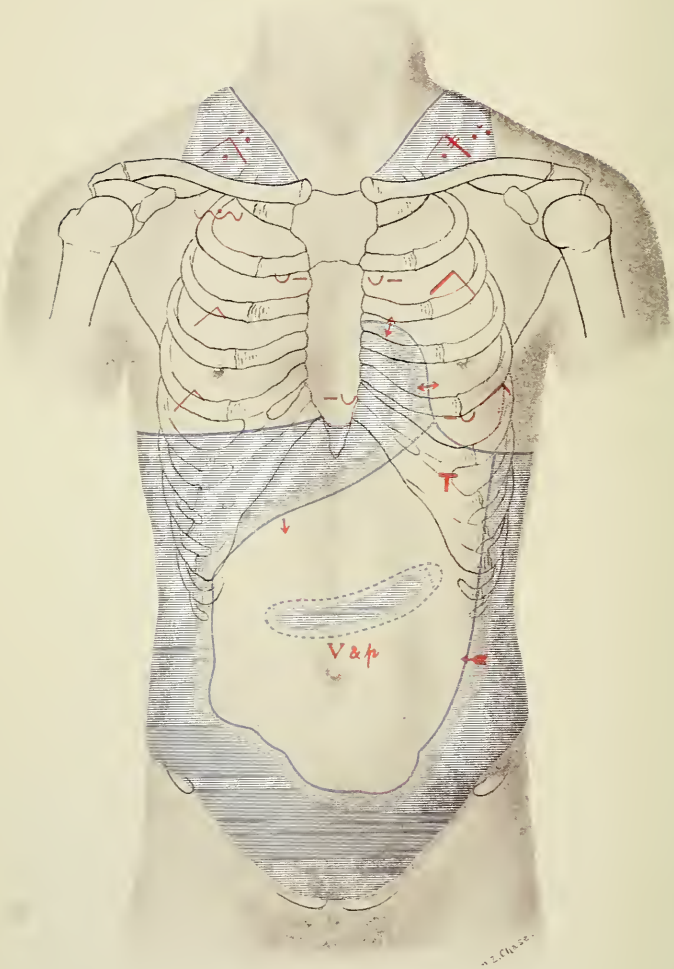
Fig. 193.



Tuberculous peritonitis. Subnormal temperature.

While in a number of instances the symptoms are acute and alarming, in the larger proportion of cases the process is more *chronic*, and is attended by characteristic local and general symptoms. In the prolonged and moderate cases there may be continued *fever* of moderate degree, or it may be remitting in type. In old people the temperature is frequently subnormal. (See Fig. 193.) There is more or less rapid *emaciation*. The *sweating* is profuse and characteristic. The fever is high but irregular in type, in more severe cases approaching the remittent form. The general symptoms resemble typhoid fever. Indeed, symptoms of the typhoid state may ensue.

PLATE XXXVII.



Tuberculosis of the Peritoneum.

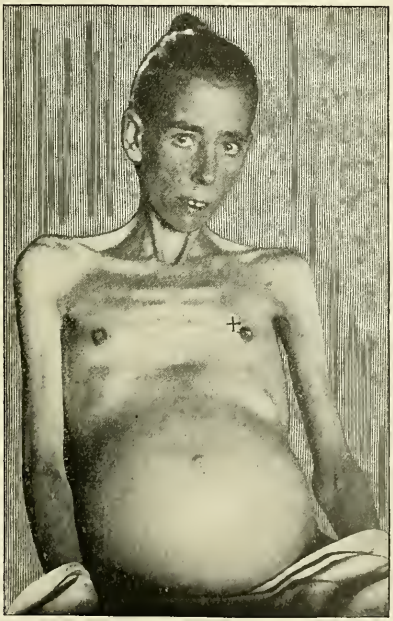
Abdominal exudate (not freely movable); omental tumor. Consolidation at apices.

The Local Symptoms. Four classes are seen : (1) Abdominal enlargement with effusion ; (2) enlargement with tumors ; (3) a combination of the two ; (4) enlargement without evidence of fluid or tumor in the abdomen. In the latter form and in the forms in which tumors are present the abdomen subsequently may undergo retraction.

1. *Enlargement with Effusion.* The local symptoms and physical signs are those of ascites. The abdomen is never as distended, however, as in the ascites of cirrhosis of the liver. Often the fluid is confined by adhesions which may distinctly localize it in the right or left quadrant of the abdomen, in which situation fulness and fluctuation may be readily detected.

2. *Tuberculosis with Tumors.* (Plate XXXVII.) The tumors are usually in the upper zone of the abdomen, and may be localized in either quadrant, or extend from the right to the left. They are usually due

FIG. 194.



Tuberculous peritonitis ; pulmonary tuberculosis. + The site of cardiac impulse.

to tuberculosis of the omentum, with secondary contraction. In some instances a hard, indurated tumor, somewhat tender on pressure, may extend across the abdomen midway between the xiphoid cartilage and the umbilicus. It may be as low as the umbilicus, and vary from two to four inches in width. It may be continuous with the liver-dulness. In other instances more distinctly localized masses may be felt. These may be to the right or to the left of the umbilicus. In other instances they are hard, slightly tender, with an irregular surface. They may be movable and vary with the position of the patient. I have

never seen tuberculous masses in the lower quadrants. In children with tabes mesenterica they may be made out close to the vertebral column in the median line, extending to the brim of the pelvis, although at the lower portion they are not so distinct. The dulness over the tumors is varying, depending upon the relation to the bowels and the degree of intestinal distention. Instead of dulness a modified tympany may be observed, or muffled resonance.

3. *Cases in which Effusion and Tumors are Present at the Same Time.* These present symptoms common to the two conditions, although the tumors are not so distinctly defined.

4. *Absence of Effusion and Tumors.* When effusion and tumors are not present the thickened peritoneum and more dense intestinal walls lead to a modified dulness over the entire abdomen. When retraction takes place the resonance is of a woodeny character, the abdomen is more or less tender, and ill-defined indurations may be present. The term *carreau* is applied to this induration.

In not a few instances the local physical signs may apparently be due to inflammation of the liver, on account of extensive perihepatitis. In the case of a child under my care the local signs during life were of this character, and the symptoms were simply those of loss of appetite, with discomfort, weight, and fulness below the sternum. Both the right and left lobes of the liver were covered with an enormous thickening due to tuberculous inflammation. Simple plastic peritonitis occupied the lower zone.

Apart from the general symptoms and the local physical signs the other symptoms are not distinct save those due to tuberculosis in other situations. The appetite is usually poor, there is some atonic dyspepsia, vomiting may occur at regular intervals; the bowels may be constipated, although in my experience they have usually been relaxed. The patient becomes anæmic, the skin harsh and dry. Emaciation may progress to an extreme degree. Eruptions and boils may break out, some œdema of the ankles may occur. Death takes place from exhaustion and from the development of tuberculosis in other localities.

The *diagnosis* is difficult. Cases belonging to the first and fourth classes above mentioned probably present the greatest difficulties. The age modifies the difficulty of diagnosis. Peritoneal tumors, with or without effusion in young subjects, are almost always due to tuberculosis. In the aged they must be distinguished from carcinoma or chronic peritonitis from other causes. The association of diarrhœa with the symptoms is rather against carcinoma. Sacculated effusions may be confounded with abdominal tumors, as of the ovary. The resemblance is more pronounced if the tubercles develop primarily in the tubes or uterus. In a recent case the autopsy disclosed a large caseating ulcer inside of the uterus, and tuberculosis of the Fallopian tubes and peritoneum. The right tube was chiefly affected. The effusion during life was sacculated in the right lower quadrant, was not movable with the patient, and fluctuated both on external palpation and with bimanual palpation *per vaginam*. It was impossible to distinguish it except that there was dulness instead of resonance in the flanks. As Osler has pointed out, the association with salpingitis

must arouse suspicion, particularly if at the same time disease is found in some other organ of the body, as the apex of the lungs or the pleura. In males the primary lesion is often in the testicles. The history of the case and the development of the disease in an irregular manner, associated with gastro-intestinal disturbance rather than disturbance of uterine function, are points in favor of tuberculosis. Tympanites is of frequent occurrence.

Diseases of the Stomach.

The stomach is a canal in which the food is detained for the purpose of solution. Its walls are made up of mucous membrane, muscle, and peritoneum. It is richly supplied with bloodvessels. Because of its great functional activity it has an abundant nerve-supply. It is, moreover, surrounded by rich plexuses of sympathetic nerves, through which and its special nerve, the pneumogastric, it is in intimate relation with every organ of the body.

The Symptomatology. The local symptoms of disease of the stomach are dependent upon : (1) The morbid process which affects it ; (2) the effect of the process upon the anatomical structure of the organ (atrophy, dilatation, tumor), whereby the size is affected ; (3) the effect upon its function.

1. *The Morbid Process.* The symptoms due to the *morbid process* are not different from the symptoms of similar morbid processes elsewhere, save that they are modified by the function of the organ or its special construction as a canal. Hence, congestions are attended by discharge of mucus ; inflammations are attended by pain and by a flow of mucus and pus ; ulcers by pain and the accidents of ulceration (hemorrhage) ; malignant disease by pain and swelling (tumor), and its accidents, hemorrhage and obstruction ; while to each belong the general phenomena which attend it. But the stomach is highly sensitive and resents the intrusion of disease or of that which (1) causes disease or (2) irritates the affected part. Expression of this resentment is shown in hyperæsthetic symptoms (see the Neuroses), as *pain ;* in the abolition or derangement of function ; and in the great pathological reflex act of the stomach—*vomiting.* It will be seen later that this may be a symptom of every local morbid process of the organ, either directly because of the disease or of its exciting cause, both of which are operative in irritant inflammations ; or indirectly because the process has set up undue sensitiveness. In the latter instance any material, as food, which the stomach is accustomed to receive, becomes as much an irritant as mucus, pus, or blood.

2. *Anatomical Symptoms.* The morbid processes modify the anatomical structure and lead to other morbid conditions, as we see when dilatation succeeds inflammation or obstruction of the orifices. The symptoms of the secondary conditions are the same as elsewhere—in atrophy, diminution in size ; in dilatation, increase in size, with retention and fermentation, and finally discharge of the contents by vomiting.

Nerve Mechanism. In the consideration of the symptomatology of gastric diseases the anatomical relation, by its vascular and nervous

connection, must be considered. The student is sufficiently familiar with physiology and pathology to know that each organ has a representative in the central nerve-mass, the brain, and that disease in one organ will influence the function and create morbid symptoms in another which is related to it through intimate nervous connections.

The central representative or centre is influential in proportion to the power and activity of its peripheral adjunct. It is, moreover, influenced by higher centres, the psychical, and it in turn modifies them. It influences or modifies lower centres, (1) functional, (2) vasomotor, (3) motor, or (4) sensory. The result of this mechanism is: 1. That functional alteration or organic disease of (*a*) the gastric centre, or (*b*) of centres of higher control, or (*c*) of the nerve that connects the centre and the organ—pneumogastric nerve—produces gastric symptoms. 2. That gastric diseases produce symptoms in other organs, as cardiac palpitation (reflex). 3. That disease of other organs produces gastric symptoms or disease, as the vomiting of pregnancy, or of renal calculus, or of disease of the testicle, or the gastritis of nephritis. Thus vomiting is caused by emotion (high centre), influencing the pneumogastric (lower centre); by a tumor pressing or destroying the pneumogastric centre; or by a tumor, as aneurism, pressing on the pneumogastric nerve. I have taken the simplest illustration. When we come to the study of gastric neuroses the extraordinary influences of the nervous mechanism will be appreciated; or, when hysteria is studied, the physiology of its extreme gastric symptoms will be recognized. When the mechanism and clinical course of vomiting are studied it will be found among other causes to be frequently due to affections of the blood, the poisons of which irritate cerebral centres or nerve plexuses in the stomach.

Vascular Mechanism. But gastric diseases also arise because of the *vascular supply.* Thus in heart disease with venous stasis the gastric veins become the seat of congestion, with consequent gastric catarrh; or hepatic disease will cause portal congestion and gastric catarrh.

3. *Functional Symptoms.* Any local disease of the stomach must influence its function; therefore, conversely, functional symptoms must be present in all local diseases. But functional disorder may be present without local anatomical change; the impairment is nearly always induced through the influences of the nervous system. The functions of the stomach are to digest and to absorb the products of digestion. The former function is motor and chemical, the completeness of which depends upon mixture of the food with, and solution in, the gastric juice. The symptoms, therefore, must be due to changes (1) in the motor, (2) in the secretory, and (3) in the absorptive functions of the organ. The functions may be increased or diminished; the former are the primary and usually temporary aberrations; the latter succeed the former, and are permanent. The functional symptoms, therefore, are the symptoms of what we know as *indigestion* or *dyspepsia.* They are described in the account of the subjective symptoms and also in the section on Gastric Neuroses.

Toxic Symptoms. The toxic symptoms arising in gastric disease are worthy of a few words. They are nervous symptoms due to the

absorption of ptomaïnes or imperfect products of assimilation. If absorption of the toxines takes place suddenly and in large amounts, coma and convulsions occur (Kussmaul's symptom); or, if gradually, hypochondriasis, melancholia, mental depression, with vasomotor phenomena of various kinds, arise.

It is observed, therefore, in unravelling the symptomatology of gastric disease, that we must first note: (*A*) The subjective symptoms due (1) to morbid processes, (2) to alterations of function, (3) to alterations of size (sense of fulness, etc.). (*B*) The objective symptoms due (1) to morbid processes, (2) to alterations of function, (3) to alterations of size.

Diagnosis from Disease of Contiguous Organs Functionally Related. The student will soon learn that diseases of the stomach which are functional in character cannot be differentiated with ease from diseases in other organs functionally related. He will find that to draw hard-and-fast lines between gastric and intestinal indigestion, or between so-called disordered gastric and hepatic function, is generally impossible. Organs which are closely related in physiological function, and which have nerve-supply and blood-supply in common, cannot be differentiated when disordered function is considered. Hence, indigestion and biliousness, or simple acute gastritis and duodenitis, are beyond the pale of close discrimination. In fact, the symptoms of each blend in a manner.

In addition to the examination of the stomach, in order to judge correctly of the nature of gastric lesions, as may be inferred from what has been written above, we must ascertain (1) whether the gastric symptoms are dependent upon disease of other organs—particularly the eye, nose, and genitalia, the heart and kidneys—by an examination of each organ; and (2) whether other symptoms are created by gastric disease.

The Stomach in Other Diseases. Diseases of the stomach may frequently mask other diseases; in other words, patients will complain of gastric symptoms which are but concomitant phenomena, behind which there are graver conditions. Thus, in disease of the kidney, in phthisis, in chronic bronchitis, in emphysema, in valvular disease of the heart, catarrh of the mucous membrane of the stomach is of frequent occurrence, depending upon the primary disease.

In *tuberculosis* the local gastric symptoms often are the more prominent features. Thus in the earlier stages of phthisis loss of appetite and vomiting are of constant occurrence. The dyspeptic symptoms in a large number of cases precede the pulmonary symptoms, and may be so pronounced as to mask them entirely. The patients are usually delicate and anæmic; they complain of loss of appetite and mild indigestion; there is some regurgitation of food; they are feeble and languid. They are treated for chronic catarrhal gastritis, but do not improve. On examination of the lungs the physician is surprised to find a small area of consolidation, and upon inquiry will find subjective symptoms of tuberculosis to have been present for a considerable time. Every practitioner is familiar with the scores of patients with phthisis, even when the disease is far advanced, who believe that their symptoms are entirely due to disorder of the stomach. In addition

to the early catarrh that precedes tuberculosis, other gastric symptoms may occur. The well-known association of simple ulcer and phthisis is familiar. Both occur at the same time of life, yet the gastric symptoms may prevent investigation into those of pulmonary origin. In *anæmia* and *chlorosis* changes in the digestive tract are common. On account of the general blood-condition the functions of the stomach are impaired. Here, too, we frequently have the association of ulcer with the general condition. Danger of overlooking either is not so great as in tuberculosis.

In *valvular affections of the heart*, chronic catarrh of the stomach may result from venous congestion. The symptoms may point to the gastric condition alone. In all cases of chronic gastric catarrh it is necessary to examine carefully into the condition of the heart. Over and over again patients apply for treatment not on account of cardiac symptoms, but because of gastric disorder. They will be treated in vain unless the primary cardiac affection is ascertained. Many cases of gastric catarrh have been cured by the use of digitalis. In disease of the *kidneys* the stomach is frequently involved. Vomiting and other symptoms of gastric disorder may occur long before dropsy or any objective sign which would lead to a correct diagnosis. The gastric symptoms are due to chronic uræmia. In other conditions of the genito-urinary tract gastric symptoms also occur. This is particularly noticeable in long-standing retention from chronic obstruction. Renal tumors may cause only disturbances of digestion, while gastric symptoms due to movable kidney are well known. The symptoms in the latter condition arise, first, from mechanical causes, as the pressure of the kidney on the pylorus, and, secondly, from the influence on the nervous system.

Disease of the Liver. The intimate relationship of the liver and the stomach is such that when one is the seat of serious functional disturbance the other is likely to be affected. Frequently it is impossible to draw fast lines as to which organ is the primary seat of disorder. The abuse of alcohol frequently induces chronic gastritis, and also causes cirrhosis of the liver. On the other hand, cirrhosis of the liver is frequently the cause of chronic gastritis secondary to the portal congestion.

Diseases of the Nervous System. The relationship of disease of the central nervous system to disturbance of the gastric functions has frequently been adverted to. (See Vomiting.) In sclerosis of the posterior columns of the cord this is more striking than in any other spinal disease. Not only do we have gastralgia and gastric crises, but moderate symptoms of indigestion, with hyperæsthesia and slight gastralgia, may be the first symptoms of locomotor ataxia.

Diabetes. Diabetes may continue (in its course) for a long period of time, during which the patient is thought to have stomach-trouble, before an examination of the urine reveals the true nature of the case.

Opinions differ as to the relationship of *gout* and *rheumatism* to gastric disorder. Some writers believe that a specific gouty inflammation of the stomach, due to the uric-acid diathesis, is of frequent occurrence, and that one of the prominent manifestations of gout is dyspepsia in all its forms. The French consider gastric disturbances to be frequent

expressions of the rheumatic diathesis. The relationship of the two, however, is thus far not fully developed, although, in these conditions, it is not usual to overlook the presence of either of the diatheses when symptoms of gastric disturbance occur. It is essential to bear in mind that in persons of a rheumatic or gouty diathesis gastric disturbances are more likely to occur than in healthy individuals; their successful management depends upon the recognition of the fundamental diathesis. It is more than probable that gastric disorders, along with defective metabolism, is primary in both affections.

The Data Obtained by Inquiry.

The Social History. In no other group of diseases than in those about to be considered, unless those of the nervous system, is it more important to inquire into the social history. This is true, because most of the so-called gastric disorders have their foundation in neurasthenic states, the probability of which, of course, must be carefully sifted from the many possibilities. *Age.* Early age predisposes notably to gastro-intestinal disorder. In later life the catarrhs which arise from improper exposure or indiscretions in eating or occupation are common. The menopause is often associated with gastric disorders. The *sex* is not of great diagnostic significance, except from its relationship to the excesses in eating and drinking of one class. Those *occupations* which prevent out-door exercise, or which compel exposure to toxic substances, or require stooping or constrained positions, or overtax the eyes, invite gastric diseases. *Habits* of eating and drinking, both as to time and mode of eating, and the character of food and drink, must be brought out in the inquiry. The use of tobacco and other stimulants and narcotics must be noted. The hours devoted to vacation and work are to be learned, as fatigue bears a great part in gastric disease.

The Family History. Heredity plays but a small part except in gastric carcinoma and in gastric neurasthenia.

The History of Previous Disease. The occurrence of infectious diseases antecedent to the gastric disorder must be inquired about, for, either because of the attendant gastritis or of the resulting defective innervation, they predispose to gastric disease. The excessive feeding in the convalescence of typhoid fever, it seems to the writer, is frequently the cause of gastric dilatation. Any prolonged illness which weakens the muscular system and lowers the tone of the nervous system will be likely to cause gastric disease.

It will be learned elsewhere that gastric affections occur secondary to many local diseases, as of the heart, the lungs, and the kidneys. Inquiry as well as an objective investigation must be made, to determine the presence of possible primary diseases. Disorders which interfere with the mechanical support to the intra-abdominal organs must be inquired for. Pregnancy, antecedent ascites, or a large tumor may so weaken the abdominal muscles as to lead to gastro-enteroptosis. Finally, a history of the ingestion of corrosive poisons must be sought for in cases of gastritis.

It is very important to learn whether the patient has been subjected to the various causes of neurasthenia, which, with the history of the occurrence of neuropathic symptoms, make valuable data, pointing to the nature of many gastric neuroses.

The Subjective Symptoms. The following subjective symptoms may be complained of: Disorder of appetite, bad taste in the mouth, thirst, eructations, pyrosis, distress or weight after meals, burning after meals, flatulency, nausea, vomiting, constipation, diarrhœa, pain, vertigo, and cardiac palpitation. Nearly all the subjective symptoms are gastric neuroses, and will be detailed in the chapter devoted to the neuroses.

Bad Taste. It is usually due to acute catarrh. It may be present in chronic catarrh. It is said to be characteristic of the acute form of gastritis popularly known as biliousness.

Thirst. Thirst is not a symptom of gastric disorder alone; it is a symptom of diabetes and all conditions in which the body has lost fluids, as water by sweating, vomiting, or purging, or by evaporation and combustion (fever); or blood by hemorrhage. It is common in acute and chronic gastritis, particularly in the alcoholic form.

Distress, Weight, and Burning. They are frequent complaints, and may come on immediately after meals. They may be due to dyspepsia, hyperacidity, dilatation, bacterial fermentation, and flatulency. They exist in varying degrees, either singly or combined. (See Gastric Hyperæsthesia.)

Nausea. This symptom is usually associated with vomiting. In some persons it is impossible to excite vomiting, although they may suffer intolerably from nausea. Nausea is akin to vomiting in its mechanism and clinical associations (*q. v.*). It is a common incident in chronic interstitial nephritis. In old people, with arterial sclerosis and defective renal elimination, it is common. It may be due to irritating ingesta, to hyperacidity, to gastrectasia, or to toxins formed within the stomach.

Vomiting. Vomiting takes place when the stomach is compressed by the abdominal muscles and diaphragm, coincidently with relaxation of the so-called cardiac sphincter of the œsophagus. Sometimes there are nausea and violent efforts at expulsion on the part of the stomach, but no vomiting occurs, because the cardiac orifice of the stomach is not opened at the same time. Again, there may be profound relaxation of the œsophagus, but no compression of the stomach by the diaphragm and abdominal muscles. Both factors must operate at the same time to result in vomiting. This explains why it is that some persons suffer extreme nausea and have even violent retching, but are unable to vomit.

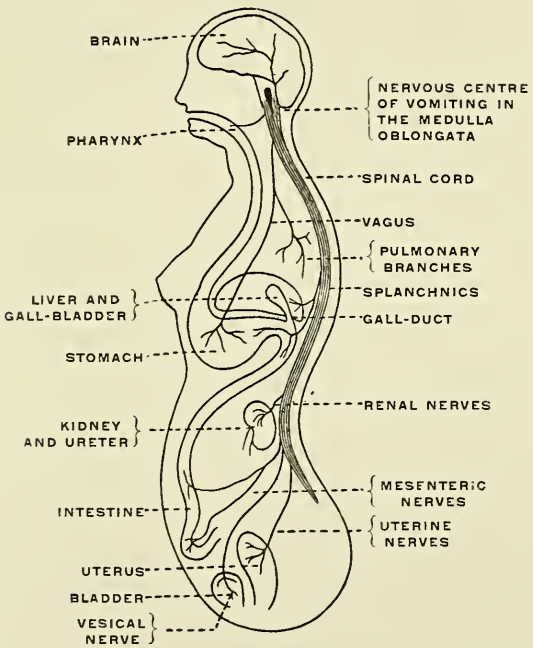
It is to modern physiologists—Schiff and Budge and Brunton—that we owe a correct explanation of the physiology of vomiting.

From them we learn that there is a nervous centre for vomiting, which is seated in the medulla oblongata, in close proximity to and intimately connected with the respiratory centre. It is to this centre that impressions are sent from the brain itself or from various portions of the body by their nerve-supply, and from this centre motor impulses are transmitted to the muscles concerned in the act of vomiting,

and to the stomach and œsophagus. In his usually graphic manner Brunton has described the entire mechanism.

By a very good diagram (see Fig. 195) the author indicates the afferent nerves which transmit impulses to the vomiting-centre, exciting it to action. They are: pharyngeal branches of the glossopharyngeal; pulmonary branches of the vagus; gastric branches of the vagus; gastric branches of the splanchnic; renal, mesenteric, uterine, ovarian, and vesical nerves. Fibres pass downward from the brain, conducting impressions to the vomiting-centre from the organs of special sense, from the brain-substance or its membranes when the seat of disease, or from central ganglia excited by emotion or imagination.

FIG. 195.



The nervous mechanism of vomiting.

From this it is seen that vomiting is a reflex act; that its mechanism is quite simple; and that a proper understanding of this mechanism is essential to a correct appreciation of its pathology and treatment. Reference has not been made to the vomiting that occurs in the initial stage of many fevers, and in septicæmia, uræmia and allied affections, and to the vomiting of hysteria. In the former it is doubtless due to the direct action of the poisoned blood on the centre, but it can also readily be seen to be due to the propagation of impulses to the centre from the brain that is irritated by the blood. If the phenomena of hysteria are due to an abeyance of the processes of inhibition, the occurrence of vomiting can be said to arise from the non-control, by

higher centres, of this centre. (From "Vomiting, Physiological and Clinical." *Trans. Penna. State Med. Soc.*, 1887. Musser.)

The significance of vomiting in a given case can sometimes be determined very readily, and sometimes it remains in doubt after very careful examination and questioning of the patient. In seeking for an explanation of vomiting it is of importance to find out the *previous* health of the patient; whether it occurred after the patient had been ill for a longer or shorter time, or suddenly, when he was in apparent health, or whether it formed one of the initial symptoms of an acute disease.

Again, inquiry should be made as to the supposed *cause* of the vomiting; whether it was excited by the taking of food, drink, or medicine, or by some disgusting sight or odor.

Further, the *time* of the occurrence of the vomiting should be ascertained, as well as its frequency, and whether preceded by nausea, pain (noting its locality), injury, coughing, jaundice, or constipation.

The *position* of the patient at the time the vomiting occurs sometimes furnishes a valuable clue to its cause.

The *effect* of the vomiting is sometimes of aid in diagnosis. In ulcer and migraine, for example, it affords marked relief.

Finally, the appearance and quantity of the matter vomited are very important. (See Objective Signs.)

Character. Vomiting may occur occasionally, persistently, or periodically. It may be projectile and painless, or difficult and painful. The former is characteristic of cerebral disease or reflex vomiting; the latter of local gastric disease. When vomiting occurs *suddenly*, without antecedent illness, it usually indicates some local affection of the stomach, or is due to some nervous impression, or marks the onset of some acute general disease.

Vomiting in Gastric Disease. The local affections of the stomach attended by vomiting are acute and chronic gastritis (especially the catarrhal form), dyspepsia, ulcer, cancer, and dilatation.

In *acute gastritis* there will be a history of an acute illness marked by severe local and general symptoms. The cause of the gastritis may be found to be overeating of highly seasoned or indigestible food; abuse of alcohol, narcotics, or sedatives; drinking water to which the patient is unaccustomed; poisoning with such drugs as arsenic and mercury; sudden changes in atmospheric conditions in susceptible persons. The vomiting is preceded by nausea, epigastric pain and tenderness, and often followed by profound prostration.

The vomited matters consist, first, of the contents of the stomach (which may throw light on the cause of the attack), then of mucus, saliva (which has been swallowed), bile, and, in grave cases, altered blood.

In *chronic gastritis* vomiting often occurs in from half an hour to an hour and a half after eating, the food being only partly digested and sometimes coated with mucus. It does not produce the prostration that vomiting in acute gastritis does, and is followed by some relief to the gastric uneasiness and pain. The emaciation may suggest cancer of the stomach.

In *ulcer of the stomach* vomiting is rarely absent. It occurs usually soon after taking food, and its occurrence affords relief to the gastric pain. There is nothing characteristic in the vomit unless it contains blood. Welch thinks that gastric hemorrhage in recognizable amount occurs in about one-third of the cases.

In *cancer of the stomach* vomiting is an almost constant symptom, but it may not occur until comparatively late in the disease, or, more rarely, may be one of the earliest symptoms. Usually it does not appear until dyspeptic symptoms have persisted for some time. There is no uniformity in the frequency of its occurrence or in the character of the vomit. As a rule, vomiting occurs at a longer interval after taking food than in the case of ulcer, and the ejection of food does not give as much relief to the patient. Vomiting may occur every day or several times a day in the early stages, but if *dilatation* of the stomach develops, as it usually does in cancer of the pylorus, vomiting may be deferred for several days, and then be correspondingly more copious in amount. Blood, frequently altered by gastric juice so as to resemble coffee-grounds, is a common constituent of the vomit. (See Under Inspection.)

Vomiting in Infections. Vomiting frequently marks the onset of acute diseases, especially *pneumonia* and the *eruptive fevers* and *yellow fever*. Excessive vomiting generally indicates that the case will be severe.

Reflex Vomiting. Nausea and vomiting are excited in some persons by the *sight* of blood, or by a horrible or loathsome spectacle; others are more susceptible to foul *odors* and disgusting *tastes*.

Vomiting is frequently reflex, that is to say, irritation at some point is transmitted by the proper afferent nerve to the vomiting-centre and then reflected to the stomach. Vomiting of this character occurs in *pregnancy*, diseases of the *appendix vermiformis, ovaries, uterus, bladder, prostate gland, lungs, nose, eyes, kidneys, intestine, peritoneum, liver, gall-bladder*, and *bile-ducts*.

Vomiting is found to be of reflex origin when there is no local affection of the stomach present and no general disease to account for it, and when a remote source of irritation can be discovered, the removal or mitigation of which checks this vomiting. The particular organ which is the source of the irritation must be determined by a careful physical examination guided by the indications furnished by the age, sex, time of occurrence, habits, and other symptoms which accompany the vomiting.

The nausea and vomiting from which many women suffer during the early months of *pregnancy* are most marked on rising in the morning; they are aggravated if the patient has been on her feet much or has been subjected to any exhausting or worrying influence; on the other hand, they are relieved by quiet and the recumbent posture. In diseases of the ovary, uterus, bladder, and prostate there are local pain, catarrhal symptoms, inflammation or noticeable enlargement.

The *lungs* are probably not often the cause of reflex vomiting. Rarely, however, *phthisis* is so masked by gastric symptoms and vomiting as to be overlooked. More frequently it is the act of coughing and the effort to expel the sputa from the throat that produce the

vomiting. Expectoration tickles the throat, and may have the same effect as the finger or feather in inducing vomiting. This seems to be the explanation of the vomiting which follows a hard spell of coughing in pertussis.

Peritonitis may be suspected to be the cause of vomiting if there has been injury to the peritoneum from a surgical operation, or if it has been exposed to infection through the uterus and tubes, or from disease of organs surrounded by it, as the vermiform appendix. Vomiting may be the only symptom present except collapse. The fluid is not only ejected, but regurgitated, and may appear to flow from the stomach. Large amounts of fluid are discharged, clear or of a green color.

In the vomiting due to the passage of a *renal* calculus or *gallstone* the colicky pains and their location definitely point to the source.

Vomiting in Toxæmias. Vomiting is also a marked symptom of toxæmias; they produce vomiting probably by direct irritation of the vomiting-centre. Among such diseases are the *specific fevers*, notably *scarlet fever* and *yellow fever; sewer-gas poisoning;* diseases of the liver and kidney, which produce *cholæmia* and *uræmia*, particularly cirrhosis of the liver and interstitial nephritis.

Cyclic Vomiting. This condition was described by Leyden in 1882 as periodic vomiting. Cases in children have been recorded by Snow and others. Clinically, the vomiting is sudden in onset, severe, and consists first of the contents of the stomach, and later of acid mucus. There is usually a febrile reaction at the onset, but this may be absent in adults. The abdomen is almost invariably retracted. There is usually a degree of prostration which is out of proportion to the local manifestations, and may be dangerous. There may be narcosis, delirium, or great restlessness. These gastric crises recur at intervals of six weeks to six months, and will recur periodically in spite of the utmost care as to diet. This disease is probably a gastric neurosis, and has analogies with migraine. There is no reason to believe that it is reflex in origin. It may be due to the accumulation of toxic substances.

The vomiting of *uræmia* usually occurs in the morning. It is accompanied by nausea and depression. Whenever morning nausea and vomiting occur in an adult without obvious local cause the urine should be examined. Other confirmatory signs are high-tension pulse, accentuation of the aortic second sound, and hypertrophy of the heart.

Cerebral Vomiting. Vomiting due to *cerebral disease* is well recognized. In early life it is a characteristic feature of meningitis and tumor of the brain. It is likewise of moment in later life. I am of the conviction, however, that it is not sufficiently recognized as one of the first symptoms of apoplexy. True, we find that apoplexy occurs after a full meal, when the attack is associated with indigestion, with efforts at vomiting; and I do not here refer to such cases, but to cases of painless, often watery vomiting, occurring without nausea and without retching. A sudden, violent expulsion of the stomach-contents, ceaseless, unrelieved by remedial measures, has been seen by the writer to precede other signs of apoplexy by from thirty minutes to twenty-four hours. In all cases of apoplectic character the pulse is slow and

full, while in nausea and vomiting from other causes, in the aged particularly, it is weak and feeble. Moreover, some alteration of breathing is noticed. It is either irregular, or slow, or unduly hurried. It proves the intimate relation of the vomiting and the respiratory centres. Further, strength is seen, not weakness; in the apoplectic the face is congested, not pallid as in simple sick stomach. The other peculiarities of cerebral vomiting have been indicated.

Crises. Sudden attacks of vomiting with hyperacidity, with or without pain, often occur in *locomotor ataxia.* Such attacks occur in other affections, as hysteria. They occur in movable kidney, and are known as Dietl's crises.

Diagnosis. Vomiting is readily recognized. It is often productive of serious symptoms. It may cause apoplexy or cerebral congestion; it may cause acute overdistention of a dilated heart, as in aortic regurgitation. If it continues for any length of time, and much fluid is ejected, it is attended by anuria, and rapidly followed by collapse. It also induces thirst.

Flatulency. Flatulency is an accumulation of gas in the stomach or intestines. It is a very common source of complaint on the part of patients. Gastric flatulency is marked by a distention of the stomach, with the discomfort which it occasions, and by the eructation of gas at variable intervals after the taking of food. When the gas is the result of the fermentation which accompanies the production of the fatty acids flatulency is frequently accompanied by pain, which is relieved by eructations. When the distention is great or long continued, disturbances in the action of the heart, particularly palpitation and intermittency, are likely to occur. Occasionally it interferes with the breathing, and, from the apprehension which this symptom and palpitation excite, faintness and inaptitude for mental and physical work may arise.

Flatulence may be due to *carbonic acid,* which is generated and retained on account of motor deficiency. It is seen in the middle-aged and in the old. *Air* swallowed with the food or the saliva is an occasional cause. Flatulence may also be due to the regurgitation of pancreatic juice, as in fixation of the stomach-wall and open pylorus. It comes on four or five hours after eating, and is caused by decomposition of the carbonates of the pancreatic juice setting free carbonic acid. Flatulence from bacterial fermentation is seen in dilatation of the stomach, and is usually continuous. It also occurs in chronic indigestion. Flatulence in rare instances is due to disturbance of the interchange of gas between the blood and the contents of the stomach. Normally it is known as *gastro-intestinal respiration.*

Excessive flatulency is a common manifestation of hysteria. Such patients may complain of something rising into the throat from the stomach and smothering them (*globus hystericus*). There may also be tympanites, and even phantom tumor. It may be necessary to anæsthetize the patient completely, to diagnosticate the latter from genuine tumor.

Vertigo. The stomach is but one of a number of sources of vertigo. Some patients find by experience that certain articles of food,

such as oysters or lobsters, have to be avoided because they produce vertigo, although digestion is good, and more indigestible articles can be taken without inducing any such result.

In other cases acute indigestion from overeating, particularly if it result in the development of an acid condition of the stomach, is apt to be accompanied by vertigo when the stomach symptoms are most severe. Usually the vertigo is associated with headache, more or less intense; it is relieved by lying down and closing the eyes, but does not wholly disappear until all the symptoms gradually subside after free vomiting. Some persons are subject to so-called "blind" headaches—headaches accompanied by dimness of vision, more or less mental confusion, and uncertainty of gait, possibly with staggering, and often with vertigo. Such headaches appear to be due to an acid condition of the stomach, and are relieved by alkalies or vomiting.

It is difficult to separate the vertigo of chronic gastric or gastro-intestinal dyspepsia from that of lithæmia or latent gout. Probably both are due, not to any local irritation transmitted to the brain, but to the circulation in the blood of toxic products of digestion which act upon the brain. The vertigo is not so severe as in acute indigestion or acute dyspepsia, but is constant. In some patients it is associated with an unconquerable aversion to walking alone upon the street.

Pain. *Cardialgia* is a form of discomfort in the epigastrium scarcely amounting to pain, but attended by heartburn or acidity. *Gastrodynia* is a violent pain spoken of as cramp or spasm of the stomach. The pain is transient. *Gastralgia* is a form of pain with features like that of neuralgia, occurring when the stomach is empty. (See Gastric Neuroses.)

Location. Pain in the Epigastrium. Pain referred to the stomach is situated in the upper zone of the abdomen, below the ensiform cartilage, between the ribs of the two sides, usually in the median line. It may be along and under the left ribs. Pain in this situation may be due to a number of causes: 1. To myalgia, neuritis, or neuralgia of the intercostal nerves, which terminate in this situation. (See Abdominal Pain.) 2. Localized peritonitis or perigastritis, which may be secondary to or caused by infection or injury of the peritoneum from disease of contiguous organs. 3. Affections of the pancreas may cause pain: *a*. Pancreatic colic, a rare condition associated with diarrhœa, intestinal dyspepsia, and salivation. The pain is paroxysmal, the attacks lasting two or three hours. *b*. Pain due to carcinoma of the pancreas, darting or lancinating in character, associated usually with tumor, jaundice, and emaciation. *c*. Pain due to pancreatic hemorrhage. It is sudden and extremely severe, attended by collapse. 4. Pain in this situation may be due to aneurism of the aorta or of the cœliac axis. It is constant, of a boring character, and may be associated with shooting pains along the course of the lumbar nerves. The physical signs of aneurism are present. 5. Pain in this region may be due to hepatic colic. 6. It may be due to disease of the vertebræ. We should look for the sixth or seventh dorsal vertebra to be affected, hence higher up posteriorly than the area affected in front would indicate. 7. Affections of the stomach. Of these we have: *a*. Gastralgia

in all its forms. (See Gastric Neuroses.) *b*. Acute and chronic gastritis. *c*. Gastric ulcer. *d*. Carcinoma of the stomach. To the first class belongs a peculiar pain which occurs in locomotor ataxia, and which, on account of its sudden onset, with alarming and frequently repeated vomiting, is known as a gastric crisis.

Pain in the Left Hypochondrium. It may be due to a dilated stomach or distended colon.

Pain of Gastric Origin. In diseases of the *stomach* pain is a very common symptom. It is of all degrees, from a mere sense of discomfort or uneasiness to agony. In *atonic dyspepsia* there may be no local gastric symptoms except a feeling of weight and fulness, while in *nervous dyspepsia* there is usually uneasiness or discomfort after eating. In *gastralgia* the pain is characteristic: it usually comes on while the stomach is empty, and frequently recurs daily at the same hour. At first the pain is slight and easily borne, but it gradually increases in severity. Each succeeding paroxysm is worse than the preceding one, until a climax of agony is reached. In character the pain is gnawing and cramp-like, doubling the patient up, and after subsiding leaving him moist with cold sweat and in partial collapse.

In catarrhal dyspepsia there are pain and uneasiness in the stomach after eating, with tenderness on pressure. If flatulence coexists, there will be temporary relief to the discomfort upon the eructation of gas.

In *ulcer* there is a more or less constant feeling of soreness in the epigastrium. After taking food the dull pain is aggravated and becomes sharply localized. Frequently there is pain in the back at the same point, a little to the left of the spine and between the midscapular region and the lumbar vertebræ. The pain usually occurs sooner after taking food than in the case of cancer, and is more frequently relieved by vomiting. Attacks of gastralgia are not rare, and the pain may shoot down the arm.

In gastric *cancer* pain may be wholly absent throughout the entire course of the disease; but, as a rule, pain is more continuous than in ulcer, less severe, not so sharply localized, does not come on so soon after taking food, and is not relieved to the same degree by vomiting. Paroxysms of gastralgia are not so common.

In *acute gastritis* the pain and its character vary with the intensity of the inflammation. If due to the irritation of some toxic agent which has been swallowed, the pain is severe and burning; if the result of imprudence in eating and drinking, the pain is of a dull, sickening character. In either case there is more or less tenderness on pressure. Sometimes, in mild cases of catarrhal gastritis, firm pressure from a broad surface affords at least temporary relief to the distress.

Time of Pain. The significance of pain depends on the time of its occurrence. Pain coming on before eating or when the stomach is empty is due to gastralgia. It is relieved by food. When it comes on after eating, it is usually due to organic disease of the stomach, as ulcer or carcinoma; but it may be due to neurasthenia. It must not be confounded with the pain that occurs from two to four hours after meals, caused by intestinal indigestion or some pancreatic affection.

When the pain is *diffused*, it is due to hyperacidity and bacterial fermentation, as in dilatation, catarrhal gastritis, and simple indigestion. When *localized*, it is due to ulcer or cancer, and is associated with tenderness. It may extend to the back.

Alterations of Appetite. Loss of appetite, or *anorexia*, may be due to a number of diseases. It is present in all forms of organic disease of the stomach except occasionally in ulcer. In the majority of cases of this affection it is present. It may or may not be present in gastric neuroses. Every one is familiar with the loss of appetite due to nervous impressions, as emotions, anxiety, or mental care. It is of frequent occurrence in disorders remote from the stomach, which modify the condition of the organ reflexly. In the section on Vomiting will be found statements showing the influence of central disease and disease of distant organs upon the stomach. Through the same channels and through the same mechanism, and hence by the same group of causes, loss of appetite may be produced. Loss of appetite is a constant accompaniment of the moderate gastritis which attends all fevers. Reference cannot well be made to all the conditions which induce this symptom. In all forms of anæmia, in all chronic wasting diseases, and in functional and organic disease of the nervous system the appetite is lost. The writer has been particularly impressed with the importance of determining the presence or absence of suppuration in some portion of the body, in all cases in which there is loss of appetite or disgust for food, the cause of which is not of gastric origin.

Boulimia, or excessive appetite, sometimes occurs. It is popularly thought to be due to worms in children. It is a common symptom in the earlier periods of diabetes, and is said to be present in disease of the mesenteric glands. It occurs also in gastric neuroses. Perversion of the appetite, in which all sorts of substances are greedily swallowed, occurs in hysteria, dementia, and pregnancy. It is known as *pica*.

Regurgitation of gases or food matter is a frequent symptom of gastric disorder. It is also known as belching or eructation. It may be limited to the discharge of gas, although sometimes imperfectly digested food also regurgitates. (See Rumination.)

Regurgitation of the gastric juice alone causes an unpleasant taste, and the fluid is hot and acrid. The juice is usually brought up in the belching of gas.

Pyrosis, or waterbrash, is a common symptom in some forms of dyspepsia. It may occur in the morning when the stomach is empty, at which time large amounts of fluid are ejected. The fluid is thin and watery, sometimes acid, sometimes tasteless. In other cases the fluid is slightly alkaline. The fluid is ejected without vomiting. Sometimes the discharge begins immediately after eating. The late Dr. Chambers thought that the fluid was saliva which was swallowed and retained in the lower part of the œsophagus by a spasm of the cardiac orifice, and when a sufficient amount was collected, gushed back into the mouth. Pavy and Handfield Jones believe that the fluid is secreted by the stomach, while, on the other hand, Roberts, who found the liquid to possess diastatic power, believes it to be due to saliva. *Acid eructations* from hyperacidity or fermentation occur one or two

hours after meals. They rarely occur in dilatation, but are common in overfeeding.

Palpitation. Increased action of the heart is a common symptom of indigestion due to flatulency or an overloaded stomach. It occurs in the middle period of life, in the anæmic and neurotic, in cardiac disease, and in those who use tea and tobacco to excess.

Cough. Cough is a frequent symptom of gastric disorder. It may be due to the pharyngitis, which has been set up by acid eructations; it may be mechanical, when a distended stomach presses upon the diaphragm, or it may be reflex. Cough after meals in patients with tuberculosis or other pulmonary affection is usually due to pressure upon the diaphragm.

Dyspnœa. This occurs in many cases of dyspepsia if the subject is the victim of asthma, is anæmic, or subject to cardiac disease. In asthma it is usually reflex; in anæmia it is due to atony of the stomach and gaseous accumulation; in cardiac disease it is mechanical from the pressure of a gaseous distended stomach.

Hiccough, or singultus, is a spasm of the diaphragm. The contractions take place at more or less regular intervals, attended by a peculiar clicking sound. This sound is due to the sudden passage of air through the glottis. Hiccough may be a serious symptom. It may last but a few minutes or continue for several days. In the latter case it causes extreme exhaustion. Its occurrence in chronic disease is of bad prognostic omen.

Drowsiness is frequently seen in dyspeptics after meals. Sleeplessness is of frequent occurrence. It may be due to the irritation of food remaining in the stomach over night or to the absorption of toxic products.

Constipation. This symptom will be discussed in the chapter on Intestinal Diseases. It is present with gastric dilatation. In pyloric stenosis it is always present.

Diarrhœa. The digestion is impaired and peristalsis is in excess. Lienteric diarrhœa is an accompaniment of a gastric motor neurosis, or it may be due to the absence of HCl. In gastrectasia the fermentative products set up gastro-intestinal catarrh, which induces diarrhœa.

The Data Obtained by Observation.

The Objective Symptoms. One of the objective expressions of the *morbid process* or of altered function is seen in changes in the character of the contents of the stomach. The contents are obtained for examination when discharged from the stomach (vomit) or when removed artificially (washings). Both fluids are studied by inspection, including microscopical examination and by chemical and bacteriological examination. The sense of smell enables one to differentiate many varieties of fluids. Alteration of function is also seen in alteration of digestion, and is estimated by chemical and physiological methods. The activity of the digestion must be determined by ascertaining the duration of digestion and its degree of completeness, which depend upon three factors: (1) The motor power; (2) the absorptive power; (3) the digestive power of the gastric secretions.

To secure objective data, therefore, the following are necessary :

I. Physical examination, to determine tenderness and the size, position, and movement (peristalsis) of the stomach.

II. Examination of the gastric contents.

III. Examination of the digestive power of the stomach.

IV. Examination of the motor power of the stomach.

V. Examination of the absorptive power of the stomach.

I. Physical Examination of the Stomach. Inspection. Direct *inspection* of the stomach region often affords much positive information. When there is much loss of abdominal fat and the stomach is well distended its outlines can sometimes be traced with the eye. The best position is behind and above the patient's head while he is lying down. If the lower curvature can be traced considerably below the navel, the stomach is almost certainly dilated, and if, at the same time, there is a prominent swelling in the pyloric region, accompanied by progressive loss of weight and cachexia, the dilatation is probably due to cancer of the pylorus. A marked groove extending from the umbilicus to the ribs, about or to the left of the nipple-line, is seen in cases of dilatation when the stomach has fallen. It is the position of the lesser curvature. The lower border is also marked by a groove extending in a curve from the pubis toward the first groove.

Peristaltic waves may be seen to move spontaneously, or after tapping the region or applying an ether spray or faradism. When the pylorus is obstructed anti-peristaltic waves may also be seen. The waves of the muscular contraction begin at the cardiac end or fundus, and extend to the pylorus; hence, they begin under the ribs of the left side and extend downward toward the right lower quadrant. They vary in extent with the amount of dilatation. (See page 729.)

Distention of the stomach with carbonic oxide (see Percussion), or, better, with air by means of a hand-bulb syringe, frequently brings the outlines of tumors of the pylorus plainly into view, while at the same time any tumor lying behind the stomach becomes less distinct, and false tumors due to spasm of the gastric muscular coat vanish. Distention also helps to map out the whole stomach and to separate it from surrounding viscera. It enables one to estimate the size and position of the stomach. Hence, by this means descent can be told from dilatation.

Gastrodiaphany or Transillumination of the Stomach. Einhorn has succeeded in transilluminating the stomach by an Edison lamp fastened to a soft-rubber tube. The wires to the battery are carried through the tube. After the stomach contents have been removed the patient is to take one or two glassfuls of water. The apparatus after lubrication is then inserted. The examination must be made in a dark room. By means of gastrodiaphany the position and size of the stomach are determined, to a certain extent, and the presence of tumors of the anterior wall of the stomach is recognized. The results are not strictly accurate, however, as transillumination of the intestines is brought about if they are empty. The form and size of the stomach are not so readily brought out as the topographic relation of tumors of the stomach and those in the vicinity of that organ. It is of service in some cases to distinguish dilatation from gastroptosis.

Röntgen Light. The outline of the stomach may be observed by the use of X-rays, provided the patient has been given 10 or 20 grains of subnitrate of bismuth.

PALPATION. Palpation of the stomach is closely associated with auscultation, inasmuch as the former also elicits sounds (succussion, gurgling) which are helpful in diagnosis. The hand must be placed flat upon the abdomen and pressure made by bending the ends of the phalanges. To make deep palpation, gradually increasing pressure with a rotary movement must be employed. It may be of advantage to palpate in the knee-elbow position, so that deeply seated tumors, if movable, may fall to the abdominal wall. (See Auscultation.)

But palpation elicits information independently of auscultation, chiefly in conditions of disease. *Epigastric pulsation* is common in anæmia; in nervous dyspepsia; in valvular disease of the heart, particularly tricuspid regurgitation, producing a liver-pulse; and in the rare cases of aneurism of the abdominal aorta.

Increased resistance may be due to the hypertrophy of the muscular coat which coexists with distention of the stomach. When the stomach is shrunken and the resistance increased, it may be due to a diffused carcinoma of the walls of the stomach; or, rarely, to the so-called "fibroid stomach," the atrophy and thickening of the walls being due to chronic gastritis.

Increased resistance limited to the pylorus is found in *carcinoma.* The same effect produced by a tense right rectus muscle must be excluded.

Position of Gastric Tumors. Cancers of the pylorus are situated usually between the xiphoid cartilage and the umbilicus, frequently a little to the right of the median line; but they may be found below the umbilicus, and, exceptionally, still lower down. Adhesions to neighboring organs commonly prevent the tumor from being moved. When it has formed adhesions to the liver or diaphragm it moves with respiration.

As a rule, tumors due to gastric cancer are small, hard, and irregular, and gradually increase in size.

Non-malignant tumors are occasionally found, and also tumors due to adhesions around old ulcers, and to puckered scars. The latter are distinguished from cancerous tumors, not by the physical examination, but by their duration and clinical history.

Another method of determining the position and size of the stomach is by *internal exploration* combined with external palpation. A bougie is introduced into the stomach and swept over its entire internal surface, the position of the bougie being followed from point to point by the palpating hand. This method is not advisable when it is possible to make a diagnosis without it.

Pain and Tenderness. Tenderness is elicited by palpation in gastritis, in dyspepsia, especially the catarrhal form, in ulcer, and in cancer. In gastritis and dyspepsia the tenderness is usually diffuse and is not constant; in cancer the tenderness is usually limited to the seat of the tumor, but is not so marked nor so sharply localized as in ulcer. In ulcer tenderness is rarely absent; even when there is no

pain, it is very decided, and is so localized, sometimes, that it can be covered with the tip of the finger. Pain in the stomach from ulcer is chronic, circumscribed, and variously described as burning and wound-like. It is aggravated by palpation, and by food or drink, especially hot stimulating drinks, and relieved by cold, soothing drinks. It is accompanied frequently by pain in the corresponding vertebræ.

Diffuse pain is met with in acute and chronic gastritis, and in cancer of the stomach-walls.

Percussion. *Position of the Stomach.* (Plate XXXVIII., Fig. 1.) The stomach does not occupy a fixed position, and is a distensible organ. It is depressed by downward pressure of the diaphragm in deep inspiration, by emphysema, left pleural effusions, enlargements of the liver and spleen, and tight lacing; it is raised by any causes which greatly distend the bowels or peritoneal cavity—tympanites, peritoneal effusions, tumors, etc. Moreover, after food is taken, the stomach is distended and its position changed, being rotated anteriorly from below, the greater curvature rising and looking more forward, while the anterior surface has a more upward presentation.

The cardiac orifice of the stomach is fixed by its passage through the diaphragm and by peritoneal attachments which it receives there. It is behind the sternal insertion of the left seventh rib. The pylorus, on the contrary, is freely movable when the stomach is empty; it is nearly in the median line, but when the stomach is full it is pushed several inches to the right; it lies between the right sternal and parasternal lines, on a level with the tip of the xiphoid cartilage.

Obrastzow (*Deut. Arch. für klin. Medicin*, Bd. xliii. 5, 417–456) divides the space between the navel and the xiphoid cartilage into three equal parts, and says that the *lower border* of the stomach, both in men and in women, is in the lower or supra-umbilical third.

In children under fifteen years the lower border rarely extends to the umbilical line; after fifty years, on the contrary, it often extends below the navel. In conditions of bad nutrition it falls nearly to the navel.

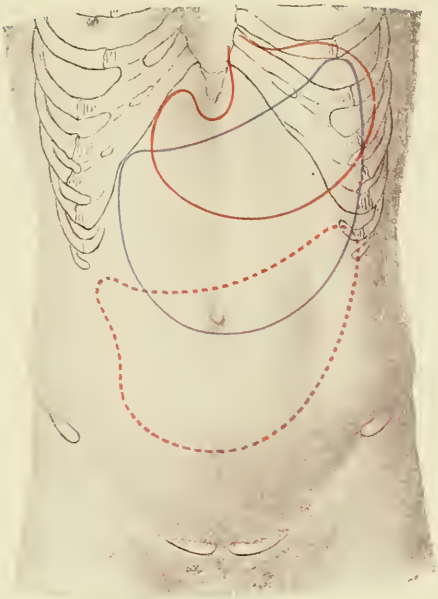
According to Pacanowski and Wagner, the *upper border* of the stomach, in the left parasternal line, lies at the lower border of the fifth rib or in the fifth intercostal space, rarely at the fourth rib or in the sixth intercostal space. In the left nipple-line it lies from the fifth interspace to the sixth rib, occasionally in the fourth interspace or at the seventh rib. In the anterior axillary line it lies at the lower border of the seventh or eighth rib, rarely above the sixth rib, never under the eighth rib.

Traube has called special attention to the left lower portion of the thorax which projects over the stomach, "the half-moon-shaped space." The upper limit is a crescentic line starting from the sternum in the sixth interspace and extending, in a curved line corresponding approximately to the curve of the rib, to the axillary line. It is known as "Traube's line." In health this space gives a tympanitic note, unless the stomach or transverse colon is full, or the omentum very fatty. In left pleural effusion it is dull. (See Diseases of Lungs.)

A part of the anterior portion of the stomach and its lower border

PLATE XXXVIII.

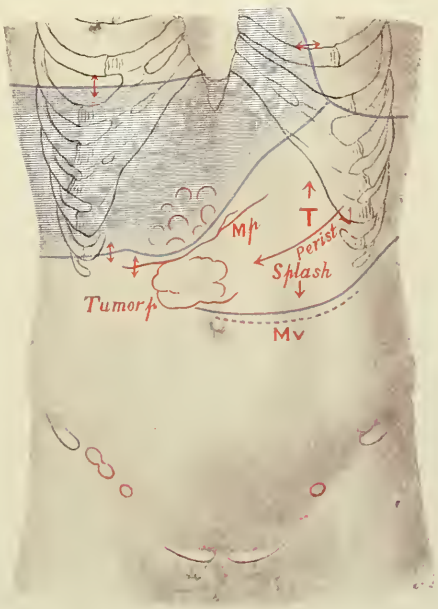
FIG. 1.



Normal Position and Displacements of the Stomach.

Solid red line—Normal position of distended stomach. Blue line—Atonic dililation. Dotted red line—Gastroptosis.

FIG 2.



Carcinoma of the Stomach with Pyloric Stenosis.
Metastases in the Liver.

can be determined by percussion. Ordinarily, the most suitable position for examining the stomach is the recumbent one, with the knees drawn up, so as to relax the abdominal muscles.

The stomach contains air at all times, but the amount varies greatly.

The percussion-note is tympanitic, high in pitch, frequently with a metallic ring; its quality is peculiar—"stomach tympany."

The percussion-area of the stomach is *increased* (1) by causes external to the stomach; contraction of the liver, old pleurisy with retraction of lung, emphysema, former pregnancies, bad nutrition, and tumors pulling down the stomach; (2) by intrinsic causes; distention of the stomach.

Conversely, the percussion-area is *diminished* by causes external to the stomach; enlargement of the liver and spleen, left-sided pleural effusion, pneumothorax, and hypertrophy of the heart.

Actual *diminution in size* of the stomach itself is difficult to demonstrate clinically with certainty. If upon inflation the great curvature remains at a higher level than 3 to 5 cm. above the umbilicus, diminution in size is highly probable. But even then the lower border may be prevented from descending by adhesions to surrounding viscera.

Enlargement of the stomach is generally due to *dilatation*, and is best marked clinically by a low position of the greater curvature. Dilatation of the stomach, according to Boas, can be separated from descent of the organ only when the greater curvature is more or less below the level of the navel, and when the greatest height of the stomach exceeds 10–14 cm. (4 to $5\frac{1}{2}$ inches). But descent and dilatation are frequently present together. (Plate XXXVIII., Fig. 1.) It must not be forgotten that when there is descent the normal tympany is lowered and the tympanitic area above the ribs is replaced by dulness.

Sometimes when the stomach is distended by air the right margin will be seen to extend far beyond the ordinary limits. Michaelis points out that this may be due to defective motor power, especially if the right margin is more than 9 cm. from the median line. The distention to the right is due to actual enlargement and not to dislocation. The author believes that dilatation of the antrum of the pylorus causes this enlargement. Enlargement of the stomach downward is usually associated with good motor power, whereas enlargement to the right is an indication of feeble motor power.

Auscultatory percussion is a most satisfactory method of determining the borders of the stomach and its size. Its area can readily be defined from that of the liver, spleen, and colon: First, with the stomach normal; second, inflated by gas; third, filled with fluid. It is well to determine the results in the recumbent posture, and then in the upright, so as to determine if the stomach falls from its normal position. Liquid may be injected through the stomach-tube, or the patient may drink successive portions, percussion being employed after each amount (eight ounces) taken. After the site of the dulness is fixed, have the patient lie down. The fluid falls backward and the air in the stomach comes anteriorly; the dull note is replaced by a tympanitic note. The change is a sign the fluid is in the stomach, and serves to distinguish stomach from colon tympany. The force required for

percussion should be very light; indeed, a fillip with the nail is sometimes sufficient. It may even be well to allow the blow to glance from the surface, as the perpendicular stroke brings out the general abdominal resonance. The use of coins is sometimes of advantage. In *dilatation* of the stomach the percussion-note sometimes varies in tone over the viscus from dull to tympanitic, or *vice versa*, because the organ contracts under the influence of the blows. Some have described a clinking percussion-sound, not unlike that of the "cracked pot," over the thorax.

Auscultatory friction is also employed in the same manner as auscultatory percussion, while rubbing the finger tips over the surface lightly. As long as the rubbing is made over the hollow organ over which the stethoscope is placed, and not moved more than two inches from it, the friction is heard distinctly.

In order to separate stomach tympany from that of the colon, which resembles it, the stomach may be distended with gas, while the colon contains solid or liquid matter; or, if the colon be filled with gas, the patient may be allowed to stand and drink a glass or two of water. In either case the contrast between a dull and a clear note marks the boundary between stomach and colon.

Ziemssen recommends carbonic acid (developed by mixing sodium bicarbonate and tartaric acid) to distend the stomach; the quantity employed for adult men is seven grammes of bicarbonate of soda and six grammes (one and one-half drachms) of tartaric acid. Adult women should receive one gramme less of each.

As carbonic acid sometimes causes an uncomfortable oppression, ordinary air is preferred by some. It can be forced in by a hand-bulb syringe attached to an ordinary stomach-tube. The percussion-note over tumors of the pylorus is imperfectly tympanitic. Welch describes it as "tympanitic dulness." Less frequently it is dull, and rarely it is flat.

Auscultation. Auscultation can determine whether or not there is obstruction at the cardiac orifice. On listening over the œsophagus with the stethoscope, when the patient is swallowing a liquid, a spurting sound is heard, followed in from five to ten or twelve seconds by a second sound, which marks the escape of the fluid from the cardiac orifice of the œsophagus into the stomach, so-called "deglutition-murmur." When there is obstruction of the cardiac orifice the second sound may be delayed as long as a minute.

When the stomach is partly filled with fluid a *succussion* or *splashing* sound can be produced by moving the patient quickly from side to side, or by quickly compressing the stomach and allowing it to rebound again immediately. Such compression may be made alternately, first in the neighborhood of the fundus of the stomach and then in the region of the umbilicus. Both hands should be employed. The splashing sounds are also developed by rapidly tapping, with the finger tips held perpendicularly, the region between the ribs and the transverse umbilical line on the left side. The ear need not be applied to the body, but kept near by while the movements are made. Such sounds are abnormal if they are heard long after digestion should be

completed and the stomach empty. If they are heard more than three hours after a light, or six hours after a full meal, it indicates slow digestion or deficient motility, and gives the approximate position of the lower boundary of the stomach.

Normally, after drinking fluids, a splashing sound is not developed lower than the umbilical line. If it is heard below this line, it is an indication of dilatation or of deep position of the whole stomach. Dilatation is very probable if the splashing sound is heard below the navel in a fasting stomach. A good idea of the extent and location of the splashing, and hence of the lower boundary, can be secured, if auscultation is conducted when inflation is practised with air.

Furthermore, this sound is a sign of atony. If 50 to 100 grammes of water be swallowed, no splashing sound is heard unless there is atony of the stomach-walls; but, if the atony is pronounced, a smaller quantity will be sufficient to develop the sound. It is to be remembered that the splashing sound of itself does not indicate disease. It is significant only when taken with other signs, and also when it is found after more than one examination.

II. Examination of the Gastric Contents. Either the contents are secured with a stomach-tube or the vomitus is examined.

Mode of Procedure. 1. A test-breakfast (Ewald), or a test-dinner (Leube), is administered, or the *fasting* stomach contents removed. *Ewald's test-breakfast:* It consists of one or two ounces (35 grammes) of bread and a cup of tea ($\frac{1}{3}$ litre), or the same amount of water. *Leube-Riegel test-dinner:* A large plate of soup (400 c.c.), a large portion of beefsteak or other meat, some potatoes, and a roll are taken, and examination is made three or four hours after the meal. (See Boas' Meal. Lactic Acid.) 2. Remove the contents of the stomach one hour after breakfast is taken, by aspiration or by expression. *Aspiration* consists in the withdrawal of the stomach-contents by suction; either with the ordinary stomach-pump, by means of a bottle exhausted of air, as employed for paracentesis, and connected with the stomach-sound, or by connecting the sound with a hand-ball aspirator or Politzer bulb.

Expression consists in compression by the abdominal muscles, as if straining in defecation. The patient takes a deep inspiration and then contracts the muscles as above. If the tube is long enough it can be bent, so as to assist expression with siphonage.

Aspiration is less disagreeable to the patient, and is necessary when the stomach-contents are not fluid enough to flow easily.

Expression is not to be employed when there are old ulcers, ulcerating carcinoma, phthisis with antecedent hæmoptysis, or a disposition to menorrhagia.

These methods supply the most reliable information of the condition of the stomach and its secretions; because, when once withdrawn, the character of the secretions can be ascertained accurately and the quantity measured; moreover, being able to choose the time of examination, we can decide whether or not what is found corresponds with health, and if not, in what particular it indicates disease. These methods permit a diagnosis to be made before other methods supply sufficient data.

A soft-rubber tube, with two good-sized openings near its distal extremity, should be selected. Stockton suggests a tracing of rings around the tube one inch apart, beginning twenty inches from and ending thirty inches from the lower extremity, for the purpose of measuring the length of the tube inserted. In healthy adults the distance from the incisor teeth to the lower border of the stomach is about twenty-two inches. In dilatation it may be from twenty-four to thirty. The distance is partly determined by success in the siphonage. If the return flow of fluid does not take place, it is well either to withdraw the tube or push it further on; for, if too long, it may curve above the level of the fluid, or, if too short, it may not reach the fluid.

After the tube is moistened, oiled, or coated with the white of an egg, the patient should be seated, and the tube at once passed to the back of the pharynx, and, with or without guiding by the finger, pushed toward the œsophagus. It is at once grasped by the œsophagus or lower pharynx, and, if the patient is instructed to swallow and to breathe slowly, it is rapidly carried downward by deglutition. Mucus that accumulates in the mouth after the tube is passed should be allowed to dribble outward and not be swallowed. It is often of advantage to reassure the patient by having him pronounce the letter "a" or some small syllable. It is not necessary to extend the head backward. The tube is then attached to the apparatus used for paracentesis, or to a tube entering a bottle in which a vacuum is created by an ordinary rubber bulb apparatus; or to the aspirator of Boas, which is a modification of the ball-syringe. A valve is placed between the stomach-sound and the syringe.

If a hard tube is used, it must be guided by the operator, who should stand back of the patient, supporting the head, which should not be thrown too far backward. The tube can be passed by the operator seated in front of the patient. This kind of tube is used with the stomach-pump.

Normal Gastric Contents. The amount of fluid, after digestion of a test-breakfast has continued for one hour, is from 30 to 40 c.c. After filtering the filtrate is clear, yellow, or yellowish-brown in color. If the digestion is normal, the fluid should contain free hydrochloric acid and no lactic acid. It should also contain pepsin, rennin (the milk-curdling ferment), and organic acids. Albuminoids should be converted into proteoses and peptone, and starches into achroödextrine, dextrose, or maltose.

Physical and Chemical Examination. The steps taken are as follows:

A. Physical examination:

1. The reaction.
2. The odor.
3. The character and quantity. Inspection.

B. Chemical examination.

It is to be observed that perfect familiarity with the products of and the length of time required by normal digestion is very essential.

1. Reaction. The normal reaction of the contents of the stomach

is usually acid, from the hydrochloric acid of the gastric juice. It may be alkaline in cases of hemorrhage, or in the vomiting known as waterbrash.

2. Odor. The odor is sour normally, but it may be aromatic from the presence of the fatty acids, fecal in obstruction of the bowels with fecal vomiting, and, finally, may indicate the nature of poisonous ingesta—ammonia, phosphorus, carbolic acid. The dark, frothy material from a dilated stomach is of a foul, yeasty odor.

3. Inspection of the Stomach-contents. By ordinary inspection the *quantity* and the *character* of the vomitus or stomach-contents are noted. With the aid of the microscope the various micro-organisms are sought for. In this manner most valuable information as to the digestive, motor, and absorptive power is ascertained. Not only do we learn whether digestion has taken place or not, but also the variety of food that is undigested—albuminoids or hydrocarbons.

The Quantity. Fasting Stomach. If a person has taken no food or drink between the evening meal and the following morning, the stomach should not contain more than three and one-half fluidounces; more than this is abnormal.

The Character. By it we learn the *digestive power.* If undigested food is found after digestion should be normally completed, there is deficient digestive energy. No undigested food should be found longer than six or seven hours after an ordinary meal of mixed foods.

By inspection of the gastric contents we learn much regarding the *motor power.* Boas states that an abnormally great quantity of solid matter and a small amount of chyme indicate an abnormal retention of the latter, which is usually brought about by motor weakness (atony, dilatation of the stomach), or dilatation in conjunction with insufficient absorptive power. Sometimes, when there is a large residue in the stomach, the contents separate into three layers. The uppermost is mucus or undigested food; the second, generally the thickest layer, consists of fluid; and the lowest layer is chyme. Such a formation, he says, points to abnormally long retention as the result of stenosis and consecutive dilatation, or to motor weakness.

The stomach should be empty much sooner if only starches are taken, as in Ewald's test-breakfast. One hour after the administration of a test-breakfast of 35 grammes of white bread and 300 grammes of water there should remain 40 c.c. Hence, if after such a breakfast there is found a much greater quantity, then motor or absorptive insufficiency may be considered to exist. A filtrate of 100 to 300 c.c. is very probably due to organic obstruction to the outflow, stenosis of the pylorus, adhesions, or dislocation of the pylorus. Of course, to make sure that the stomach contains nothing at the time of giving the breakfast, it must first be emptied. The character of the food taken is observed, as undigested particles may be seen in the contents.

We can discover by inspection if food is brought up by *vomiting or regurgitation.* Regurgitation of food from the œsophagus can be told from vomiting by the appearance of muscle-fibres, if meat has been taken. If it is vomited, the fibre is in a state of disintegration; if not, it is whole.

Mucus is found in small quantity normally, but is increased in catarrhal affections of the mouth, throat, or stomach. When its source is the mouth, *saliva* also is generally present. Mucus is recognized by its stringy, tenacious character. *Chemical diagnosis.* Add the mucus, gently shaking, to cold water; pour off the supernatant water; add a little liquor potassæ. The mucus is dissolved by the alkali. To the solution add acetic acid; a precipitate is formed which is insoluble in an excess of acetic acid. In this manner mucus is distinguished from the precipitate of syntonin, as the latter is soluble in an excess. *Pigmented mucus* in vomitus is usually from the bronchial tubes.

Bile and intestinal juice may be regurgitated into the stomach as the result of violent emesis, or when the pylorus is much relaxed, or in stenosis of the duodenum below the common duct; bile is then present in large quantity if the stomach is dilated.[1] Bile is recognized by the usual tests (see under Examination of Urine), and intestinal juice by its peculiar properties and the presence of leucin and tyrosin. Absence of bile in the vomitus is an indication of pyloric stenosis.

Blood is found in ulcer; cancer; acute, especially toxic, gastritis; injuries to the mucous membrane from the use of the sound for expression, and violent retching. It is also common in cirrhosis of the liver, and may occur in purpura, peliosis rheumatica, the hemorrhagic diathesis, and in yellow fever. Blood mixed with gastric mucus may come from the lung, the act of coughing having excited vomiting.

If the blood is unaltered, it can be distinguished from all other substances by microscopic examination. Occasionally the blood has the appearance of coffee-grounds. The hemorrhage has taken place slowly under these circumstances. In fact, the more rapid the bleeding the brighter the red color of the blood. The *hæmin test* serves to distinguish it. The suspected material is filtered and a little of the filtrate evaporated in a watch-glass; when dry a small portion is mixed with finely pulverized salt upon a glass slide; it is then covered with a cover-glass and one or two drops of glacial acetic acid allowed to flow under the cover-glass. The acetic acid is evaporated by slowly heating the slip over a small flame, and when dry a few drops of water are allowed to flow under the cover-glass, to dissolve the salt. If the vomit contained blood, brown rhombic crystals of hæmin (hydrochlorate of hæmin) will appear under the microscope. As they are very small, a magnification of about 300 diameters will be necessary to bring them readily into view. The *guaiacum* test may be fallacious, as the same color-reaction takes place when bile or saliva or a starch, like potato, is in the test-liquid. It is performed as follows: Add two or three drops of the tincture of guaiacum to a small portion of the gastric contents in a test-tube and pour ozonic ether on the surface. When the liquids meet a blue color develops. Bile may be distinguished from blood by Gmelin's test for the former—color-reaction with nitric acid. If blood is present in the stomach-contents, it may be detected by the test for iron. To the gastric contents, "coffee-

[1] Hochhaus. Berlin. klin. Woch., 1891, No. 17.

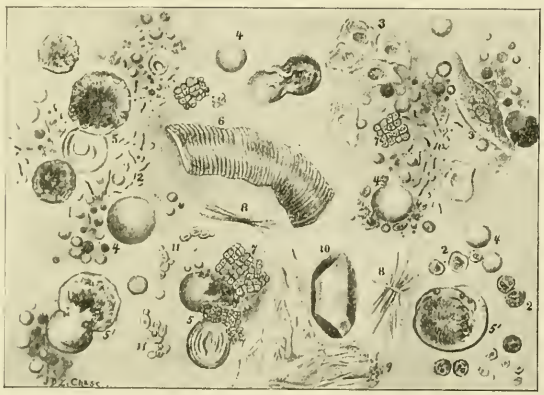
grounds," in a porcelain capsule, add a small quantity of potassium chlorate and a few drops of a strong acid, HCl. Heat over a flame and add a few drops of a 5 per cent. solution of potassium ferrocyanide. If iron is present, Prussian blue is formed.

Pus is rarely present in sufficient quantity to be detected by the naked eye, but it sometimes occurs in phlegmonous gastritis and when an abscess has ruptured into the stomach. In microscopic amounts it may be found in severe catarrhal affections. Pus may be in the vomitus and yet come from the lungs. It is usually a mucopus, and is told by the pigmented pellets or strings of mucopurulent material.

Fecal matter is vomited in complete obstruction of the bowels, and, according to Vierordt, in severe diffuse peritonitis. It is recognized partly by its appearance and partly by its odor.

Worms are sometimes vomited; the round worms not so very infrequently; oxyurides and ankylostomata rarely.

Fig. 196.



Microscopical appearance of stomach-contents.

1, red blood-corpuscles; 2, leucocytes; 3, squamous epithelium; 4, fat-globules; 5, starch granules; 5′, starch changed by action of the gastric juice; 6, muscular fibre; 7, sarcinæ ventriculi; 8, fat-crystals; 9, piece of orange; 10, phosphatic crystal; 11, yeast fungi; 12, bacilli and micrococci.

Microscopical Examination. The illustration (Fig. 196) shows the various matters which may be found in vomited matter. Briefly, they are columnar and squamous epithelium; white blood-corpuscles acted on by gastric juice; red blood-corpuscles. The corpuscles are usually isolated. The red are rarely perfect, and in the white little more than the nucleus remains. From the food we may also find muscle-fibres, fatty globules, and fat-needles, elastic fibres and connective tissue, starch-granules, and vegetable cells. Muscle-fibres are recognized by their transverse striation. Fat-globules are soluble in ether, and are recognized by their refracting powers. Starch-granules stain blue with iodo-potassic-iodide solution.

In addition, *fungi* of many forms are found, as the mould-fungi;

the yeasts (torulæ), and fission-fungi. The latter are recognized after staining by the iodo-potassic-iodide solution, which colors them blue. The most important fission-fungi are the sarcinæ ventriculi. They are of a dark gray tint, stain mahogany-brown to reddish-brown with the above-mentioned solution, and resemble in shape corded bales of goods. (See Bacteriological Diagnosis.) The *torulæ* and *sarcinæ* are present when fermentation is in progress, and hence indicate delayed digestion from motor insufficiency or deficient digestive energy.

B. Chemical Examination. A chemical examination is made to determine (1) the presence of free acids; (2) the degree of total acidity of the stomach-contents; (3) the presence of free HCl; (4) the presence of lactic acid; (5) the presence of volatile acids; (6) the presence of products of digestion and the digestive power; (7) the presence of pepsin; (8) the presence of rennin; (9) the carbo-hydrates. Hydrochloric acid is the normal acid of the gastric juice. Normally lactic acid is found during the first half-hour of digestion, when starches have been taken. When only meats have been taken lactic acid is not found early in digestion. The secretion of hydrochloric acid is not delayed until then, but is at first combined, and cannot be detected as free acid until half or three-quarters of an hour afterward.

1. Free Acids. The most sensitive test for free acids is *Congo red*. Filter-paper soaked in a saturated solution of the dye and allowed to dry is turned a deep blue if free acid is present. Prepared with a weak solution, the filter-paper is turned to a light blue by HCl and violet by organic acids. Wolff [1] was able to detect one part of HCl in 20,000 parts of water. When no reaction is obtained, therefore, entire absence of acidity may be assumed.

Benzo-purpurin test-papers are made as follows: Soak strips of filter-paper in a saturated solution of benzo-purpurin and dry. They are purple. If hydrochloric acid is present they are turned dark blue. The color is not removed by shaking with ether. If organic acids (butyric or lactic) are present, it is turned brownish-black, but the color is removed by shaking with ether. Von Jaksch states that if hydrochloric acid and the organic acids are present a brownish-black color is also produced, hence the dark blue and the volatile brownish-black reactions only are important.

The presence of free acids, as indicated by the Congo red or benzo-purpurin tests, shows that:

a. HCl—inorganic acid—may be present alone.

b. Lactic, butyric, or acetic acid—organic acids—one or all, may be present without HCl.

c. HCl and one or more of the organic acids may be present together.

Free acidity may be due (1) to fixed acids—hydrochloric or lactic acid, *fixed acidity;* (2) to volatile acids—butyric or acetic acid, *volatile acidity*.

2. The Total Acidity. This is determined by titration. The stomach-contents must be well shaken; if there is mucus in excess, it must be strained off through coarse muslin. Fill a Mohr's burette

[1] Trans. Philadelphia County Medical Society, 1889, vol. x. p. 305.

with a decinormal solution of caustic soda.[1] To 10 c.c. of the filtered gastric fluid add two drops of a saturated alcoholic solution of *phenolphthalein.* Allow the caustic-soda solution to drop slowly from the burette into the fluid, until a faint rose-red color is produced which does not disappear on shaking. The color is produced by the action of the alkali on the phenolphthalein. Four to 6 c.c. of the caustic soda solution are required to neutralize the acid in *normal digestion.* The degree of acidity is expressed in percentage. Thus if 4 c.c. neutralize 10 c.c., the total acidity will amount to 40 per cent., or if 6 c.c. are required, to 60 per cent.

If more or less than the amount just indicated of the alkaline solution is required to neutralize the acid, the total acidity is increased or diminished, and hence is abnormal.

Töpfer's method: To 10 c.c. of stomach-contents in a beaker, add 3 to 4 drops of a 1 per cent. solution of sodium alizarin sulphonate, then add a decinormal solution of sodium hydrate until a violet tint appears corresponding to the hue produced by adding 4 drops of alizarin solution to 5 c.c. of a 1 per cent. solution of sodium carbonate. The solution reacts to all factors producing gastric acidity except combined HCl.

Martin recommends the following modification of the above: "To 20 c.c. of the stomach-contents add three or four drops of a saturated alcoholic solution of phenolphthalein, and dilute with water to 300 c.c. Place 150 c.c. of this mixture in each of two flasks, and place them side by side on a sheet of white paper. To one of the flasks add decinormal solution of sodium hydrate until a red color appears; the exact time of appearance can be determined by comparison with the liquid in the other flask. When a pinkish tinge appears the acid liquid is neutralized. A control estimation may be made with the second flask."

Ewald's method of expressing the total acidity is by a number. The number is the same as the quantity of decinormal sodium hydrate solution requisite to neutralize 100 c.c. of the gastric contents. Thus if 50 c.c. of the soda solution neutralized 100 c.c. of the stomach-contents, the acidity of the latter would be expressed by the figure 50. The figures can be converted into terms of hydrochloric acid, as a decinormal solution of sodium hydrate is a liquid of a constant strength, 100 c.c. of which exactly neutralize 0.365 gramme of hydrochloric acid. It may be expressed in terms of hydrochloric acid. If 50 c.c. of decinormal sodium hydrate are required to neutralize 100 c.c. of the stomach-contents, this would be equal to 0.18 gramme per cent. hydrochloric acid, as 3.65 grammes hydrochloric acid are neutralized by the 4 grammes of soda in a litre (1000 c.c.) of the decinormal solution.

3. Free Hydrochloric Acid. The gastric contents are now filtered. *Tropæolin* 00 is declared by Boas to be an absolutely certain test for HCl. A saturated alcoholic solution is of an orange-yellow

[1] Decinormal solution of sodium hydrate is of the strength of 4 grammes of pure sodium hydrate to the litre of distilled water. The sodium hydrate must be pure and made from sodium. This weight of sodium hydrate (4 grammes) will exactly neutralize 3.65 grammes of hydrochloric acid.

color. Three or four drops of it are placed in a white porcelain dish and spread upon the sides by rotating it. The same amount of the fluid to be tested is then allowed to trickle down the sides of the dish and intimately mixed with the tropæolin. (Or evaporate the dye to dryness and then add the suspected liquid.) Upon heating the dish over a small flame splendid lilac-blue to blue streaks, characteristic of HCl, will appear if that acid is present. No organic acid gives the same color.

Tropæolin paper is turned brown by gastric juice containing HCl, the brown changing to blue upon the paper being heated. Organic acids give a brown color also, but it disappears upon heating.

Töpfer's test for the detection of free HCl is as follows: Dimethylamidoazobenzol is employed in a 0.5 per cent. solution of alcohol. To a few cubic centimetres of filtered stomach-contents one to four drops of the reagent are added in a test-tube or beaker. If hydrochloric acid is free a rose-red color is produced when the filtrate is added to the reagent. The drug reacts to HCl only when the latter is in a free state. Its reaction is not interfered with by salts, peptone, glucose, chloride of sodium, or starch. If organic acids are present in a concentration of from 0.5 to 0.8 per cent. a reaction may be brought about, providing albumin or peptone is present.

Phloroglucin vanillin, introduced by Günzburg, is also a very sensitive test for HCl. The following combination is said by Boas to be more sensitive than the ordinary one, which contains only 30 grammes of absolute alcohol:

Phloroglucin	2.0 (gr. xxx).
Vanillin	1.0 (gr. xv).
Alcohol (80 per cent.)	100.0 (f ℥iij).

Three drops are put into a porcelain dish and an equal quantity of the stomach filtrate. Upon *cautious* heating over a *small* flame a beautiful carmine surface is formed, especially at the edges. The same color is not produced by inorganic acids. Filter-paper soaked in it and moistened with a few drops of stomach-filtrate, containing HCl, changes on heating to a beautiful carmine, which is unaltered upon the addition of ether. *Günzburg's* original test is employed with the same solution, except that 30 parts of alcohol are used. One drop of the solution and one drop of the fluid to be examined are evaporated to dryness on a water-bath. The appearance of a rose-red color indicates the presence of hydrochloric acid.

Congo-red Test. Boas' method is a modification of that of Mintz. Ten c.c. of the gastric fluid are shaken with 100 c.c. of ether until organic acids are removed. The Congo-red test is then employed until the grayish-blue discoloration cannot be secured.

Boas' Resorcin Test. Dissolve 5 grammes (gr. lxxv) of resorcin and 3 grammes (gr. xlv) of cane-sugar in 100 c.c. (f ℥iijss) of weak spirit. Apply the test in exactly the same way as Günzburg's. A similar rose-red coloration, if free hydrochloric acid be present, is produced. It is the cheapest solution that can be employed.

Caution. In testing for the presence of HCl it is better to give the patient a meal which is known to be digestible within a certain time

by stomachs in a normal state, otherwise HCl may appear to be absent, because it is still combined with albuminoids. Ewald's test-breakfast is the simplest. In one hour the contents of the stomach may be aspirated and tested for HCl.

Amount of Free HCl. If by previous tests HCl is found alone, its percentage is easily calculated. To a measured quantity of the gastric fluid add, drop by drop, from a burette a decinormal alkaline solution until the acid is neutralized. This can be determined by checking the titration from time to time, and examining with Günzburg's reagent. One c.c. of the alkaline solution is equivalent to 0.003646 HCl, the limit of Günzburg's reaction. Multiply the number of c.c. required to neutralize 10 c.c. of the gastric solution by 0.003646, and again by 10, the result will be the percentage of acidity. If 6 c.c. are used, the percentage will be $6 \times 0.003646 \times 10 = 0.218$, within the *normal range*, which is from 0.14 to 0.24 per cent. Günzburg's test can be used to estimate the quantity of HCl. This is applied by diluting the stomach-contents until the test is not responded to. In health the limit of response is found when one part of HCl is found in 20,000 parts of the fluid. In abnormal conditions, when the gastric fluid is diluted one-half, the proportion is 2 to 20,000, or 1 to 10,000. If the fluid is diluted to ten times its original strength, it is 10 to 20,000, or 1 to 2000.

The following method is reliable and easy of employment. To two or three drops of Töpfer's solution of dimethylamidoazobenzol are added 10 c.c. of gastric contents, and a decinormal soda solution allowed to flow in, drop by drop, until a yellow color takes the place of the red. The number of c.c of solution of soda which will neutralize the free HCl in 100 c.c. of stomach-contents is multiplied by 0.00365. The result is the percentage of HCl. If 4 c.c. of soda solution is required to remove the red color, multiply 0.00365 by 40, the number equals 0.14 per cent. free hydrochloric acid.

4. Lactic Acid. If the stomach-contents are colorless, apply the following tests; if they are yellowish, make an ethereal extract, as described below, and then use the tests. Its presence may be determined by Uffelmann's reagent: Mix one drop of pure carbolic acid with five drops of a dilute solution of neutral ferric chloride. Add sufficient water to render the whole of an amethyst-blue color. To this add a few drops of the gastric fluid. A mere trace of lactic acid will change the blue to a light yellow or greenish yellow. The test for lactic acid is simulated when phosphates, glucose, or alcohol are present in the gastric juice. The lactic acid should be removed by extracting with ether, as follows: 50 c.c. of gastric contents are reduced to 10 c.c. by heat in an evaporating-dish over a water-bath. After the concentrated solution cools add 50 c.c. of ether. The volatile acids are driven off by heat, the lactic acid is dissolved by ether, and hydrochloric acid remains in the residue. Apply the test for lactic acid to the ethereal extract if it is acid. The following is more delicate: Add one drop of liq. ferri perchloridi to 50 c.c. of water; add suspected solution; the presence of lactic acid causes a yellow coloration.

Boas uses the following: When a substance containing lactic acid

is heated with oxidizers, such as manganese dioxide and sulphuric acid, the lactic acid is decomposed into formic acid and acetic aldehyde; the latter is detected by the formation of iodoform with an alkaline solution of iodine; peptone and alcohol, which react similarly, are eliminated by concentrating the filtrate to a syrup. As carbohydrates also yield aldehyde when treated with oxidizers, a watery solution of an ethereal extract of the condensed gastric filtrate of a trial-meal free from lactic acid must be used.

Arnold (*Journ. Am. Med. Assoc.*, Chicago, 1898, vol. viii. p. 21) gives a new test for the detection of lactic acid in the stomach-contents.

a. 0.2 c.c. saturated alcoholic solution of gentian-violet in 500 c.c. of distilled water.

b. Tinctura ferri perchloridi (*U. S. Pharm.*, 1890), 5 c.c.; distilled water, 20 c.c.

A drop of solution *b*, added to 1 c.c. of solution *a* in a porcelain basin, gives a blue color, which changes to a green or yellow-green on the addition of a few drops of filtered stomach-contents should lactic acid be present.

5. The Volatile Acids. These acids are best detected by their smell, their volatility, and their reaction.

Butyric acid is recognized by the pungent odor of rancid butter given off when the stomach-contents are evaporated. It is recognized by the following reaction: To a small quantity of the liquid add a small quantity of alcohol and two drops of strong sulphuric acid; heat for a short time; a characteristic smell of butyric ether, like that of "pineapple rum," is given off.

Butyric acid is also detected by Uffelmann's reagent. A few c.c. of the filtered gastric fluid are shaken with three or four times the amount of ether. The ether is poured off when it rises on the top, and fresh ether added and the washing repeated. After the third washing the ether that cannot be poured off is evaporated by means of a water-bath. Add a few drops of water to the residue and then an equal amount of the reagent. The characteristic color is produced. It strikes a tawny yellow color with a reddish tinge. As much as one part of the reagent in 2000 is required.

In addition to Uffelmann's test the volatile acids may be detected by boiling a few c.c. in a test-tube, over the mouth of which blue litmus-paper is attached. If acid is present, its vapor will change the blue to red. *Acetic acid* is recognized by its odor, particularly after heating the solution. It may be detected as follows: Secure an ethereal extract of the gastric contents (as above), evaporate in a water-bath, and dissolve the residue in water. Neutralize the watery solution with sodium carbonate, and then add neutral ferric chloride solution. A blood-red color results if acetic acid is present.

Alcohol is detected by its odor and by Lieben's iodoform-test. Distill the stomach-contents, add to a portion a small quantity of liquor potassæ, and then a few drops of iodine-iodide of potassium solution. A precipitate of iodoform takes place slowly if alcohol is present. If acetone is present, it forms rapidly.

6. The Products of Digestion. The ultimate products of digestion are the proteoses and peptones. If they are present in the stomach-contents, it shows that hydrochloric acid and pepsin must have been secreted in the stomach. If vomiting occurs soon after food is taken, or if there is obstruction at the lower end of the œsophagus, these products are not present. Syntonin is a product of digestion which precedes the two above given. To ascertain if digestion has taken place, it is necessary only to test for syntonin and then employ the biuret test. *Syntonin* is detected by neutralizing the gastric contents with a solution of sodium hydrate. The precipitate is *syntonin*, which is soluble in an excess of alkali, and may be again precipitated by an alkali. After filtration and removal of the syntonin, proteoses and peptone are detected by the biuret test.

7. Pepsin. If HCl is present, add 5 c.c. of a gastric filtrate to a small piece of egg-albumin. Allow digestion to take place for several hours at 37° to 40° C. Non-digestion indicates absence of pepsin.

If HCl is absent, pepsinogen is found alone. Add two drops of a 25 per cent. HCl solution to 10 c.c. of the gastric contents. Add to this solution a small portion of egg-albumin. If it is dissolved, pepsinogen was converted into pepsin by HCl.

8. Rennin (the milk-curdling ferment). This may be detected as follows: From 5 to 10 c.c. of cow's milk of neutral reaction is boiled and added to neutralized and filtered gastric juice. Place the mixture on a warm bath heated to 30° or 40° C. The casein of the milk is precipitated in flakes in from twenty to thirty minutes if the ferment is present.

9. The Carbohydrates. Add a few drops of Lugol's solution to the gastric contents. If starch is present, it turns blue. If erythrodextrin, it becomes purple. If the digestion has proceeded so far as to change starch into dextrose, the iodine hue remains unchanged. The starches should be completely digested an hour after they are taken into the stomach, hence in health the iodine hue should not change after this time.

III. The Digestive Power. Günzburg has introduced the use of iodide of potassium in the following way: From three to five grains are placed in a rubber tube with extremely thin walls; the ends of the tube are then bent and brought into apposition with each other and fastened in that position with three fibrin threads made firm by preservation in alcohol. The whole packet is then pressed into an empty gelatin capsule and given to a patient to swallow one-half hour after a test-breakfast. The saliva is tested for iodine every fifteen minutes. The more rapid the solution of the capsule and fibrin threads the sooner the iodine can be absorbed and appear in the saliva, and hence this rapidity is an index of the digestive energy.

The method is liable to fallacies. Solution of the fibrin may take place in the intestine instead of the bowel, and the threads may be loosened by the acids of fermentation instead of by digestion. Nevertheless, the test is a valuable one, especially when aspiration is inadmissible.

The digestive power can be estimated by ascertaining (1) the presence of gastric juice and (2) its activity.

1. *The Gastric Juice.* Wash out the fasting stomach with 400 c.c. of lukewarm water; test by litmus-paper for neutrality, then inject 50 c.c. of a 3 per cent. solution of soda. Allow the solution to remain twelve minutes and then remove by washing out the stomach with 400 c.c. of water. If the HCl secretion is normal, the soda solution is neutralized. If it is deficient, the solution remains alkaline. The presence of pepsin is then to be determined.

2. *The Activity of the Gastric Juice.* The white of one or two eggs should be boiled in four ounces of water and then administered. Remove the stomach-contents one-half hour later. The stomach should be emptied by lavage beforehand. The residue removed will show if digestion is complete, and proteoses and peptones may be tested for by the biuret reaction.

Test for the Activity of the Gastric Juice and of the Movements by a Test-meal. Ewald's test-breakfast must be employed if the patient cannot bear more solid food, otherwise Leube's test-meal should be used. If digestion is normal, the stomach-contents removed from five to seven hours after a test-meal are neutral and contain a few flakes of mucus. At the end of five hours the stomach-contents are acid and contain peptone, some undigested muscle-fibres, and starch-grains. If the stomach contains undigested food at the end of seven hours, the contents are acid and contain peptones, indicating delay in digestion.

IV. The Motor Power. Ewald and Sievers have suggested the use of salol; fifteen grains are given, and normally salicylic acid should be detected in the urine in from forty to sixty minutes, or in seventy-five minutes at the latest. If it is deferred still longer, motor insufficiency is indicated. The sign is of value only when the excretion is delayed. Urine containing salicylic acid gives a dark, brownish-red color upon the addition of a drop of tincture of the chloride of iron.

Klemperer's *oil-test* is more accurate, although disagreeable. One hundred grammes of oil are placed in the stomach by the stomach-tube. In two hours the stomach-contents are removed by aspirating, previously adding a little water. The amount of oil is dissolved by ether, the solution evaporated, and the residuum of oil weighed. Seventy-five to eighty per cent. of the oil should be discharged in two hours.

V. The Absorptive Power. Penzoldt and Faber recommend the administration of three grains of chemically pure iodide of potassium —*i. e.*, free from iodic acid—a short time before dinner. Any fragments of free iodine adhering to the iodide of potash are first carefully washed away. The saliva is tested for iodine with starch-paper and fuming nitric acid. If absorption is active, a violet color is obtained in from six and one-half to eleven minutes, and a blue color in from seven and one-half to fifteen minutes. Zweifel directs that 3 grains (0.2 gramme) of iodide of potassium be administered in a gelatin capsule, and 3½ oz. of water (100 c.c.) taken; iodine is detected in about eight minutes in the saliva. The character of the food taken is said to have considerable influence in retarding the appearance of the reaction, so that the blue reaction may not appear for forty-five minutes.

Boas states that in dilatation of the stomach the reaction may be delayed to two hours, and in cancer as long as eighty-two minutes. Both *motor* and *absorptive* power are recognized also by digestive delay.

Clinical Value of a Chemical Examination of the Vomitus or Stomach-contents. It cannot be gainsaid that the chemical examination of the stomach-contents is of the utmost clinical value. It is just as certain, however, that the results attained by such examination should not be final in the formation of a diagnosis; that alone they do not meet the expectations of clinicians. This is particularly so when we attempt to deduce a scientific therapeusis from such examination. To rely upon the results of such examination alone would lead to failure. The diagnosis, and, therefore, the rational therapeusis, must rest not alone upon a chemical examination, but also upon other methods of examination of stomach-contents, the physical examination of the stomach, the history and progress of the case, and the subjective symptoms. In short, a general view must be taken, and all methods of inquiry employed.

Diseases of the stomach require for their correct estimation broader lines of investigation than almost any other organ of the body. Moreover, the practitioner must not be discouraged if he cannot employ chemical methods with the skill of the laboratory expert. The simple methods detailed above can be conducted by any educated physician. For practical purposes, it is only necessary to determine the total acidity, the presence of free acids, the presence of free HCl, the presence of lactic acid and of the volatile acids.

Finally, the clinician must not be discouraged if the stomach-contents cannot be secured, on account of the contraindications previously detailed. An approximate diagnosis—probably not so precise or final—can usually be made by means of a physical examination of the stomach and a consideration of the symptoms.

The results of the chemical examination have the clinical value estimated herewith. In the first place, we find whether the acidity is increased or diminished.

1. *Diminished acidity*, or anacidity, means deficiency in the amount of HCl secreted. Diminished acidity may be due to functional or organic disease of the stomach. It occurs in fever, in chlorosis, and pernicious anæmia, chronic wasting diseases, including tuberculosis, and acute infectious diseases from functional disturbance of nervous or hæmic origin. It occurs in chronic dyspepsia from irregularities in diet. It is also deficient in congestion, acute catarrh or atrophy of the mucous membranes, and in carcinoma, which apparently modifies gastric secretion.

2. *Increased acidity* may be due to an increase of hydrochloric acid —*hyperacidity*, or to an increase of the organic acids—*increased acidity*. *a*. Hypersecretion of HCl takes place in the early stages of gastric irritation—dyspepsia. It may be increased in gastric ulcer. *b*. Increased acidity (organic acids) may be due to excess of (1) lactic acid; (2) of butyric acid, and (3) of acetic acid. Excess of lactic acid is due to fermentation of carbohydrates from the growth of the bacillus acidi lactici or bacillus lactis aërogenes; of butyric acid, to butyric acid fermentation; of acetic acid, to alcoholic fermentation of the above-

mentioned class of foods. Alcoholic fermentation is often due to the sarcinæ. In short, these acids result from bacterial fermentation, a process which takes place only when there is delayed motor power, or when the normal antiseptic—the HCl—is absent or diminished. Hence, we find these acids in weakness of the muscles, as in dilatation, in organic obstruction of the pylorus, and in cancer of the stomach; while the bacteria are found on microscopical examination.

3. *Free hydrochloric acid is diminished* in acute and chronic catarrh of the stomach (gastritis), in chronic dyspepsia, in ulcer of the stomach and duodenum, in gastric atrophy, in dilatation, in gastric carcinoma (early stage), and from all general causes which lessen the total acidity, including diabetes and Addison's disease. Of course, deficiency of hydrochloric acid means deficiency of functional activity, and goes hand-in-hand with diminished motor and absorptive power. The acid is *increased* in the early stages of irritative dyspepsia and in ulcer of the stomach, and at different periods in the gastric neuroses. The most common causes of increase of HCl are the *gastric neuroses.* Hydrochloric acid is *absent* entirely in advanced chronic gastritis and in the gastric neuroses. In the former there are evidences of fermentation. HCl is often absent in cancer, but unless constantly absent, and two or more other facts of value can be secured, the diagnosis cannot be made on the chemical examination alone.

4. *Lactic acid.* Its presence points to *fermentation,* hence it is associated with lesions that are accompanied by bacterial fermentation.

It is present in carcinoma, as pointed out by Boas. Fermentation is not the only condition in which it occurs. It is nearly always found after a meal of meat, and is known as sarcolactic acid. It may occur in chronic catarrhal gastritis. In cancer of the stomach lactic acid is the most common objective sign. Its absence does not exclude carcinoma. It may be detected before a tumor is palpable. Therefore, if lactic acid is present and free HCl absent, cancer can be pretty safely diagnosticated, particularly if stagnation of stomach-contents is also present. Boas recommends a meal which will not yield sarcolactic acid. It consists of one to two litres of oatmeal gruel, to which a little salt may be added. It should be removed by expression one hour after it has been taken. It is well to remove all food by lavage six hours before the test-meal is given.

The clinical value of the remaining chemical tests and investigations need not be explained. They indicate inability of the gastric function to accomplish digestion, but do not point to any special gastric affection. They are of value in distinguishing between gastric neuroses and an organic disease. In both there are pronounced gastric symptoms; if the examination shows normal digestive powers, a neurosis is indicated.

Gastric Hemorrhage. Hemorrhage of the stomach, *hæmatemesis,* or vomiting of blood, is due to an organic lesion, or the effects of acute irritant poisoning. The blood is vomited. Care must be taken to see that the blood is not from the upper air-passages, and previously swallowed. If hemorrhage is profuse, the blood may cause irritation of the larynx, and provoke paroxysms of coughing. It is often diffi-

cult, therefore, to distinguish between hemorrhage from the lungs and hemorrhage from the stomach.

Hæmatemesis.	Hæmoptysis.
1. Previous history points to gastric, hepatic, or splenic disease.	1. Cough or signs of some pulmonary or cardiac disease precedes, in many cases, the hemorrhage.
2. The blood is brought up by vomiting, prior to which the patient may experience a feeling of giddiness or faintness.	2. The blood is coughed up, and is usually preceded by a sensation of tickling in the throat. If vomiting occurs, it follows the coughing.
3. The blood is usually clotted, mixed with particles of food, and has an acid reaction. It may be dark, grumous, and fluid.	3. The blood is frothy, bright red in color, alkaline in reaction. If clotted, it is rarely in such large coagula, and mucopus may be mixed with it.
4. Subsequent to the attack the patient passes tarry stools, and signs of disease of the abdominal viscera may be detected.	4. The cough persists, physical signs of local disease in the chest may usually be detected, and the sputa may be blood-stained for many days. (Osler.)

The hemorrhage may continue within the stomach without exciting vomiting. The general symptoms of hemorrhage may appear, first, as pallor, dimness of vision, giddiness, or faintness. The blood which comes from the stomach is usually acted upon by the gastric juice, and is dark, clotted, and partly digested. It is often mixed with food. Its reaction is acid. In large hemorrhages the blood may be fluid and of a scarlet color; but if retained for any length of time, it is coagulated. The vomited matter has the appearance of coffee-grounds, when there is a small amount of blood. When large in amount and digested, it appears like tar.

Vomiting is usually followed by movements of the bowels. The matter discharged is of characteristic appearance. It is black or tarry. It is distinguished from hemorrhage of the intestinal canal below the duodenum by the color of the blood. In intestinal hemorrhage the blood is dark red, and not necessarily tarry. The dark stools must not be confounded with the same character of stools seen when iron or bismuth is taken. In rare instances a hemorrhage into the stomach may take place from disease of the lower part of the œsophagus.

Causes. 1. General diseases, from changes in the blood, cause gastric hemorrhage, as scurvy, purpura, hemorrhagic smallpox, yellow fever, acute yellow atrophy of the liver, and severe anæmia, leukæmia, Hodgkin's disease, and pernicious anæmia. 2. Ulcer of the stomach. 3. Cancer of the stomach. 4. Ulcer of the duodenum. 5. Portal congestion, as in cirrhosis of the liver, and other forms of chronic hepatic disease. 6. Disease of the spleen. 7. Congestion due to disease of the heart. 8. In chronic Bright's disease with atheroma. 9. Rupture in aneurism. 10. Vicarious menstruation. 11. Cohen asserts that it occurs in vasomotor ataxia.

Profuse and sudden hemorrhage, in the absence of well-marked symptoms of disease, is in nearly all cases due, either to latent ulcer, or to congestion of the stomach from early cirrhosis of the liver.

General Examination. The objective examination has thus far been confined to a study of the stomach. The student will infer from the previous chapters that in order that on the one hand the possible

cause of the gastric disorder may be determined, or, on the other, the effect of gastric disorder upon the other organs ascertained, they must be examined carefully. Moreover, valuable data in the recognition of gastric affections and the diagnosis of the various forms are secured by such examination. The general appearance of the patient, the state of nutrition, and the degree of strength furnish suggestive facts in the diagnosis. As well said by Stockton :

"The preoccupied and dejected manner observed in those suffering from continued gastric flatulency ; the restless, discomposed behavior, the stooped posture and half-surprised expression often seen in the victims of gastralgia ; the emaciated, weak, and cachectic appearance frequently accompanying chronic food stagnation, are good examples of the value of the general appearance in the diagnosis."

It must be remembered that any local source of irritation distant from the stomach, as the eyes, the nose and pharynx, the uterus and ovaries, and the rectum, may be the primary cause of gastric disorder. The study of the hepatic and intestinal functions assist in the diagnosis. Examination of the urine and the blood may enable us to determine the nature of a gastric morbid process. Even the study of the skin is of importance.

" A sallow, earthy-colored skin, showing improper secretion ; a dry, harsh skin, with too rapid loss of epithelium, showing poor nutrition ; a skin showing œdema, poor capillary circulation, lividity, or acne ; certain forms of eczema, excess of pigment, or syphilides may afford important information as to the digestion, inasmuch as some of these may be the results and others accompaniments of gastric disturbance" (Stockton).

The Blood. Examination of the blood enables us to determine the degree of anæmia which may be the cause of digestive failure. The examination must be exhaustive. If a leucocytosis is present, the gastric neuroses may be excluded. In carcinoma there is not only a severe secondary anæmia, but also poikilocytosis and a multinuclear leucocytosis. Such changes are without doubt the result of interference with the digestion because of motor inactivity. Moreover, certain gastric diseases have specific effects upon the blood. Gastric ulcer may be distinguished from gastric carcinoma, by the fact that digestive leucocytosis occurs in the former while it is absent in the latter.

The Urine. No study of a gastric disorder is complete without an exhaustive examination of the urine. For diagnostic, but chiefly for therapeutic purposes, the presence of renal insufficiency, hyperlithuria, indicanuria, glycosuria, peptonuria, and albuminuria must be tested for.

The Reaction. The reaction of the urine is modified by the state of the stomach. In health the urine is alkaline after a full meal of ordinary character. When HCl is absent from gastric contents, this normal alkalinity does not occur. Alkalinity is rarely seen in gastric carcinoma.

The Chlorides. The chlorides are lessened when a small amount of food is taken ; a similar cause lessens the amount of urea. Both are decreased in carcinoma and in benign diseases of the stomach. But

the chlorides are diminished in carcinoma without a proportionate lessening of the urea. It is this disproportion which is of diagnostic value, as pointed out by Nothnagel, in carcinoma ventriculi.

Diseases of the Stomach Characterized by Fever, with Pain and Vomiting.

Acute Gastritis. The simple variety of acute gastritis varies according to the cause, from a slight attack of vomiting after indiscretion in diet, with ordinary symptoms of indigestion, to the more severe forms ushered in by chill and attended with fever.

In the mild forms there is a sense of fulness and discomfort in the epigastrium, attended with nausea. The appetite is lost, and there may be disgust for food, and the flow of saliva is increased. There is undue acidity. On examination the epigastrium is found to be tender. The onset of the attack is attended with giddiness, flashes of light before the eyes, frontal headache, and some prostration. The pulse is increased in frequency. When this nausea is most pronounced the face is pale and the extremities cold. Vomiting then occurs, the matter ejected consisting of ingesta only slightly changed, with mucus and watery fluid. It is very bitter. It is often colored green from bile-pigment. Another attack of vomiting may be sufficient to give relief, or it may be repeated for twenty-four to forty-eight hours every hour or two. After the stomach is relieved of food, mucus and bile alone are vomited.

Examination of Stomach-contents. The reaction of the vomited matter is neutral or faintly acid. No free hydrochloric acid is present, but later lactic and fatty acids are found. Pepsin is diminished in quantity.

Twelve to twenty-four hours after the gastric symptoms intestinal symptoms may arise. Borborygmi and colicky pains are complained of, followed by diarrhœa, with some tenesmus.

Herpes labialis may occur, and some writers speak of a peculiar odor which is exhaled from the skin. The more severe cases are ushered in with chill followed by fever. The local symptoms are much aggravated. The tongue is furred, and the breath foul. The vomiting is frequent and severe. The skin is livid and the pulse becomes rapid.

Diagnosis. In the acute cases attended by fever it may be mistaken for meningitis, peritonitis, or hepatitis. The same gastric symptoms may usher in an attack of pneumonia. The possibilities of a mistake are to be borne in mind, and in all cases of vomiting with fever due regard must be paid to the possibility of the gastric symptoms being symptomatic only. It must be borne in mind that the same group of symptoms that belong to gastritis accompanies the exanthematous diseases, and diphtheria, dysentery, pyæmia, and puerperal fever. They may be of reflex origin, or due to the action of fever, poison, or ptomaïnes on the stomach. Ewald calls it sympathetic gastritis when the symptoms are the same as in the simple variety, masked, however,

by the primary disease. Sometimes, however, as in the eruptive fevers, attention is directed to the state of the stomach, to the exclusion of other conditions. And often, to the surprise of the student, an eruption or inflammation ensues, which indicates the true nature of the case.

In cases of gastritis, therefore, endeavor to find a local cause for the symptoms. If there is no history of indiscretions in diet, of exposure, of exhaustion, or mental shock, on account of which digestion might be arrested, then inquire for a history of exposure to contagious diseases and look for the earlier evidences of exanthemata. If the result of the examination is still unsatisfactory, examine the condition of each individual organ, particularly bearing in mind meningitis, pneumonia, peritonitis, nephritis, and general infections.

Mycotic and *diphtheritic gastritis* occur secondarily to typhoid fever, pneumonia, pyæmia, smallpox, and sometimes diphtheria. The mucous membrane may be covered with patches in areas or throughout its whole extent.

Some special micro-organisms irritate the gastric mucosa, as the anthrax bacillus and the sarcinæ and yeast fungi in cancer and dilatation of the stomach. Rarely tuberculous inflammation with ulceration takes place, and other micro-organisms have been described. Klebs found the bacillus gastricus with numerous spores in the tubules, as a consequence of which a gastritis was set up.

The mucous membrane itself escapes infection from micro-organisms, because of the character of its secretion. The acid gastric juice is antagonistic to and causes the death of micro-organisms. Tuberculosis, for instance, rarely attacks the stomach for this reason.

Phlegmonous Gastritis. This is a very rare affection, in which the inflammation is seated in the submucosa and leads to perforation. The onset is sudden. The chief local symptom is intense pain in the epigastrium, with a burning sensation. There are great acidity, dry tongue, and absolute anorexia. The fever is high and characterized by delirium. Chills usually accompany it. The pulse is small, rapid, and irregular. The matters vomited are first mucus, then pus. The patient is extremely restless and anxious, even delirious, and early passes into coma. Death takes place from collapse. It is impossible to make an absolute diagnosis, as local peritonitis and abscess of the liver are characterized by the same symptoms. In *abscess* a tumor may form in the epigastrium. It may occur idiopathically, but it frequently occurs in septicæmia, and follows trauma.

Toxic Gastritis. This form of gastritis is allied to the former in the severity of general symptoms. It is the result of the swallowing of irritating poisons, of which phosphorus, arsenic, bichloride of mercury, and caustic acids and alkalies are the most common. It is attended by inflammation of the mouth, œsophagus, and stomach. There are salivation and dysphagia, and constant vomiting of blood, often with shreds of mucous membrane. The patient is restless, and may have convulsions ; collapse readily develops. In mild cases, in which the local effects of the corrosive substance have been mitigated by proper antidotes, sloughs occur, leaving behind ulcers on the mucous membrane, which, after healing, result in deformity or stenosis of the œsophagus.

Some cases are attended by other symptoms peculiar to the special poison. Thus with arsenic there are choleraic symptoms; in phosphorus-poisoning the symptoms come on late after its ingestion, and are attended by jaundice and symptoms of acute yellow atrophy.

Diseases of the Stomach Characterized by Indigestion.

Functional Disorders of the Stomach. The Neuroses. Functional disturbances of the stomach are due to impairment of the motor power of the stomach, impairment of the secretory function and of the sensory function. The following table of Ewald, as given by that distinguished authority, is a classification of the various neuroses midway between the symptomatic and the etiological:

The Neuroses of the Stomach.

1. Conditions of Irritation.

a. Sensory.	*b. Secretory.*	*c. Motor.*
Hyperæsthesia.	Hyperacidity.	Eructation.
Nausea.	Hypersecretion.	Pyrosis.
Hyperorexia.		Vomiting.
Anorexia *ex* hyperæsthesia.		Colic.
Parorexia.		Tormina ventriculi.
Gastralgia.		

2. Conditions of Depression.

Polyphagia.	Anacidity.	Atony.
Anæsthesia.		Insufficiency of the pylorus and cardia.

3. Mixed Form.

Gastro-intestinal neurasthenia (dyspepsia nervosa).

4. Reflexes from Other Organs upon the Gastric Nerves.

Reflexes from the brain, eyes, spinal cord, kidneys, liver, sexual organs, and intestines manifest themselves in the forms mentioned in 1 and 2.

It must not be supposed that each of the above-named symptoms occur in an individual, or that functional disturbances may be limited to alterations of the sensory and secretory or the motor apparatus, respectively. They do not occur, as Ewald states, as distinct independent diseases, but usually in groups, "either appearing simultaneously or closely following one another during the course of the malady, passing before us like an ever-changing scene." They may arise directly from disease of the stomach, or reflexly from disease of other organs, as the brain, the spinal cord, uterus, kidneys, liver, eyes, and nose.

Etiology. Gastric neuroses are of most frequent occurrence in women, especially during the years from puberty to the menopause. The accidents of childbirth are predisposing factors. In both sexes they are of most frequent occurrence after the age of twenty years, because individuals are subjected to causes which lead to neuroses at this period of life. The gastric neuroses occur in all conditions of patients. They are more likely to occur in those who are poorly nourished or anæmic; although persons who are distinctly robust may also suffer. While more common in the residents of cities, they may occur in farmers and others accustomed to an open-air life. Although we are

oftenest called upon to treat them among the better classes, nevertheless a large number of cases are seen among the poor. To analyze more closely the predisposing causes, we have to study individually all conditions and circumstances in life which lead to wear and tear, as in business or social affairs. The causes which Beard and others have forcibly pointed out as factors in the production of neurasthenia are especially prevalent in this country.

In men, excessive devotion to business, or dissipation; in women, excesses in social life, or the restraint of home cares, with, unhappily, too often, the irritation of marital relations, are the predisposing factors which lead to the development of this class of cases. Often patients in the large cities are subject to the neuroses in the spring after the dissipations of the winter. Behind this excess there is, no doubt, in the majority of cases, a nervous temperament that is responsible for the bringing out of the symptoms, particularly if, combined with this temperament, the patients live in an unhygienic way in regard to exercise, ventilation of their dwelling-places, and drainage, combined with improper diet.

Symptoms. With the gastric neuroses other symptoms of *neurasthenia* are present, and the patient may seek advice for these symptoms, such as headaches of various kinds, changes in the mental condition, vertigo, insomnia, neuralgias, and all forms of paræsthesia. Intimately connected with the neurasthenic state is that of hysteria, and therefore in gastric neuroses *hysterical manifestations* are most common. It may be impossible completely to define the border-line between neurasthenia and hysteria, and the gastric symptoms of the former are the gastric symptoms of the latter. While, therefore, general neurasthenic symptoms are prominent, in order to reach a diagnosis upon which proper lines of treatment can be based, the condition of the individual must be viewed as a whole, and no one symptom or group of symptoms exaggerated in our minds.

Varieties. Ewald has divided the neuroses into those which arise from (*a*) *irritation*, those which arise from (*b*) *depression*, and (*c*) those in which both are combined—*mixed neuroses*.

(*a*) **1. Sensory Neuroses of Irritation.** Hyperæsthesia. The first result of irritation is *hyperæsthesia* of the stomach, which is indicated by a feeling of *fulness* and *tension*, and of *nausea*. The sensation is allied to the normal, and is also seen in chronic gastritis, as well as in hysteria, meningeal irritation, cerebral tumors, and other diseases of the nervous system. The increased irritability is such that the gentlest irritant excites discomfort or a painful sensation. There is a continuous sensation of heat or cold, of gnawing, or pulling, or burning in the organ. The local sensation reflexly influences the physical life of the patient, so that *hypochondriasis* in some form attends it. The sensations may be relieved by food, to become worse if the stomach is emptied, although in the larger number of cases the trouble is aggravated during digestion. The sensations are likely to be aggravated by fasting a longer period than usual, or by restriction of the diet. Excesses may aggravate them, and, on the other hand, they are said to follow debilitating states. Some foods, such as shell-fish, crabs and

lobsters, or oysters, and strawberries, are likely to increase the peculiar sensations in the epigastrium, exciting mild depression, or burning, or even nausea. The excitation from these foods is usually due to peculiar idiosyncrasies of the individual. On account of the same idiosyncrasies, pruritus, erythema, and urticaria occur, with headache and some fever.

Deviations from the Sense of Hunger. *Hyperorexia.* When hunger is exaggerated it is known as boulimia, or *hyperorexia.* It may be temporary or permanent. When permanent it is obstinate, weakening, and exceedingly unpleasant. It may occur alone or be a symptom of various diseases of the nervous system, as manifest disease of the brain, neurasthenia, hysteria, and psychoses. It complicates such disorders as diabetes, and may be of temporary duration in convalescence from acute disease. The disorder accompanies migraine, or hypochondriasis, and exophthalmic goitre. Analogous to it is perversion of the appetite, as seen in pregnancy, in children, and in mental disorders.

Anorexia. Loss of appetite, or repugnance to food. In the first instance, there is simply loss of appetite ; in the second, there is repugnance toward food, or nausea at the sight of it. Loss of appetite accompanies dyspepsia in all forms. In the gastric neuroses it occurs spontaneously, or is due to hyperæsthesia of the stomach, and therefore may arise from central or peripheral conditions of irritation. It is commonly seen following central nerve perturbation. The patient is hungry, and sits down to the meal fully prepared to satisfy himself. The first mouthful is at once followed by anorexia, which may almost amount to nausea. On account of these symptoms the patient eats less and less of solid food, which soon results in disturbance of nutrition affecting the higher centres. On the other hand, profound mental disturbance may be an exciting cause, so that after the death of a friend, or shock of any kind, the patient is unable to take food. Loss of appetite may be the only manifestation of the gastric neurosis, but because nutrition is so seriously interfered with, it soon results in other local or general symptoms. Fenwick points out that its relationship to emaciation and enfeeblement is such that grave organic diseases may be simulated. Thus it may be mistaken for phthisis, and a general examination alone is sufficient to distinguish it.

Gastralgia. *Pain* in the stomach occurs in organic disease, as in ulcer or cancer, or forms of gastritis. It also attends a gastric neurosis, and may be the only symptom of this neurasthenic state. Such pain is functional, and is found in anæmic, neurotic women. It may, however, occur in all classes. It is characterized by sudden pain in the epigastrium, usually without regularity, though at times it may be distinctly periodic. There may not be any definite relationship between the attack of pain and the taking of food, though it is most apt to occur when the stomach is empty. Some kinds of food may aggravate it, though, in general, eating relieves the pain. If the epigastrium is examined, it will be found to be free from tenderness, and indeed pressure with the palm of the hand may give relief. The pain is of an agonizing character, sometimes sharply localized, or again diffuse.

It may even resemble the girdle-sensation. On account of the severity of the pain the patient may be compelled to double himself up to relax the abdominal muscles. The breath is short, and speaking is done in a whisper. The attack is attended by more or less collapse, and the patient may complain of the sensation of impending death. There is pallor of the face, which is distorted with pain, and the brow is covered with perspiration. The pain may radiate along the spinal nerves in close situation to the stomach, and there is often vigorous pulsation of the abdominal aorta.

The attack may last but a few minutes or continue for hours. It sometimes terminates suddenly with vomiting, or is relieved as soon as food is taken. After the attack the patient is exhausted and relaxed, and passes an abundance of urine of low specific gravity.

The gastralgias that are due to disease of the central nervous system are often most puzzling. Rosenthal has written exhaustively on this subject. Types of gastralgia of this character are seen in the *gastric crises* of tabes, first described by Charcot. Recent observers have found that it is due to sclerotic degeneration of the vagus nucleus. The patient is suddenly seized with severe pains, which may begin in the groin and ascend along both sides of the abdomen to the epigastrium, to which point they are fixed. Pain in the shoulders occurs at the same time. The pains are characteristic of lumbar ataxia in their lightning-like rapidity. With the pain the heart's action is increased in rapidity and force. There is no rise in temperature. At the same time there is uninterrupted and painful vomiting, which is attended by nausea and vertigo. The gastric pain may continue uninterruptedly for two or three days. It belongs to the pre-ataxic period, so-called, but is almost sure to continue throughout the whole course of the disease. The nature of the stomach-contents bears no relation to the pain. The frequency of the attacks is variable. They may recur at long periods, or as frequently as once a month or once a week. Another special characteristic is the sudden relief that is given without cause.

Neurasthenic Gastralgia. Neurasthenic gastralgia occurs in patients who are suffering from neurasthenia, and is divided by Rosenthal into two forms, the one irritative, the other depressant; these are related by transitional forms. The early symptoms of neurasthenia (*q. v.*), particularly in the irritative form, with painful points in the nape of the neck and between the scapulæ, or often lower down on the vertebræ, with neuralgias and paræsthesia in the upper and lower extremities, are attended by periodical gastralgia. The gastralgia is characterized by a boring sensation which, during the attack, radiates over the lower ribs to the median line. It is accompanied by vasomotor symptoms and symptoms of cerebral anæmia. In the *depressant* form the patient complains of weight and fulness, or a dragging sensation after eating, which is constant instead of paroxysmal. The neuralgic pains are not so marked, motor exhaustion is not so prominent, and the pain in the back is not so intense as in other varieties. In both instances on deep pressure over the region of the nerve-plexuses which follow the bloodvessels in the abdomen, there is sharp and unpleasant pain radiating to the epigastrium. Burkart considers these

painful points to be present in all cases, while Richter believes that pressure over the stomach and abdomen is not painful. With such pain there is usually increased pulsation of the abdominal aorta, particularly during the time of the paroxysm. In neurasthenic gastralgias there is increased sensitiveness to the electrical current and increased irritability of the sensory nerves of the trunk, which may also be extended to the limbs.

Neurasthenic gastralgia must be distinguished from the gastralgia of organic disease and the gastralgia of hysteria. The gastralgia of organic disease is recognized by observing the condition of the stomach when fasting and by studying the secretion. In organic disease there is retarded digestion ; in gastric neuroses digestion is completed in the normal limit of time—seven hours. Hysterical gastralgias are recognized by the presence of the usual symptoms of hysteria, in which the psychical factors occupy a prominent place, associated with convulsions, paralyses, pupillary inequalities, hemianæsthesia, and electrical sensibility. Most characteristic, however, is the alternation of hysterical gastralgias with neuralgia, or neuroses in other organs.

(*a*) **2. Secretory Neuroses of Irritation.** Hyperacidity and Hypersecretion. Hyperacidity is the increase of the normal amount of hydrochloric acid secreted, due to a neurosis of the secretory function. Hyperacidity begins when the amount of acid in the fluid withdrawn from the stomach in the usual way is between 60 and 70 per cent. It must not be forgotten that it is a symptom of gastric ulcer, but it exists as a neurosis independent of any organic lesion of the stomach. It has been observed in nervous diseases, as hysteria and melancholia, and as a reflex symptom in gallstones and renal calculus.

Hypersecretion occurs in two forms, the *periodical* and *constant*. The acid is not necessarily increased. The periodical occurs after eating ; it has no direct connection with food. It is seen in neurasthenia and locomotor ataxia. In chronic hypersecretion the gastric juice, which is usually hyperacid, is in excess, so that the fasting stomach may contain large quantities, even to a pint and a half, without food and only slightly tinged by bile. In chronic hypersecretion the digestion of starches is delayed, but that of albuminoids is very prompt. After an abundant meal consisting of meat and starches the meat disappears entirely. Hypersecretion occurs in about half of all the stomach disorders, according to Riegel. It is more common in men than in women. The acid fluid causes the hyperæsthetic conditions in the gastric region previously described. Pain and eructation, heartburn or gastralgia, vomiting of sour masses, occur with the digestive disturbances of chronic gastritis. The tongue is usually clean and the appetite increased rather than diminished. As a result, atony of the muscular coat takes place, followed by gastrectasia. The neurosis is then converted into an organic lesion, and the symptoms of dilatation arise.

Reichman's disease is a hypersecretion of the gastric juice, and there are two forms—the acute, which is generally of nervous origin, and the chronic. The latter is seen in emaciated persons ; the stomach is

dilated, and succussion-splash is readily obtained. The diagnosis is made in part by examination of the gastric contents, which are removed five to six hours after the meal. The quantity will be found large. On standing, the material becomes separated into three layers —an upper, frothy layer; a middle, turbid, yellowish layer, and a lower, consisting of starchy matter. In order to determine that hypersecretion exists, the stomach-contents are removed in the evening, and the viscus washed out thoroughly until the water is no longer acid in reaction. The patient receives no food until the next morning, when, after the proper interval, the contents of the stomach are again evacuated. From 30 to 600 c.c. (1 to 19 ounces) of fluid will now be obtained, which, on examination, proves to be active gastric juice. The disease is chronic.

In order to make a diagnosis the secretions must be secured while fasting. The patients usually improve on albuminous food, which differentiates it from gastralgia and pyrosis of acid fermentation. Alkalies give temporary relief.

Gastroxynsis is a gastric neurosis in which, after mental overexertion or profound emotional disturbance, there is sudden vomiting of acid fluid, continuing for a considerable time. It is closely allied to migraine.

(*a*) **3. Motor Neuroses of Irritation.** Eructations. *Eructations* and *belching* are phenomena of the gastric neuroses of motor origin. They usually occur in hysterical subjects rather than in neurasthenics. In the latter they are associated with other sensations, particularly oppression and tension in the epigastrium. In hysteria they occur alone. There is increase in the contractility of the stomach, the pyloric sphincter contracts powerfully, and the stomach is distended; gas is expelled at the cardiac end of the stomach. They may be due to paralysis of the cardiac end of the stomach rather than to contraction of the pyloric end. They occur involuntarily generally. They must not be confounded with the pseudohysterical vomiting which Bristowe has described. In the latter instance the gas is raised from the œsophagus by contraction of the muscles of the neck. Hysterical eructation is very frequently of œsophageal origin. The belching is loud and may occur in paroxysms. The gas is odorless, and hence is distinguished from the gas of dyspepsia and fermentation; it is in all probability the result of the swallowing of air.

Pyrosis. *Pyrosis*, or *heartburn*, is the raising of sour masses from the stomach. The stomach-contents are not necessarily hyperacid. If acid, as in the normal gastric juice, or hyperacid, the regurgitation causes severe acrid and burning sensations. It is probably due to heightened contractility of the muscular coat of the stomach with pyloric contraction, which overcomes the weaker cardia.

Pneumatosis. Excess of gas in the stomach. When the stomach is overdistended the diaphragm is pushed up, pressing on the heart. The patients are seized with severe dyspnœa. At first inspiration is difficult, and finally both inspiration and expiration become difficult. Palpitation of the heart and pulsation of the peripheral arteries take place. There is fulness of the head and a sensation of impending

death. The patient may become unconscious. Relief can only be afforded by belching, when the attack rapidly subsides. Introducing a stomach tube gives immediate relief.

Nervous Vomiting. (See Subjective Symptoms and Gastroxynsis).

Tormina Ventriculi. *Peristaltic Unrest.* Characterized by borborygmi and gurgling, which begin immediately after eating, are heard at a considerable distance, and are a source of great annoyance. It is a common symptom of the gastric neuroses.

Rumination (*Merycismus*). Rumination is a rare condition in which the patients regurgitate and chew the cud like ruminants.

(*b*) **1. Secretory Neuroses of Depression.** Anacidity. *Anacidity* of the gastric juice as a neurosis is found in hysterical persons and in neurasthenics. (See chemical examination Absence of Hydrochloric Acid).

(*b*) **2. Sensory Neuroses of Depression.** Anæsthesia. In conditions of depression *polyphagia*, or the want of a feeling of satiation occurs; if gluttony is excluded, it is a morbid condition of extreme rarity.

(*b*) **3. Motor Neuroses of Depression.** Atony, or Atonic Dyspepsia. It accompanies gastritis; it also occurs as a primary neurosis. The innervation of the nerve-centres regulating peristalsis is disordered. The primary disorder may be local or central. The movement of the chyme is tardy or insufficient. Atony should be applied to the disease of the motor function only, or, as Rosenbach states it, to insufficiency of the stomach. The symptoms develop gradually. At first oppression during digestion occurs, with swelling and fulness of the stomach.

There is mental and physical torpor during the time of the digestive act. The symptoms become aggravated, and eructations occur, vomiting begins, and gradually the fermentative symptoms become most pronounced. At this period it is putrid, or fermentative dyspepsia. By the usual tests the motor power of the stomach is found to be diminished. The secretions are also scanty.

Relaxation at Orifices. *Relaxation of the Cardiac and Pyloric Ends of the Stomach from Conditions Resembling Paralysis.* When the cardiac end is relaxed eructations and regurgitations occur. If large quantities of the material from the stomach are regurgitated and expectorated, the condition is pathological. It may lead to serious changes in nutrition. It may exist for years without bad results. It must not be confounded with the regurgitation from diverticula of the œsophagus. In the latter regurgitation is produced at will.

(*c*) **Mixed Neuroses.** Nervous Dyspepsia. According to Ewald, this is the true gastric neurasthenia, which combines all forms of gastric neuroses. The clinical picture is made up of a combination of various neurosal symptoms. Leube considers nervous dyspepsia a group of symptoms of a cerebral nature due to abnormal irritability of the sensory nerves of the stomach during the normal digestive processes, the symptoms of which are hyperæsthesia and nausea, hyperorexia, anorexia, parorexia, and gastralgia. He thinks the true

peptic activity of the stomach is unchanged. Although the anatomical or physiological explanation of the condition is difficult, the clinical symptoms are those of irritation of paralysis, the manifestations of which are intermingled, sometimes one and sometimes the other being most prominent. (See table, page 797.)

The one characteristic feature is that the symptoms are mild. With severe forms of gastralgia nervous vomiting and boulimia do not occur. Symptoms of intestinal indigestion are usually associated in a mild degree. Constipation is of the most common occurrence, although in some cases there is diarrhœa. In other cases the intestinal indigestion is much aggravated, with mild gastric disturbances and anorexia, repugnance toward taking food, furred tongue and mild nausea, constipation and colicky pain, either diffuse or in separate painful spots. . The abdomen is distended and tympanitic, sometimes to a marked degree. It is called *flatulent dyspepsia*. Along with the gastric and intestinal symptoms, the general nervous symptoms to which the term neurasthenia is applied are present. These nervous manifestations sometimes precede the local gastric symptoms, but as the latter develop the former become more aggravated. The dyspeptic conditions, as Ewald puts it, are on a neurotic basis, or are such as may occur in the form of reflex neuroses in chlorosis, menstrual disorders, uterine and ovarian disease, and intense physical or psychical excitement. As far as we know there are no great alterations in the chemical functions when anatomical and pathological changes are absent. An indigestion of short duration, a mild catarrh, recurring hyperæmia, have been the primary cause of nervous symptoms in the digestive organs.

Diagnosis. There are no characteristic symptoms, and the student must bear in mind that it may be necessary to make several examinations and listen to the story of the subjective symptoms frequently before a conclusion can be arrived at. This is all the more necessary because of the frequency in which organic lesions and neurasthenic conditions are present at the same time. The course of the disease must be observed for a long time, all possible causal factors investigated, and all the general signs of neurasthenia carefully considered. In addition, it may be necessary to use therapeutic tests. If the possible organic diseases are not relieved by such measures, there must be a deeper basis for the gastric symptoms. Just as in neurasthenia and in neurasthenic states elsewhere, the peculiarities, idiosyncrasies, and all the associations in the life of the individual must be considered in connection with the general and local symptoms of the neurasthenic state. Great stress must be placed upon the study of individual symptoms, their mutual relationship, and their changeable occurrence. In gastric neurasthenia gastralgia is more diffuse than the pain of ulcer or cancer of the stomach. It is not so much dependent upon food as either of the others, particularly ulceration. In gastric neurasthenia vomiting is rare. The vomiting is composed of mucus mixed with bile and food in various stages of digestion. It is never bloody, nor does it contain decomposed masses. Hysterical vomiting occurs with ease and regularity compared with the vomiting of neurasthenia. The vomiting in neurasthenia is bitter, due to the presence of peptones. In gastric neurasthenia the

stools are changeable in character. They do not contain undigested remnants of food, or mucus, or blood. The form of the feces is variable.

Differential Diagnosis. Neoplasms, ulcers, strictures, and dilatation are distinguished by physical signs or characteristic symptoms. In gastric neurasthenia the stomach should be empty seven hours after taking a meal. The results of the chemical examination are not sufficiently definite for diagnostic purposes, for at times the same chemical changes are present, as in ulcer, carcinoma, and chronic catarrh. The diagnosis must be based largely, as previously intimated, upon prolonged observation and a carefully taken history, and upon the general condition of the patient. The cases must not be mistaken for costal neuralgia, although it is not usually easy to be led astray. Reflex gastric neuroses are seen, as indigestion, gastralgia, or vomiting. The types are interchangeable, although vomiting occurs in the more acute reflexes, indigestion in the more chronic. The cerebral disorders which give rise to vomiting are meningitis, abscess, and tumor. The vomiting may be transitory, or may be persistent. There is usually hypersecretion of the gastric juice. The vomiting may usher in the disease or develop during its course. If vomiting is of long standing its possibly reflex origin should always be investigated. (See Vomiting.)

Gastralgia is sometimes a reflex from lesions in the cervical and dorsal portions of the cord ; not only in the posterior columns, but also in disseminated sclerosis. Vomiting occurs, and the attack is known as a *gastric crisis.*

Chronic dyspepsia is a frequent reflex disorder of diseases of the sexual organs, as amenorrhœa and dysmenorrhœa, in the climacteric period, and in chronic inflammations of the uterus. In malpositions and tumors, and in pelvic exudations with traction, in ulcers, in ovarian tumors, the so-called dyspepsia uterina of Kisch is common.

Chronic Gastritis. *Causes.* 1. Previous attacks of acute gastritis.

2. The local irritation of badly cooked or poorly masticated food, and of alcoholic and other beverages.

3. The local irritation of urea in chronic Bright's disease, and of products of putrefaction in constipation.

4. In anæmia chronic gastritis is of frequent occurrence, and in venous congestions from any cause, but particularly from disease of the heart or diseases which interfere with the portal circulation. It occurs secondarily to diabetes, gout, rheumatism, nephritis, and tuberculosis.

5. It is a constant attendant upon local disease of the stomach, as cancer, dilatation, and ulcer, and of local disturbance of the circulation.

The symptoms are those of *chronic indigestion.* There is a dry, pasty, or salty taste in the mouth, especially in the morning. The tongue is coated over its entire surface, or has red patches at the base ; its papillæ are always swollen and its edges marked by the teeth. Aphthæ recur frequently. The lips are dry and often chapped. The appetite is poor or capricious. Although there is no great thirst, the patients crave fluids with their meals, and acid drinks are grateful. After eating there is a feeling of oppression and distention in the epigastrium, frequently followed by belching. The gaseous

eructations are odorless or foul, and rancid regurgitation with pyrosis is frequent. The acidity is due to fatty acids and not to hydrochloric acid, as in hypersecretion. Vomiting is invariably present, but occurs irregularly. It is usually preceded by nausea. The most characteristic form is that in which mucus is vomited in the morning on rising. Constipation usually exists; it may alternate with diarrhœa. There are flatulency and rumbling in the intestines.

General Symptoms. The nervous symptoms are the most pronounced. The mental activity is diminished, there is a feeling of languor or torpor, especially after eating. Headache is frequent after eating, and the patient may become morose and hypochondriacal. Attacks of vertigo are common. Itching of the skin and coldness of the extremities are not rare. Sleep is deeper and longer than is natural, but is disturbed by dreams, and is not refreshing. Yawning is frequent. Pharyngitis usually attends the attack, with hacking cough and expectoration, or hawking of mucus.

The pulse may be weak and irregular, and at times there is an evening rise of temperature. The urine is scanty, high-colored, and usually loaded with urates.

Three forms are seen: (1) *Simple chronic gastritis;* (2) *chronic mucous gastritis;* the term "chronic catarrh of the stomach" is applied to both conditions. If the condition lasts a long time, it results in (3) *atony*, with dilatation of the stomach, or with atrophy. *Atrophy*, or *atrophic gastritis*, is secondary to the chronic form, or to stenosis of the œsophagus, or to cancer. The symptoms are those of pernicious anæmia. *Cirrhosis* of the stomach is also a sequence of gastritis. It is rare, and the symptoms are not characteristic of a spinal lesion. They are those of the primary disease.

Examination of the Stomach-contents. In *simple gastritis* the stomach, after digestion is completed, contains a small amount of slimy fluid. Hydrochloric acid is diminished in quantity after a test-breakfast; lactic acid and the fatty acids are present, as previously noted. Pepsin and the milk-curdling ferment are absent or diminished. In *mucous gastritis* there is subacidity. It differs from the simple form in the excess of mucus only. In *atrophy* the hydrochloric acid and pepsin are diminished, or absent altogether after the test-breakfast. The fasting stomach is empty. There are no fermentation acids. Atrophy must be distinguished from *cancer* and *subacid neuroses.* The latter occur in younger individuals than those subject to atrophy. A bloody tinge in the stomach-contents, or hemorrhage, may be the only distinguishing mark of cancer. It is often impossible to make a diagnosis.

Diagnosis. The diagnostic features of chronic gastritis are: First, long duration; second, persistence of local symptoms; third, recurrence of local symptoms after food, the symptoms being aggravated by stimulants, or stimulating food; fourth, moderate pain; fifth, absence of cachexia; sixth, absence of tumor; seventh, flatulency. Hemorrhage is rare, and there may or may not be vomiting, while the quantity of hydrochloric acid is variable. Finally, the cause is usually definite.

Dilatation of the Stomach (Gastrectasia). (See Plate XXXVIII., Fig. 1.) It is caused by obstruction at the pyloric orifice, either from cancer, the cicatrix of an ulcer, or fibrous stricture. It follows atony and degeneration of the walls of the stomach which occur in the course of chronic gastritis. It may attend paralysis of the stomach. Excessive eating or drinking are the only probable causes independent of organic disease. Hence, we have (1) obstructive and (2) atonic dilatation.

The dilatation may be *acute*. The term *acute paralytic distention* is also applied to this condition. The cases are extremely rare. There is sudden enlargement of the upper portion of the abdomen, with pressure upon the surrounding structures. The heart is dislocated and its action much interfered with; collapse follows and may end in death. At first there may be some belching, but the patient is soon unable to remove the gas, and suffers from extreme discomfort, palpitation, and dyspnœa. The vomiting may occur at once or later. It is persistent and excessive. On physical examination the stomach yields the same physical signs as in chronic dilatation.

Chronic dilatation develops slowly. The *symptoms* of it are superimposed upon the causal disease. There is marked dyspepsia, with flatulency, pyrosis, and other symptoms of fermentation. The tongue is pale and furred, or red, smooth, and shiny; or it may be soft and flabby. If frequent vomiting has attended the causal disease, it now occurs at longer intervals; the amount is excessive, greater than the normal stomach would hold, and is made up of partially digested and fermented food and large amounts of mucus. The stomach-contents contain sarcinæ, torulæ, and other products of fermentation. Hydrochloric acid is usually absent, but there is a large excess of lactic and fatty acids. The patient loses flesh and strength; becomes irritable, depressed, and more or less melancholy. The patient is subject to vertigo and to attacks of nocturnal asthma. The nervous symptoms of chronic gastritis are also present.

Sleeplessness is quite common. In some cases there is excessive thirst because of the small amount of nutriment and fluid absorbed. Cardiac palpitation and irregularity are common, and dyspnœa may occur on account of the distention. Tetany has been observed in cases of dilatation, especially after lavage.

Physical Examination. The diagnosis is not complete without *physical examination.* On *inspection* the abdomen is large and prominent, and the outline of the stomach can sometimes be seen. Peristaltic movements of the organ are often seen. The movement is from left to right. The heart is lifted upward. On *palpation* the peristalsis can be felt, and with one hand on the stomach, tapping with the other, a *splashing sound* can be detected. Or the hand may be placed over the stomach (patient standing) and the body quickly shaken. On palpation the striking or pushing hand should be compressed over the false ribs. A tumor can sometimes be felt in the region of the pylorus, or below the umbilicus. On *percussion*, when the stomach contains gas, a tympanitic note is heard. After drinking water dulness may be detected between gastric and intestinal tympany if the patient stands up. The dull note disappears when he resumes the recumbent posture.

Before taking water tympany is not so marked in the upright as in the recumbent posture, because the stomach is dragged back or down. The tympany extends high up in the chest on the left side, so that Traube's half-moon space is exaggerated. It may extend as high as the fourth interspace on the left side. Cardiac dulness is increased and the apex of the heart is lifted upward and to the left. In the axillary region the tympany may extend as high as the sixth rib. There is usually atrophy of the spleen, so that unless very careful light percussion is performed the splenic dulness cannot be brought out. The lower limit extends below the transverse umbilical line, and may even extend midway to the pubis. If there is *gastroptosis*, the half-moon space becomes dull on percussion, the stomach tympany falling to a lower level. On *auscultation* succussion can easily be elicited. Sometimes the sound is sizzling, as if there was effervescence. Heart-sounds may be transmitted clear and metallic over the tympanitic stomach. With *auscultatory percussion* the border of the stomach can often be defined accurately. Percussion must be commenced far away from the stomach-limit and conducted toward it. (See Examination of the Abdomen.)

Stenosis of the Pylorus. Usually, obstruction is caused by malignant disease. Hypertrophic stenosis occurs in rare instances and leads to dilatation, as indicated above. The condition may be congenital or acquired.

Acquired stenosis may be the result of chronic gastritis, or develop independently, sometimes as part of a general proliferation of connective tissue. (See case of author, *Path. Soc. Trans.*, vol. xi. 1881-83, p. 216.) If, to the physical signs of tumor of the pylorus, be added the signs and symptoms of dilatation, we have the clinical picture of hypertrophic stenosis of the pylorus. It is extremely rare to find complete obstruction.

Congenital hypertrophic stenosis, as Metzler and Caudley point out, has for its characteristic features: (1) Vomiting, occurring without apparent cause and persisting in spite of treatment; (2) the absence of bile from the vomited matter; (3) obstinate constipation; (4) marasmus; (5) the presence of a tumor in the region of the pylorus; (6) the absence of abdominal distention except from dilatation of the stomach itself in some instances; and (7) the absence of signs or symptoms of gastritis and of the more common forms of intestinal obstruction. Diagnosis depends entirely on the characteristic symptoms arising during the first few weeks of life and the presence of a tumor.

Diseases of the Stomach Characterized by Pain and Vomiting.

Cancer of the Stomach. The clinical symptoms are varied. Gastric cancer may occur without any symptoms whatever, and be discovered after death from other causes. On the other hand, general marasmus and cachexia may be present, without local symptoms. In some cases the gastric symptoms are slight, and obscured by the symptoms of secondary growth in the liver or peritoneum.

Typical cases are those which occur late in life, with symptoms of chronic gastritis. These symptoms may continue for months before anything further is observed. Gradually the uneasiness and discomfort after eating increase to actual pain. Loss of appetite is marked, and in spite of careful treatment there is loss of flesh and strength. The usual vomiting of chronic gastritis gradually becomes more frequent. The general appearance of the vomitus is at first like that of chronic gastritis. Soon it becomes streaked with blood, or a moderately large hemorrhage may take place. The vomited matter is dark in color, like coffee-grounds in appearance. The relation of vomiting to the time of taking meals depends upon the seat of the disease. If at the cardiac end of the stomach, the vomiting may take place at once. If in the greater curvature, within twenty minutes or one hour and a half after taking food. If at the pyloric orifice, the vomiting is delayed several hours. As the disease advances, and obstruction becomes more complete at the cardiac orifice, food is immediately regurgitated, unless secondary dilatation of the œsophagus takes place. When there is gastric dilatation the vomiting may take place at longer intervals and be characteristic of the vomitus of dilatation. Constipation is the rule.

Tumor. After the symptoms of chronic gastritis have continued for some time without relief a tumor may be detected, depending upon its situation and size. (See Tumors of Abdomen.) If the growth is situated at the cardiac orifice of the stomach, it is often impossible to detect it. If at the pyloric orifice, the tumor is found to the right of the median line above the umbilicus, but may be forced down by the weight of the stomach and felt at the umbilicus. (See Plate XXXVIII., Fig. 2.) When dilatation follows pyloric tumor it may be still lower down, as in a case of the writer's, in which it was found two inches below and to the right of the umbilicus. In tumor of the greater curvature the mass is detected below the margin of the ribs on the left side, and may be as low down as the umbilicus. If the greater curvature is involved, the organ usually atrophies, and hence the physical signs indicating the lower border of the stomach are higher up than in health.

It is necessary to exclude tumors due to other causes. This is sometimes difficult—indeed, as far as the location and physical characters are concerned, often impossible. The most pronounced diagnostic feature of tumor of the pylorus is the occurrence of secondary dilatation of the stomach. For a differential diagnosis of tumors in this region, see Palpation of Abdomen.

Symptoms due to Metastasis. The *liver* is the most frequent seat of secondary growths. The organ enlarges, and its surface is covered over with nodules. (See Plate XXXVIII., Fig. 2.) Jaundice occurs in rare instances. The enlarged liver may cover the stomach and hide the local mass. The *inguinal* glands enlarge. At times there is enlargement of the *supraclavicular glands*, suggestive also of intra-abdominal carcinoma, from other causes.

The general symptoms are those of *emaciation* and *cachexia*. The *emaciation* is extreme, and in some cases may be out of proportion to the local symptoms.

The symptoms of *cachexia* are those of emaciation and anæmia. The *anæmia* becomes profound. The pallor of the face is striking, often it is of a yellowish and straw-colored hue. It must not be confounded with jaundice—examination of the conjunctivæ is usually sufficient to distinguish the two. The skin is flabby, and the subcutaneous fat is entirely lost; the emaciation is not so marked as in cancer of the œsophagus, except when there is complete cardiac stricture. The nutrition of the skin suffers, boils are common, and ulcers may occur. Subcutaneous hemorrhages are seen in the terminal stages on the backs of the hands, on the dorsum of the feet, on the legs and arms. There is slight œdema of the ankles.

General atrophy of the internal organs takes place, so that the heart becomes small; it loses its strength, the patient becomes weaker and weaker, the pulse rapid and feeble.

If *fever* occurs in the course of the disease, it is usually due to secondary accidents, as suppuration in a tumor, or perforation with septic peritonitis. The usual course of the temperature is normal until the later stages, when it is subnormal.

Examination of the Stomach-contents. Hydrochloric acid may or may not be absent, depending upon the amount of gastric catarrh. Lactic acid, on the other hand, is commonly present even in the earliest stages, and when associated with absent HCl is very diagnostic. Boas' test-breakfast must be given. For an accurate diagnosis repeated examinations must be made. Other general and local conditions, as fevers on the one hand, or dilatation on the other, are attended by absence of hydrochloric acid at times. In carcinoma it is the persistence of the absence which is diagnostic. Pepsin and the milk-curdling ferment are not changed.

The Urine. Indican in increased amount, acetone and diacetic acids may be present in the urine; otherwise there is no change.

Diagnosis. In the diagnosis of gastric cancer the following must be borne in mind: 1. The age of the patient. 2. The occurrence of causeless dyspepsia without relief. 3. Rapid loss of flesh and strength, with cachexia. 4. The occurrence of pain in the epigastrium, continuous, increased by food, but not relieved by vomiting, as in ulcer, and not distinctly localized. 5. Tumor—hard, circumscribed, followed by the physical signs of dilatation, if in the pylorus. 6. Vomiting is necessarily associated with the taking of food, in which fragments of cancer may be found; blood-cells are common; they may be detected on microscopical examination, or by the test for hæmin. 7. Examination of stomach-contents. (*a*) Except in dilatation the fasting stomach is empty; (*b*) hydrochloric acid is often absent, whereas lactic acid is present; (*c*) delayed absorption is present, indicated by motor tests. 8. Hemorrhage. In small amounts, usually of characteristic, coffee-ground appearance. 9. Metastases—above the left clavicle; in the liver; in the inguinal glands; rarely in the lungs and peritoneum. 10. Eichhorst speaks of persistent itching of the skin and insomnia as characteristic symptoms. 11. Finally, the comparatively short duration of the case. Rarely does it extend over a period of two years.

DIFFERENTIAL DIAGNOSIS OF GASTRIC CANCER, GASTRIC ULCER, AND CHRONIC GASTRITIS. (WELCH.)

GASTRIC CANCER.	GASTRIC ULCER.	CHRONIC CATARRHAL GASTRITIS.
1. Tumor is present in three-fourths of the cases.	Tumor rare.	No tumor.
2. Rare under forty years of age.	May occur at any age after childhood. Over one-half of the cases under forty years of age.	May occur at any age.
3. Average duration about one year, rarely over two years.	Duration indefinite; may be for several years.	Duration indefinite.
4. Gastric hemorrhage frequent, but rarely profuse; most common in the cachectic stage.	Gastric hemorrhage less frequent than in cancer, but oftener profuse; not uncommon when the general health is but little impaired.	Gastric hemorrhage rare.
5. Vomiting often has the peculiarities of that of dilatation of the stomach.	Vomiting rarely referable to dilatation of the stomach, and then only in a late stage of the disease.	Vomiting may or may not be present.
6. Free hydrochloric acid usually absent from the gastric contents in cancerous dilatation of the stomach; lactic acid much increased.	Free hydrochloric acid usually present in the gastric contents.	Free hydrochloric acid may be present or absent.
7. Cancerous fragments may be found in the washings from the stomach or in the vomit (rare).	Absent.	Absent.
8. Secondary cancers may be recognized in the liver, the peritoneum, the lymphatic glands, and, rarely, in other parts of the body.	Absent.	Absent.
9. Loss of flesh and strength and development of cachexia usually more marked and more rapid than in ulcer or in gastritis, and less explicable by the gastric symptoms.	Cachectic appearance usually less marked and of later occurrence than in cancer, and more manifestly dependent upon the gastric disorders.	When uncomplicated, usually no appearance of cachexia.
10. Epigastric pain is often more continuous, less dependent upon taking food, less relieved by vomiting, and less localized than in ulcer.	Pain is often paroxysmal, more influenced by taking food, oftener relieved by vomiting, and more sharply localized than in cancer.	The pain or distress induced by taking food is usually less severe than in cancer or ulcer. Fixed points of tenderness usually absent.
11. Causation not known.	Causation not known.	Often referable to some known cause, such as abuse of alcohol, gormandizing, and certain diseases, as phthisis, Bright's disease, cirrhosis of the liver, etc.
12. No improvement, or only temporary improvement, in the course of the disease.	Sometimes a history of one or more previous similar attacks. The course may be irregular and intermittent. Usually marked improvement by regulation of diet.	May be a history of previous similar attacks. More amenable to regulation of diet than is cancer.

Cases of cancer of the stomach may present only symptoms of anæmia. In this manner the disease has been confounded with *pernicious anæmia*. The blood is never reduced in cancer to the degree it is in pernicious anæmia, nor does it present the characteristics found in anæmia.

Ulcer of the Stomach. Simple round ulcer of the stomach may occur at any age, but is most common in young anæmic women. It

may be the result of an erosion of hemorrhagic infarcts by the gastric juice. Stockton believes it to be a neuropathic change.

The Symptoms. The symptoms are variable. The cases have been divided by Welch into four classes: (1) Those in which there are no symptoms whatever, the ulcer having been found after death from other diseases; (2) no symptoms until the sudden occurrence of hemorrhage, or perforation; (3) the symptoms of chronic gastritis or gastralgia only; (4) typical cases, with the characteristic symptoms, *pain, hemorrhage,* and *vomiting.* The symptoms of gastric ulcer may develop suddenly.

Pain. The pain is localized; it is usually confined to a small area in the epigastrium. It may be seated behind the cartilage of the sixth and seventh ribs, or may be complained of in the back, between the eighth and ninth dorsal vertebræ, extending as low down as the first and second lumbar. It is of a burning or gnawing character, is increased by food, and comes on in from two to ten minutes after the ingestion of food. It is relieved by vomiting, or after the act of digestion is completed; but a persistent, dull pain or a feeling of soreness remains. In addition to the ordinary pains, there may be attacks of gastralgia. The pain is increased by pressure. It may be modified by the position of the patient. It may be relieved by lying on the back when the ulcer is in the anterior wall; or relieved by lying on the abdomen when in the posterior wall.

Vomiting. Vomiting occurs shortly after the ingestion of food. It is not attended by retching. The vomited matter may contain blood.

The vomited matter and the contents of the stomach contain hydrochloric acid, which may be in excess. Eichhorst thinks it is always in excess.

Hemorrhage. Blood in the vomitus gives it a brown or reddish color. It may be detected by the usual methods. Hemorrhage may occur, however, independently of the act of vomiting. It varies in amount from half a pint to a quart. It may be so severe as to cause collapse. Sometimes, instead of being discharged as a profuse hemorrhage, the blood may gradually ooze from the ulcer and collect in the stomach before being vomited. It is then altered by the acid gastric juice. Sometimes the blood is not vomited, but passed by stool, which is then tarry. Tarry stools also follow the vomiting of blood. In the course of ulcer a hemorrhage may be so severe that death takes place before vomiting occurs. The stomach is then found to be filled with blood.

The stomach bougie should not be used; the nature of the contents must be determined by an examination of the vomited matter.

The General Symptoms. If the cases are of long standing, the face is anxious and the lines are sharpened. If there is much hemorrhage, anæmia ensues. There is not much wasting and no fever. Chronic dyspepsia and constipation may be present during the intervals in which the severe symptoms are in abeyance. The period of abeyance varies, and the symptoms may come on without cause, as in gastric crises, during which time the vomiting may persist for two or three days. I saw a young girl of twenty years with most severe gastric

hemorrhage and classical symptoms of ulcer. With careful treatment she improved. After marriage she remained well until pregnancy. During the first periods of this condition vomiting was extreme; it then subsided, whereupon, without warning, a gastric crisis took place. The vomiting of blood continued for many days, and the symptoms of gastric ulcer remained for a month.

One of the characteristic features of the disease is the recurrence of symptoms after a long period of abeyance. A patient under my care during the last ten years has had three undoubted attacks. It is possible that during each period ulcers healed, to be followed after a time by the occurrence of new ulcers.

Diagnosis. The diagnostic features are: 1. The age. 2. The long duration. 3. The occurrence of emaciation up to a certain point only; most of the patients are under-weight and have a gaunt look, particularly males. 4. The characteristic pain. 5. The vomiting. 6. The hemorrhage. 7. The periods of relief from symptoms. 8. The absence of marked nervous symptoms which attend gastric neuroses. 9. The absence of dilatation of the stomach. 10. The hyperacidity of the gastric juice.

The Accidents of Ulcer of the Stomach. 1. The occurrence of perforation. Sudden severe pain, with collapse. The pain is usually in the epigastrium, but may be in the back as high as the seventh or eighth dorsal vertebra.

2. Hemorrhage, which may cause death immediately, with either vomiting of blood or retention in the stomach.

3. With healing of the ulcer, stenosis at the pyloric orifice may take place, with subsequent dilatation of the stomach.

Diseases of the Intestines.

The intestine is a canal of varying dimensions, the physiological office of which is to propel material received from the stomach, and to permit of the digestion and absorption of that which is to serve for the nutrition of the body. The canal is richly supplied with bloodvessels and lymphatics. It is made up of mucous membrane, muscle, and peritoneum. For the purpose of digestion, fluids are secreted, either from the intestinal glands or large neighboring glands which discharge into the canal.

Diseases which affect the canal impair or cause an abeyance of the physiological offices. As these offices—absorption and digestion—are essential to nutrition, it is not surprising that the body-weight and strength are impaired. We know too little about the function of digestion to utilize such knowledge in diagnosis. Intestinal digestion is also dependent upon the healthy performance of the functions of the liver and pancreas. It is difficult to draw fine lines of distinction even in health, and intestinal pathology is closely interwoven with hepatic and pancreatic pathology.

Alterations of the function of the intestine as a canal give rise to distinctive symptoms. Either its movements are too frequent and rapid, causing *diarrhœa*, or too sluggish, causing *constipation*. Obstruction of

the canal leads to symptoms common to such a condition (see Morbid Process), modified by the physiological duties and the anatomical structure of the canal.

The morbid processes are hyperæmias, inflammations, degenerations, and new growths. The symptoms that attend these processes are not different from the symptoms that attend such processes in similar structures elsewhere. It must not be forgotten that the function of the canal is influenced by each process. On account of the process we may have *pain* and *fever;* on account of the impaired function, *pain, flatulency, diarrhœa,* or *constipation, change in the character of the stools,* and *impaired nutrition.* Some of the above morbid processes may lead to the mechanical condition, *obstruction.*

The morbid alterations of the intestinal tract are ascertained by data obtained by *inquiry* and by *observation.* The data obtained by inquiry include the subjective symptoms—pain, and discomfort from flatulency. By observation the general condition of the patient, the presence of tenderness, alterations in the size and shape of the abdomen, and other physical phenomena are observed. The feces are carefully studied, with the object of determining modifications of the function of the bowel, the presence of ingredients due to some morbid process, as serum, blood, pus, or mucus, or of extraneous matter, as worms or foreign substances. The feces are studied by the naked eye, by the microscope, and by bacteriological methods.

One symptom may be the chief manifestation of a disease, as pain of lead-colic, diarrhœa of several morbid disorders, constipation of others. In the discussion of the special symptoms a consideration of the diseases of which the symptom is the main expression will be taken up.

Parasites. The intestine is the recipient of material for nutrition. Parasitic forms of animal life, or their ova or spores, may enter the intestine with the food. They either remain in the intestinal tract or wander into other structures. They include animal and vegetable parasites, such as forms of protozoa, vermes, and fungi. While the canal is open to infection by various micro-organisms, it is the natural habitat of others, which may become deleterious agencies when the conditions of their environment are changed. Thus the bacillus coli communis is, in man, with normal epithelial structure and normal secretions, an innocuous parasite which, when inflammation sets in, may become nocuous.

The symptoms produced by the *protozoa* and *fungi,* or by their products, the ptomaïnes, are of an infectious or toxic nature. Inflammation is produced locally.

The symptoms of *worms,* if retained in the intestinal canal, are: (1) Reflex in nature; (2) symptoms due to catarrhal inflammation; (3) symptoms due to action of the parasite on the blood—anæmia; (4) symptoms due to wandering of the parasite, as in trichinosis. (See Feces.)

Symptoms of the Tæniæ and Bothriocephali. There may be no symptoms save discharge of the parasite or portions of it by the rectum. In others the symptoms of intestinal dyspepsia or intestinal catarrh

are observed. Headache, giddiness, lassitude, and itching at the nose and at the anus are said to be present. The patient becomes hypochondriacal. Convulsive disorders occur. Hysteria, forms of epilepsy, grinding of the teeth at night, and restlessness attend the habitation of the parasite in the intestine. In all convulsive disorders the possibility of worms as a cause must be remembered.

Symptoms of Ascarides. (1) Gastro-intestinal catarrh; (2) symptoms of obstruction (rare); (3) symptoms due to wandering—as to the hepatic duct to the stomach, or to the vagina; (4) nervous symptoms of reflex origin; (5) the worm or its ova in the feces.

Symptoms of Oxyuris Vermicularis. (1) Gastro-intestinal dyspepsia or catarrh; (2) itching or heat at the anus, worse in bed; (3) vesical and rectal tenesmus; (4) erythema about the anus; (5) priapism; (6) vulvitis and vaginitis; (7) the worms in the feces.

The Strongylus. The symptoms are local, with the symptoms of profound anæmia. The discovery of the ova in the feces distinguishes this form of anæmia from other varieties.

The symptoms due to the presence of the trichina spiralis and filaria will be discussed in appropriate sections. (See Blood and Infectious Diseases.)

The Intestines in other Diseases. The relationship of intestinal disorders to affections of other viscera will be discussed with each symptom. It must not be forgotten that derangement of this tract may have its origin in local causes or in causes remote from the intestinal tract, or in some general condition of the individual. Thus diarrhœa may be due to inflammation which is primarily local, or which may be secondary to infection. Nothing is more common than to see diarrhœa in a general infection, such as septicæmia. In exophthalmic goitre the diarrhœa is not due to a local cause, but to some as yet unknown nerve disorder. Constipation may be due to central brain disease, to a general condition like diabetes, or be of local origin.

It must be remembered that the diagnosis of an intestinal lesion is never complete without determining its causes. Thus enteritis and ulceration occur in typhoid fever, in cholera, and in other infectious disorders, all of which are to be passed in review in making up a diagnosis. Diarrhœa is a symptom in Bright's disease, and the causal relationship must always be borne in mind.

Differential Diagnosis. Intestinal disease or disorders are not usually confounded with disease of other structures. It is worthy of remark, as a fact which is sometimes overlooked, that symptoms of intestinal obstruction are frequently due to peritonitis. Tumors of the intestine must be distinguished from tumors of the peritoneum, the stomach, pancreas, and liver, and the uterus and ovaries. The history, the seat and physical character of the tumor, and the associate symptoms point to the true condition.

Arteries of the Intestine. The intestines are supplied by the mesenteric arteries. Its branches may become the seat of *emboli.* The symptoms are sudden pain, intestinal hemorrhage, and discharge of a portion of intestine. The patients are the subjects of atheroma or heart disease.

The Subjective Symptoms.

The Data Obtained by Inquiry. Pain. *Colic.* Colic is the term applied to *paroxysmal pain* in the abdomen. It is characterized by suddenness of onset and by alteration of intestinal function. It attends all forms of inflammation of the intestinal tract. It is applied to a peculiar affection known as lead-colic, due to local effects of lead. The term *colic* is also applied to painful affections of the hepatic ducts, pancreatic ducts, the ureters, and the uterus. Intestinal colic is the form at present referred to. In addition to the inflammation of the intestinal tract, it may be due to *indigestion* with flatulency. When it occurs suddenly without local cause it is known as *enteralgia.*

Intestinal Colic. The colic of intestinal indigestion occurs suddenly, or it may be preceded by signs of intestinal indigestion. The *pain* is chiefly in the umbilical region and radiates from that point. It is relieved by moderate pressure or warmth. The patient is restless and irritable. The face is anxious. The pain causes him to roll about and double up. There is a cold sweat, and the pulse is small and hard. Prostration or *collapse* rapidly ensues. *Nausea* and vomiting follow the pain, and there are gaseous eructations. *Distention.* The abdomen is distended and tympanitic on percussion. The pain may be relieved by the passing of flatus. *Cramps.* Spasm of the muscles of the calves is common. The *cramps* are very painful; the muscles become knotted. The hands and feet are also cramped. The pain is said to be due to spasm of the intestine, and is known also as spasmodic colic. It is certainly due to distention or to irritation.

If the intestinal colic is due to *indigestible food,* it may have been preceded by an attack of acute indigestion, and the griping pains may have developed at long intervals, with gastric and intestinal flatulency. Vomiting may precede or attend the attack, and diarrhœa follow. If the colic is due to *gas* alone, there is great tympanites. If it is due to *feces,* it has been preceded by a history of constipation, and there may be fecal masses detected in the rectum or along the colon.

Fever. The presence of fever is against intestinal colic, and points to inflammation in some portion of the abdomen; moreover, in inflammation the pain is constant, but localized and aggravated by pressure. The skin is hot and dry.

Diagnosis. The sudden severe pain, often relieved on the discharge of gas, with gastro-intestinal disorder, tympanites, the occurrence of cramps in the extremities, and the localization of pain to the umbilicus, all point to the true nature of the affection. A history of indiscretion in diet, or exposure, aids in the diagnosis. In colic the pain may come on suddenly, or increase gradually from a sense of discomfort or soreness. The pain at its height is described as agonizing, and of a boring or shooting character, abating for a time and then increasing, until the patient rolls and twists in agony and breaks out into a cold sweat. The pain may shoot from the seat of greatest intensity to the shoulders, back, chest, or iliac region.

It must be distinguished from *enteralgia.* The latter comes on slowly and lasts for hours or days. The pain is situated around the

umbilicus, and is relieved by deep pressure, although the skin may be hyperæsthetic. Sometimes the abdomen is retracted; there are no signs of indigestion, and flatulency and borborygmi are absent.

Lead Colic. If the enteralgia is due to lead, there is a history of exposure to that metal. The blue-line on the gums, with obstinate constipation but no vomiting, and the occurrence of neuritis due to saturnine-poisoning, point to the true nature of the case.

Hepatic Colic. In hepatic colic the pain is situated in the region of the liver, and may radiate to the shoulder or back. It is sometimes fixed in the right parasternal line about the cartilages of the sixth and seventh ribs. The attack is attended by vomiting, usually of bilious fluid. It occurs in women most frequently; the patients are almost always over forty years of age. It may be followed by jaundice. There is local tenderness, and there may be some swelling in the region previously mentioned. The bowels are constipated, and after the attack may contain gallstones.

Renal Colic. In renal colic pain begins in the kidney and then extends along the ureter. It is always more localized to the right or left of the median line in the abdomen. It is more frequently in the lower portion of either of the upper quadrants, three inches to either side of the median line, depending upon the kidney affected. From this region the point of maximum intensity and of local tenderness moves to the lower quadrant toward the median line in the oblique direction, rarely getting an inch below the transverse umbilical line. The pain then extends to the region above the pubes and down the thighs. From the first there is increased frequency of micturition. The urine is scanty, high-colored, and may contain blood. With the free micturition relief follows.

Local Peritonitis. Pain over the liver, spleen, and kidneys is generally due to involvement of the peritoneal coverings of these organs, and partakes of the character of local peritonitis. It may, however, be due to malignant, ulcerative, or inflammatory disease, and the diagnosis must be made by noting the character of the pain, its intensity, duration, seat, and the other general and local symptoms with which it is associated.

Rectal Pain. Pain in defecation may be due to piles, internal or external, or to fissure, or may be the result simply of the passage of an unusually large, hard mass. Pain from fissure is most acute and spasmodic, and persists for some time after defecation. Fibroid stricture of the rectum causes more pressure and straining at stool than real pain; but cancer is apt to be extremely painful.

Uterine Colic. In uterine colic the pain is situated in the pelvis. There is some abnormality of discharge, and a history of uterine disease. Care must be taken not to confound the sudden pain of extra-uterine pregnancy with intestinal colic or other forms of abdominal pain. In *extra-uterine pregnancy* the pain is in the lower quadrants of the abdomen to the right or left of the median line. It is sudden and intense, attended by more or less collapse. It may be attended by all the symptoms of internal hemorrhage. It may cause vomiting. The history of cessation of menses, or other signs of pregnancy, of

discharge of decidua, with the local signs on physical examination, indicate the true nature of the pain.

Pancreatic Pain. In *disease of the pancreas*, either from the passage of calculi (extremely rare) or because of pancreatic hemorrhage, there may be sudden severe pain. The pain is localized to the region below the sternum. It may be severe in the back and extend up the thorax. It occurs in paroxysms, and is attended by great anxiety and collapse.

Gastric Pain. Intestinal colic must be differentiated from pain of gastric ulcer, gastric cancer, and gastralgia. The characteristics of pain in these affections have been discussed. When *perforation* occurs in gastric ulcer the pain is usually seated in the epigastrium, but may be complained of in the back as high as the mid-scapular region. It is sudden and severe, preceded by a history of ulcer and attended by collapse. There are no evidences of indigestion. Perforation of the biliary passages is attended by pain in the hepatic region. The pain is sudden and is usually preceded by symptoms due to derangement of the biliary passages from obstruction by gallstones.

Appendicitis. Intestinal colic must not be confounded, although it frequently has been, with the pains that attend appendicitis. This is particularly the case with relapsing appendicitis. In this form only mild fever attends the attack. The patient is seized with severe pain, which may be described as occurring in the lower right quadrant, but is sometimes complained of about the umbilicus. It frequently follows indiscretion in diet, and may be attended by vomiting, and is likewise usually relieved by eructation, but not by the passage of gas, a point of great importance in the diagnosis. The attack occurs mostly in young subjects, and lasts from twelve to twenty-four hours. It may be so severe as to cause collapse. If fever attends it, and there is a mass present, the diagnosis is much easier. In the relapsing as well as the true form there is tenderness at McBurney's point. (See Appendicitis.)

Peritonitis. Intestinal colic must not be confounded with peritonitis, which may follow in any of the above conditions, or develops at other points in the abdomen. The purulent peritonitis that succeeds pyosalpinx may be attended by severe pain without much reaction. The pain, however, although complained of about the umbilicus, can be localized by pressure in the lower quadrant and in the pelvis. It may disappear after eight or ten hours, to be followed by a recurrence. The recurrence of pain is usually attended by *fever*. In the first twenty-four hours the bowels are loose, or at least readily moved. If the peritonitis continues beyond this period, it is often impossible to move the bowels.

Intestinal Obstruction. Intestinal colic must not be confounded with organic disease of the bowels with resulting obstruction. In these affections there are sudden constipation and rapid prostration. The vomiting, if present, persists and soon becomes stercoraceous. In *intussusception* the stools are characteristic. Strangulation, or ileus, is associated with a history of previous peritonitis or the presence of hernia. In the latter there may be signs at the hernial points. In the obstruction from external pressure the presence of tumors has been known

previously or can be recognized. In fecal obstruction, or the obstruction by gallstones, the local signs may be pronounced, and the pain is usually in the ileo-cæcal region. The affection is acute. Pain that extends over a long period of time, that is not due to an acute process, or attended by sevɜre acute symptoms, has been considered elsewhere. (See Abdomen.)

Rheumatism and *Neuralgia.* Intestinal colic may be mistaken for *rheumatism* of the abdominal walls. In the latter there may be a history of exposure. The muscles are extremely tender. There are no gastro-intestinal symptoms, the urine is loaded with uric acid and urates, and there may be muscular pain in other situations, or a pronounced history of previous attacks of rheumatism. In lumbo-abdominal neuralgia the pain may simulate intestinal colic. Pressure-points, where the respective nerves have their exit through the fascia, are detected.

Pain in Vertebral Disease. Just here may be considered the pain about the navel, which occurs in paroxysms, due to disease of the vertebræ. There may be caries from tuberculous disease or from pressure of an aneurism or malignant growths. Examination of the vertebræ may determine its nature.

Diarrhœa. Diarrhœa is a symptom of disorder of the intestine, which in turn is itself the cause of symptoms, just as jaundice, a symptom of hepatic disorder, is the cause of various symptoms. In diarrhœa there is increased frequency of the movements of the bowels. This is due to increased peristalsis of the intestine, which occurs from a number of causes. Not all increased peristalsis results in diarrhœa. (*A*) *Nervous diarrhœa.* Increased peristalsis may be due to some impression upon the nervous mechanism of the intestine. This may explain the diarrhœa of emotion, or that which occurs from other psychical influences. (*B*) *Catarrhal diarrhœa.* In the larger number of cases the diarrhœa is due to catarrhal inflammation of the intestinal tract. The causes of the catarrhal inflammation are many, and have been divided into primary and secondary causes. *Primary* catarrh is due to the direct influence of causal factors upon the mucous membrane. (1) It is seen after cold or exposure; (2) it occurs from the direct irritation of undigested food, and (3) from the action of irritants, as of bacteria or the products of bacteria. Catarrhal inflammation due to micro-organisms is the most frequent form that occurs in children.

Secondary catarrhs follow other lesions of more pronounced character, as ulcers. The catarrh, and hence the diarrhœa, that attends the ulceration of typhoid fever, the ulceration of dysentery, or that occurs in Bright's disease, and the diarrhœa that attends carcinoma or other organic disease of the bowel, is of this nature. In addition, a catarrh of the bowels arises from venous stasis in the mucous membrane, with chronic congestion. This occurs in organic heart disease with congestion of the liver.

Diarrhœa is a symptom of the action of certain poisons, such as mercury, arsenic, and other corrosive agents. The diarrhœa which

occurs from the irritant action of food-products and in cholera infantum is due to a toxic ptomaïne.

Diarrhœa sometimes fulfils a vicarious office. This is the case with the diarrhœa which comes on in cases of chronic Bright's disease, and in acute Bright's disease before the supervention of uræmia. When diarrhœa occurs in a person with pallor, dimness of vision, and œdema the urine should always be examined.

The Symptoms of Diarrhœa. *The Motions. Increased movements* of the bowels. The frequency of the movements varies with the cause. In the diarrhœa of nervous origin, usually after five or six movements have occurred, the patient is relieved, because by this time the cause for the nervousness has disappeared. In catarrhal diarrhœa the number varies from half a dozen in twenty-four hours to the same number in an hour. Indeed, in some severe cases the evacuations may be almost constant.

Character of the movements. The movements may be (1) *fecal*, with a small amount of *water*. They are light in color, softer than natural, but yet retain their form. They are the kind of movements seen in simple catarrh.

2. The fecal matter is mixed with *undigested food*. The feces are in scybalous masses, and the watery element is increased. They are the stools of the so-called dyspeptic diarrhœa.

3. Along with the feces more or less *mucus* is seen. The amount of mucus depends upon the seat as well as the intensity of the inflammation. Inflammations of the large intestine are attended with mucous discharge. It may be mixed with and stained by feces so that it can be recognized only by close inspection. In milder degrees of catarrh it is seen on the surface of the fecal masses.

4. The feces disappear almost entirely, and instead the evacuations are *watery*. The watery evacuations may be discolored, as in the pea-soup evacuations of typhoid fever, or they may be almost clear water, as in the rice-water discharges of cholera.

5. The evacuations may contain *blood*. Bloody discharge usually accompanies the discharge of mucus; when the catarrh is in the lower bowel blood may occur independently of the mucus. If with the mucus, it tinges it in reddish specks, or small amounts of free blood are seen. The blood may be bright in color, and then usually comes from the rectum. It must be remembered that the blood may be from hemorrhoids, or fissure, which is unduly irritated by the diarrhœa. It is then bright red and unmixed with the movement, and from its position can readily be seen to have followed it. On the other hand, it may be due to cirrhosis of the liver, with venous congestion. It may be due to the ulceration of typhoid fever, and the intense inflammation of enteritis. It is a symptom of carcinoma of the bowel, and is of frequent occurrence, almost pathognomonic, in intussusception. It must be remembered that blood of this character is discharged from the bowel independently of diseases of that tube, as in purpura, scurvy, and other blood diseases. (See Arteries of the Intestines, page 815.) If mixed with the movement, the blood may be black, as in all forms of *melæna*, or it may be dark red in color. The black blood usually

comes from the stomach or the first part of the duodenum, and may be the result of ulceration, or even from the swallowing of blood.

Microscopical and Bacteriological Examination. (See Feces.) In simple catarrhal inflammation of the tubules, on microscopical examination, but little is found except an excess of epithelium from the mucous lining. In more intense inflammations, in addition to epithelium, we find pus and blood and mucus. Micro-organisms are found, the kind depending upon the cause of the diarrhœa. In health, Booker has found at least forty varieties of micro-organisms, many of which, in all probability, are not pathogenic. In health, the bacillus coli communis and the bacterium lactis aëriformis are found. In the diarrhœa of children both forms are present in excessive numbers, because conditions favoring their growth arise, and in all probability are the cause of the irritation of the bowel. In that form of inflammation of the bowel known as dysentery, in addition to the bacteria that attend inflammation, the amœba coli is often present. It has been found that dysentery may be due to a number of causes, but that the so-called tropical dysentery is due to the protozoa first described by Kartulis, in Egypt, and in this country by Osler. (See Feces.)

Pain. The symptoms that attend increased movement of the bowels depend upon the cause and also have direct relationship to the frequency of the evacuation. The most frequent symptoms are *pain, flatulent distention,* with *borborygmi* and *tenesmus.* The pain depends largely upon the cause. If the irritant is a product of indigestion, or a bulky mass, pain is more or less severe. It is situated in the centre of the abdomen, and may extend all over it. Pain occurs before the evacuation ; it is sharp, lancinating, and is usually relieved by the movement. If the inflammation is in the large intestine, the pain may be complained of in the course of the large bowel or be more intense over the cæcum and the sigmoid flexure. The rectum may be the seat of pain or of painful sensations. This has been described as a feeling of a hot ball in the lower pelvis.

Flatulent Distention. The flatulent distention is not very great generally. The abdomen is distended, tympanitic on percussion, and tender on palpation, both of which may be more marked in the middle of the abdomen if enteritis alone is present, or it may extend along the course of the colon, as in the so-called entero-colitis of children. With the distention there are borborygmi. The rumbling usually subsides after the evacuation.

Tenesmus occurs in all forms of diarrhœa if the evacuations have been frequent. After the discharge of the contents of the bowel, particularly if from the rectum, the tenesmus is much more severe, and may be of constant occurrence. In the severe cases the tenesmus may be almost continual. On account of it prolapse of the bowel is apt to ensue.

General Symptoms. The general symptoms that attend diarrhœa depend upon the cause. In simple diarrhœa there might be slight feverishness only, with a little weakness. In diarrhœa, with excessive movements, with mucus, with or without blood, the fever is marked and may rise as high as 103°. The fever that attends dysentery is high, and usually rises rapidly at the beginning.

Prostration. More or less prostration attends all cases. It is, however, more marked when there are frequent watery evacuations. In its most pronounced degree it is seen in cholera and cholera infantum. *Collapse* rapidly ensues under these circumstances, on account of the depleting effects of the excessive watery discharge. In catarrh of the intestines secondary to typhoid fever and other conditions the general symptoms depend upon the primary disease.

Chronic Diarrhœa. Chronic diarrhœa may be due to chronic inflammation of the bowels, as in chronic intestinal catarrh. It may be secondary to the ulceration of dysentery, tuberculosis, syphilis, or cancer. It is the common diarrhœa of amyloid disease. In chronic diarrhœa the number of the stools varies, but seldom amounts to more than ten to fifteen in a day. In chronic intestinal catarrh three or four movements occur in the twenty-four hours. They usually occur in the morning, the first evacuation taking place immediately on rising and the remainder during the morning hours. They are more common in women than in men, and are readily excited by exhaustion or nervous influence, as grief, emotion, or excitement of any kind. The stools are fecal and watery, and contain some mucus. The mucus usually coats the surface of the feces. The color of the feces is not changed. The patients usually suffer fom intestinal dyspepsia, or they are subject to some gastric neurosis. They are not under weight, and except for the inconvenience of the morning hours, could attend to the ordinary demands of life. They are more nervous than most people, and are liable to attacks of hemicrania.

Membranous Diarrhœa. In a number of cases the discharge from the bowels resembles membrane. The disease is also called membranous enteritis. The discharge contains much mucus, and may be quite watery. After the feces have been passed membrane is discharged. This may be in shreds or large masses, and may also be like a cast of the bowel. The patients are usually women who are hysterical and have some menstrual disorder. Pain may precede the discharge, and continue until there is complete relief.

Constipation. Constipation may be due to a number of causes. It may be due to alteration or diminution in the secretions of the intestinal tract, as is seen in all fevers, except when they are attended by specific intestinal catarrh, as in typhoid fever. Such diminution of secretion occurs in the summer, when there is more free perspiration than in other seasons, and is present in affections attended by excess of perspiration, or exhaustive diuresis. Constipation, therefore, is a common symptom of diabetes.

In addition to alteration of the secretion, diminution in the sensibility of the nerves may exist. This is the one chief cause of habitual constipation that is so prevalent. On account of carelessness the patient loses the habit of having a regular movement of the bowel each day, and in consequence the usual stimulus is removed. Constipation also occurs from weakness of the muscles.

The three conditions—diminution or alterations in the secretions, debility of the muscles, and impairment of the sensibility of the ner-

vous mechanism—are combined influences, on account of which constipation is so prevalent in persons of sedentary habits and in persons living upon improper diet. General diseases and local disorders which influence either of the above elements cause constipation. Thus in anæmia and chlorosis, in neurasthenia and hysteria, constipation is a common condition. Its occurrence in fevers has been mentioned. In the convalescence from exhausting disease and prolonged confinement to bed constipation is apt to ensue.

Local Causes. Atony of the abdominal muscles or of the bowel is the cause. Atony is most strikingly seen in peritonitis and typhlitis, in both of which a paretic state of the bowels develops. It is seen in the aged and in cachexia along with atony of other muscles. Obstruction of the bowels, acute or chronic, usually causes constipation (*q. v.*). If the obstruction is not complete, there may be diarrhœa on account of catarrhal inflammation. Constipation often occurs on account of pain, particularly pain seated in the rectum. The pain is such that the patient shrinks from an evacuation. Frequent postponement soon causes constipation. The pain may be due to fissures, to hemorrhoids, or to fistula. Constipation occurs also from local diseases in other portions of the body, influencing, in all probability, the nervous mechanism by which peristaltic action is excited. In acute and chronic disease of the brain and cord, as meningitis and myelitis, constipation is a usual attendant. It also occurs in tetanus. If the bowel is deprived of fecal matter, evacuations cease; constipation is, therefore, a common sign of stricture of the pylorus and of stricture or cancer of the œsophagus.

Symptoms of Constipation. Constipation is characterized by diminution in the frequency of the bowel-movements. The frequency of the movements varies in health. Some persons are comfortable with an evacuation taking place once a week, or at most every third or fourth day. There are cases on record in which the evacuations took place but once a month. Cases of this class are usually due to muscular paralysis of the bowels, with secondary dilatation. The accumulation of feces is removed by a sharp attack of diarrhœa, attended by much pain. The diarrhœa sometimes continues for twenty-four hours. When it sets in fever may be present until there is thorough evacuation.

Local Symptoms. Usually the symptoms that attend constipation are local, being due to the discomfort of the accumulation of feces. The local symptoms may be limited to the rectum or extend throughout the abdomen. In the rectum there is a sensation as of the presence of a mass, which may cause some pain. The abdomen is distended; there is considerable rumbling, and sometimes peristaltic waves are seen. The accumulation of the fecal mass in the bowels may set up tormina and tenesmus, and portions of the mass may be discharged from time to time. In other words, a diarrhœa may occur, the diarrhœa of constipation, or spurious diarrhœa. The stools are small, composed of hard scybalous masses, generally coated with mucus, and streaked with blood. The evacuation does not give relief, and the desire for a movement may be more or less continuous.

On examination in constipation with fecal accumulations the outline

of the colon may be marked out by palpation and percussion of the distended abdomen. In its course masses are felt varying in size from a marble to a base-ball, and in consistence they may be soft to the palpating finger; they are never indurated like a calcareous mass, as gallstones or a mass due to malignant disease. (See Fecal Tumor.)

General Symptoms. While in many instances the general symptoms are of no consequence, in others the patients are nervous and may be in more or less impaired health, on account of the secondary effects upon the stomach. Digestion is impaired and the form of indigestion is that which attends neurasthenia.

The patients are of spare habit, usually of dark or muddy complexion. They may be depressed. There is inaptitude for mental exertion; they are more or less hypochondriacal. The tongue is constantly furred, the appetite variable; there are weight and fulness after eating, and generally some flatulency.

The Secondary Effects of Constipation. The effects of constipation upon the intestines are various and sometimes disastrous. They are *dilatation* and *ulceration*. The former may become enormous, as in cases reported by Formad and Osler. The dilatation may be so great as to distend the entire abdomen. The ulceration may be localized to the rectum, or cæcum, or extend throughout the entire large intestine. On palpation the course of the colon is tender, and fecal masses may be outlined that are painful, because of their pressure upon the adjacent ulcer. In the rectum the ulcer may be deep, and be followed by peri-rectal abscess.

Stercoral typhlitis. In the cæcum the accumulation may cause a large boggy swelling, extending in the course of the cæcum, which is tender on pressure and dull on percussion.

Fecal impaction, with secondary ulceration, is of frequent occurrence in typhoid fever. This must be borne in mind, for often serious general and local symptoms arise because it is overlooked. Recently I saw a case with diarrhœa of constipation, with some fever, which persisted for weeks after the usual course of typhoid fever. It was thought the patient had tuberculosis, or that the typhoid process was abnormally prolonged. Examination disclosed ulceration into the vagina, and the feces were constantly discharged from this orifice. It had been thought that the discharges of feces were due to diarrhœa. Of course, fever attended the process, and rendered the case all the more obscure.

In this connection must be mentioned the constipation that occurs on account of lead-poisoning, and the exhibition of drugs, as opium, or astringents. The constipation of lead-poisoning is usually attended by colic, and the blue-line on the gums is seen, while wrist-drop or other manifestations of lead may be present.

Intestinal Hemorrhage. The causes are general and local. The general causes are those that accompany hemorrhage in other localities. (See Gastric Hemorrhage.) The local causes, when the hemorrhage is small, are: inflammation of the bowel; traumatic injury to the bowel from hernia, feces, and parasites, and foreign bodies swallowed, or from corrosive poison; tumors of the bowel, as in cancer, invagination, and

ulcers. When the hemorrhage is large the causes are the congestion attending portal obstruction and liver disease, or disease of the heart with secondary obstruction; aneurism of the superior mesenteric artery, or aneurism rupturing into the intestine, and, occasionally, embolism of the artery; the ulceration from typhoid fever, from dysentery, and from syphilis. It may occur in pyæmia and septicæmia, or the acute exanthematous diseases.

The *symptoms* may be those of hemorrhage alone: collapse, pallor, failure of sight, tinnitus, vertigo, small pulse, and general restlessness. The hemorrhage must be copious under these circumstances, and is due (1) to an ulcer, as in typhoid fever; (2) to portal obstruction; (3) to an aneurism; (4) to purpura or hæmophilia.

A second group of symptoms is connected with the appearance of the discharges from the bowels. The *stools* are bloody; if the hemorrhage is low down, they are bright red and usually mixed with feces. If high up, they are tarry. The latter condition is known as melæna. (See Feces.) The passage of the stools is preceded by colicky *pains*, or there may be some rumbling. The diagnosis must be directed toward determining the cause of the hemorrhage, as well as its seat; the history, the associate diseases, or symptoms, aid in determining the cause. Examination of the rectum may afford a clue to its origin.

The Objective Symptoms.

The Data Obtained by Observation. Physical Signs. (See The Abdomen.) *Inspection.* Local and general enlargements of the abdomen have been discussed in the preceding pages. Movements of the intestines are seen in obstruction due to increased peristalsis. The intestine above the point of obstruction may swell into a well-defined tumor which becomes hard and dull, and tympanitic on percussion.

Palpation. Tenderness, peristalsis, peritoneal friction, the bubbling of gas through a constriction of the bowel, and tumors, are recognized by palpation. It is necessary often to place the patient on all-fours or in a knee-chest position.

Percussion. The normal note is tympanitic. Local areas of dulness may be due to intestinal tumor. Light percussion should be employed. A dull tympany indicates a solid mass surrounded by the distended intestines. The outline of the large intestine can be ascertained by filling it with water.

The Feces. General Considerations and Macroscopical Appearances. The number of stools in health varies chiefly with the individual and the character of the food taken. After infancy, one passage in twenty-four hours is the rule, but it is natural for some persons to have two or three, and for others to have but one passage in two, three, or four days. Such a condition is termed constipation, while pathological constipation is properly called *obstipation*. The opposite condition is known as diarrhœa. The amount and character of food and drink ingested influence the number of stools. Exercise also plays a rôle; increased or diminished peristalsis, from whatever cause, will induce diarrhœa or constipation, respectively. In disease

the greatest extremes are met with—from the non-passage of feces for days, as in obstruction, to an almost continuous discharge, as in some forms of intestinal inflammation. It is well to remember that diarrhœa may be the symptom of obstipation, as when impacted feces in typhoid causes looseness of the bowels.

The *amount* of feces varies with the quantity and nature of food. If most of the food is digested there will be but little left to form feces. In any disease that prevents the absorption of digested food or causes an increase in the fluid contents of the intestine, as cholera, the amount of feces will be increased. In health about 140 to 200 grammes are voided in twenty-four hours.

The *form* and *consistence* of healthy stools vary somewhat. They are commonly cylindrical and firm or mushy. When they remain long in the intestinal canal, and the water is extracted, they become hard and may form balls, or flattened masses known as scybala. These are frequently seen in convalescing typhoid patients. On the other hand, the feces may be without form, and are then liquid, either watery, as in cholera, or purulent or bloody. Many diseases cause such a condition.

The *odor* of feces is sometimes more or less characteristic of certain conditions. Thus the stools of nursing infants have a sour smell, while in infantile diarrhœa, and when fermentation takes place, they have an odor of sebacic acid. When urine is mixed with the passage the odor will be ammoniacal; with blood present it often has a stale odor.

The *reaction* is not constant. Thus in intestinal catarrh, with acid fermentation, it will be acid, or in alkaline fermentation it will be alkaline. The *color* of the stool varies too much to be of special diagnostic value. In health it is light to dark brown, due chiefly to the presence of hydro-bilirubin, a product of decomposition of bile-pigment, which is never normally found unaltered in the feces. It is influenced greatly by food and medicines. When certain berries, as huckleberries, are eaten, or certain medicines taken—iron and bismuth—they make the passages black. Calomel causes green stools, on account of the biliverdin discharged. Green stools may also receive their color from the presence of a bacillus which produces a green dye. Santonin, rhubarb, and senna cause yellow, and hæmatoxylon red stools. The last fact is important, as parents or nurses should always be warned to expect red passages when hæmatoxylon is given.

The feces may be red or reddish from the presence of unaltered blood; or black, when the blood has undergone changes; the so-called "tarry stools" are of this character. With a decrease in the amount of bile the stools become less colored, and if the bile is cut off they become clayey. This color may, in some cases, be due to the presence of fat left undigested because of the lack of bile. On the other hand, if from disorders of the stomach and intestine the contents pass through too rapidly, the feces may contain unaltered bile, unchanged bile-pigment, giving a green or yellow color, and showing the bile-reaction.

The *constituents of feces* that can be recognized by the naked eye are numerous. Seeds, stones, skins of fruit and berries, and the fibres of

vegetables are often seen in healthy stools. In the passages of children and weak-minded individuals foreign substances of all descriptions may be present. Foreign bodies and partially digested portions of food may be mistaken for parasites. Portions of tumors from the digestive tract may appear in the feces.

In certain diseases of the stomach and small intestine, and in those who eat very fast and do not properly masticate their food, undigested and unchanged particles of food may be seen in the stools.

Shreds of mucous membrane of varying size are passed with the feces. Von Jaksch saw such a shred 5 cm. long and 3 cm. broad in a case of cholelithiasis. Various sized pieces of membrane, consisting of transformed mucus, are passed in membranous enteritis.

Particles resembling sago-grains, perhaps the result of over-indulgence in farinaceous food, have been met with.

Gallstones in the feces have great clinical value. They may escape detection if not properly sought for. When suspected, each passage should be passed through a linen sieve, the fecal masses being softened with water. They may be found as small, crumbling masses, composed chiefly of cholesterin (intrahepatic calculi), or as hard, irregular, smoothly worn, shining, many-sided, hard stones, sometimes as large as an egg, usually the size of a pea. Enteroliths are occasionally seen. They are said to originate in the appendix.

Blood may be present in the feces in varying proportions and conditions. When found unaltered on the surface of scybalous masses, it is from the rectum or large intestine, and probably the result of traumatism. Hemorrhoids, if bleeding, may cause such an appearance, or may cause very free hemorrhage. Severe hemorrhage may come from ulceration of the rectum or colon, due to malignant disease or severe inflammation. The blood may be intimately mixed with the feces, and have its origin in the large intestine, but much more commonly it indicates a source in the stomach or small intestine. Under such circumstances it is nearly always more or less changed by the intestinal juices, and is brownish-red or black (the tarry stool mentioned above), or has the appearance of coffee-grounds. The brighter the color of the blood the nearer is the source of hemorrhage to the anus. The more retarded the passage the greater the change; while, if quickly expelled, blood from the small intestine may be passed unchanged, as in the hemorrhage of typhoid fever. The microscope detects blood when the naked eye fails to detect it. It is to be remembered that certain drugs, as already stated, may color the feces red, and simulate blood.

Mucus may be present in the passages in health, but when in any marked quantity there is a catarrh of the mucous membrane of the intestines. When hard scybala are covered with mucus, or the mucus is seen in shreds, the large intestine is the seat of a catarrh; although mucus may be mixed with thin stools, as in dysentery. Usually, however, when the mucus is finely divided and mixed with the feces, it comes from the small intestine. Mucous shreds have already been mentioned. In cholera the particles of mucus look like boiled rice, hence the term "rice-water stool."

Fatty stools, to the naked eye, appear greasy or even clayey, when there is much fat, even though bile-pigment may be present.

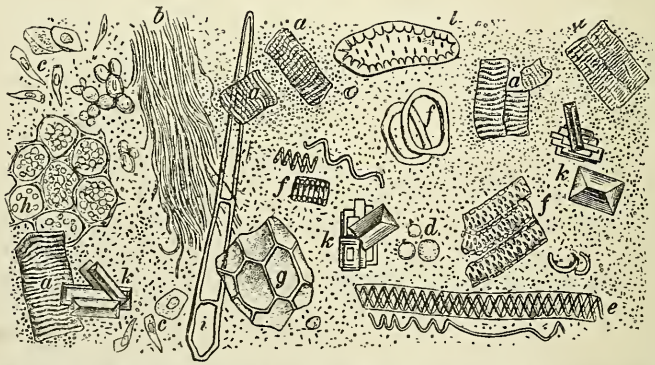
Pus may be present in large quantities from rupture of an abscess into the intestinal tract, or when there are ulcerations from various conditions, producing pus in considerable quantities.

Microscopical Examination of the Feces. Many animal parasites are not microscopic, but it is convenient to consider them in the following paragraphs. A small portion of the solid feces to be examined is placed on a slide, moistened with a $\frac{1}{2}$ per cent. salt solution, and a cover-slip applied; or if liquid, various drops are to be examined. The different constituents found will vary with the food taken as well as with disease.

A. Constituents Derived from Food. There may be portions of digested or undigested food. In general it may be said that the presence of large pieces of unchanged food, or many small particles of undigested or only partially digested food, indicates defective digestion in the stomach or small intestine. If unchanged bile is present, some particles will be colored yellow, another indication of disordered function.

From the *food* we may see muscle and elastic fibres, more or less, according to the quantity of meat eaten by the patient. The former are recognized by their transverse striation; the latter, by their double contour and curling ends. Fat may be present as fatty globules or in the form of needles, fatty crystals. Much fatty food increases their number, and they are seen plentifully in alcoholic poisoning, in jaundice, in pancreatic diseases, tuberculosis of intestines, diseases of the mesenteric glands, and enteritis. The crystals may be trans-

Fig. 197.



Collective view of the feces. (Eye-piece III., objective 8A, Reichert.) *a*. Muscle-fibres. *b*. Connective tissue. *c*. Epithelium. *d*. White blood-corpuscles. *e*. Spiral cells. *f-i*. Various vegetable cells. *k*. Triple phosphate crystals in a mass of various micro-organisms. *l*. Diatoms. (Von Jaksch.)

formed into fat-drops by the addition of acid and heat. When meat is eaten freely, areolar tissue may be present, but its presence otherwise points to defective digestion. Various forms of vegetable cells are commonly seen, in which granules of starch may be contained, or the

starch particle may be free. Undigested milk occurs in the stools of children and when diarrhœa prevails; a substance, probably casein, has been described by Nothnagel as occurring in the feces of persons who have intestinal disturbances.

In persons living on vegetables most of the above constituents will be absent, and in infants who partake only of milk, the derivatives of meat are absent, while there will be an excess of fatty crystals and fat-globules and coagulated products.

B. Constituents from the Alimentary Tract. *Epithelium.* In every normal stool will be found epithelium of the squamous variety.

Occasionally the columnar form is seen, and modified epithelial cells are very common. In intestinal catarrh their number is greatly increased.

Red Blood-corpuscles. In the majority of blood-stained stools red blood-cells are not found; in their stead will be seen masses of free blood-coloring matter and rhombic crystals of hæmatoidin. Red cells are seen in dysenteries, in bloody stools in which the blood comes from near the anus, as in hemorrhoids, and when blood is discharged with the feces soon after the occurrence of the bleeding. If there is any doubt as to the presence of blood, when the corpuscle cannot be found, a true decision can be reached by examining for hæmin-crystals, according to Teichmann's method. A portion of feces is dried and powdered, placed on a slide with a grain of common salt, and covered by a cover-slip. A few drops of glacial acetic acid are directed beneath the slip, the slide is heated just to boiling, and if blood has been present, reddish-brown rhombic crystals of hæmin will soon be found.

Leucocytes. Leucocytes are frequently seen in healthy stools. When pus is present or discharged into the intestinal canal they are found in great numbers, as in ulceration of the intestine and in abscess.

Molecular débris, or detritus, occurs in all feces as part of the waste-products.

Crystals. Fat-crystals are the most important. They have been quite fully considered above. There seems to be little doubt that the crystalline needles found in the feces are salts and fatty acids, and not tyrosin.

Charcot-Leyden crystals, similar to those already described (see Sputum), have occasionally been met with in the stools of typhoid fever patients, in dysentery, intestinal tuberculosis, and ankylostomiasis.

Hæmatoidin-crystals occur as reddish-brown, hard, needle-shaped bodies, usually in clusters, and free or enclosed in masses of mucin or a substance resembling it. They have been found in the feces of breast-fed infants, in cases of chronic intestinal catarrh, and, by Von Jaksch, in the stools of a case of nephritis.

Crystals of various *salts of calcium,* of *triple phosphate* and *cholesterin* will often be recognized, but they have no diagnostic value. When bismuth is being administered, black rhombic crystals of the sulphide of bismuth will be recognized.

C. Parasites. (*A*) *Animal* and (*B*) *vegetable* parasites flourish in the intestinal tract, and the presence of some of these in the feces is of the greatest clinical importance.

A. Animal Parasites. Following Leuckart's classification, we will consider these parasites under the secondary heads :

I. Protozoa. 1. *Rhizopoda.* This variety is made important because the amœba dysenteriæ or amœba coli belongs to it.

(*a*) *Amœba Dysenteriæ. Amœba Coli.* This protozoön has been found so many times by various observers in different parts of the world that it can now be considered to be the causative factor of so-called tropical dysentery. The subject has received special study in our own country by Osler,[1] Stengel,[2] Dock,[3] and Councilman and Lafleur.[4] The work of Councilman and Lafleur is at the present time the best that has been published in any country ; and to it the reader is particularly referred. The following notes are based on this book.

The amœbæ dysenteriæ vary in size from 0.012 to 0.035 mm. They are found most plentifully in the small gelatinous masses often to be seen in the feces. They vary in number in different cases, and in the same case at different times. The severer the lesions the more numerous are the amœbæ. When not active they are round or oblong, and highly refractive. They contain one or more vacuoles of varying size. Occasionally the division into an ectosarc and endosarc is easily made out. When thus inactive they may be confounded with swollen connective-tissue cells and compound granular bodies found in feces. The active amœbæ have, however, a characteristic movement. This consists of progression and of thrusting-out and retraction of pseudopodia. Their activity varies greatly. It is best seen when the body-heat is maintained. The stools should be passed into a clean and warm pan, and examined immediately, or kept warm until examined, and a warm

Fig. 198.

Amœba coli. (Hallopeau.)

stage should be used with the microscope. The division into ectosarc and endosarc is usually clear during activity. The ectosarc is composed of a hyaline homogeneous mass, as are the pseudopodia, while the

[1] Johns Hopkins Hospital Bulletin, May, 1890, vol. i., No. 5.
[2] Phila. Med. News, 1890.
[3] Texas Med. Journal, April, 1891.
[4] Johns Hopkins Hospital Reports, vol. ii., Nos. 7, 8, 9.

endosarc is made up, not of granular matter, but of a dense homogeneous mass enclosing vacuoles and a nucleus. The vacuoles may vary in size as well as in number. There may be one or two large ones, or the entire endosarc may appear as made up entirely of small vacuoles. The nucleus is sometimes plainly seen as a small rounded body, but is more often difficult to distinguish from the vacuoles. Dried cover-slip preparations may be stained with the various aniline dyes, but the results are not satisfactory.

The amœbæ will often be found to enclose bodies such as red blood-corpuscles, pus-cells, blood-coloring matter, bacilli and micrococci.

In examining the feces for amœbæ dysenteriæ the suggestion given above concerning the warm bed-pan and warm stage to the microscope, and, above all, the immediate examination of the stool, should be adhered to. The small gelatinous masses should be selected when present. Various magnifying powers should be used, including the $\frac{1}{12}$ oil-immersion lens.

(*b*) *Monadines*, pear-shaped, with a long slender process, are seen alive in only perfectly fresh stools. They are not found constantly in any one disease.

2. *Sporozoa*. Under this head belongs the coccidium perforans of Leuckart. They are short, elliptical bodies, which infest the intestinal mucous membrane, and may damage it badly; they are often discharged in large numbers.

3. *Infusoria*. (*a*) *Cercomonas Intestinalis*. This is a pear-shaped body, nucleated, with eight tentacles of varying length. It is found in the feces of persons suffering from various diseases, as cholera and typhoid fever, and probably of itself causes diarrhœa.

(*b*) *Trichomonas intestinalis*. Larger than the cercomonas, and covered with ciliæ at the club end. It is not diagnostic and is not common.

(*c*) *Paramœcium coli*. Larger than the preceding, 1 mm. long—oval, covered everywhere with ciliæ; may be found in diarrhœic stools.

II. Vermes. These are much more generally known and are of much more clinical value than the preceding.

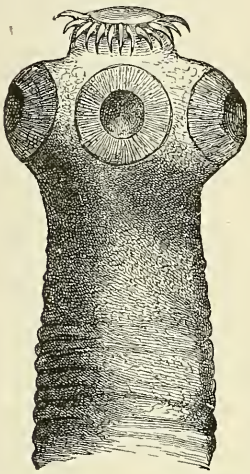
They have important clinical value, as the presence of some of them in the intestinal canal gives rise to many untoward symptoms. They will be considered under (*A*) *Platodes* and (*B*) *Annelides*.

A. Platodes. 1. *Tapeworm—Cestodes*. These parasites infest the small intestine only, to the walls of which they cling by the head. The head and neck are small; the joints are flat and form long ribbons. The distal joints continually drop off and can easily be recognized in the stools by the naked eye, and the eggs by the use of the microscope. The feces are best washed in water and broken up to obtain the eggs. As the lower joints are lost new ones take their place from above. The more important are as follows:

a. *Tænia solium* (Fig. 199) reaches a length of two to three metres. The head is the size of a pin-head. The neck is 2.5 cm. long, as thick as a thread, and without joints. The segments forming the body are short and broad near the neck, but as they increase in size there is more growth in length than in width. The average dimensions are 9

to 10 mm. by 6 or 7 mm. The head appears dark, the body white. The joints are easily detected in the feces by the naked eye. Under the microscope the head is seen to be spheroidal, with four pigmented sucking-disks surrounding at the base a rostellum, which is a "crown of hooks"—chitin hooks—about twenty-four in number. In the ripe segments, or proglottides, is seen the longitudinal uterus with about twelve horizontal ramifications to a segment. The eggs are round or oval, 0.035 mm. long, with a thick, striated shell when ripe, and contain hooklets.

FIG. 199.



Head of T. solium. × 45. (LEUCKART.)

FIG. 200.

Ova of T. solium. *a*, with yolk. *b*, without yolk, as in mature segments. The hard brown shell is indicated. (LEUCKART.)

b. Tænia mediocanellata, or *saginata*. This worm is four or five metres long. The head is slightly larger than that of the T. solium, and more pigmented, and the segments are longer, fatter, and darker. The head is supplied with four powerful sucking-cups, but has no rostellum or hooklets. The uterus in the ripe segment is much more finely branched than in the solium, and these segments have independent movement. The eggs are very similar to those of the T. solium, but may be rather larger.

c. Tænia nana. In length the T. nana is only 10 to 15 mm., and 0.5 mm. in breadth. The round head is but 0.3 mm. in diameter. The segments are all short, and at the lower end of the body are four times as wide as they are long. The head is found to have four round suckers at the base of a rostellum that can be inverted. At the base of the rostellum are about twenty-two hooklets. The uterus is oblong and filled with eggs. The eggs have a double membrane.

d. Tænia cucumerina. This parasite is found to be 5 to 20 cm. long and about 2 mm. wide. The head is placed at the thinner end, and under the microscope are to be seen some sixty hooklets regularly distributed about the rostellum, and four sucking-cups. The lower segments are decidedly larger than the upper—6 by 7 mm. When

ripe they become reddish, and contain cocoon-like bodies, in which are six to twelve eggs.

e. Bothriocephalus latus. This is the largest of the worms, measuring 7 or 8 metres. The head is somewhat drawn out, and on either side is a long, narrow sucker. There are neither hooks nor rostellum. The proglottides are short near the head, but become square further down. The uterus appears as a rosette, peculiar to this worm. The eggs are oval, and measure 7 mm. by 0.045 mm., have a shell covering, with an opening like a lid at one end. Ripe segments are thrown off in bunches, not singly.

It will not be necessary to describe certain other varieties that are rarely met with.

2. *Trematodes*, or *flukes*. *a. Distoma hepaticum* measures 28 mm. by 10 mm., and is shaped like a leaf. A short head is situated at the broad end and has one sucker ; on the under surface is another sucker, and between the two is the opening of the uterus, a highly convoluted arrangement. The eggs are brown, oval, about 0.12 mm. long, and have a lid at one end. It is not often seen.

b. Distoma lanceolatum. This round-shaped worm is about 8 mm. long and 3 mm. broad, and in other respects resembles the preceding. The eggs are more rounded and contain minute embryos. Like the D. hepaticum, it is rarely seen.

c. Distoma crassum is the largest—4 to 8 cm. long. These flukes are endemic in parts of Japan. In general these animals occupy the bile-passages or upper part of the small intestine.

B. Annelides. 1. *Round Worms—Nematodes.* A. *Ascarides. a. Ascaris lumbricoides.* This is the parasite usually referred to by the term round worm. It resembles the common earth-worm in shape and color. The male worm is about 250 mm. long, and the female 400 mm. The head is made up of three prominent lips, and is supplied with microscopical teeth. The vulva of the female is in the posterior third of the body. The eggs are rounded, brownish, 0.06 mm. in diameter, and covered, when fresh, by a rough albuminous coat over a hard shell. This worm has the small intestine for its habitat. It may pass with the stools or work its way into the stomach and be vomited (the writer has had them thus vomited during the etherization of a child of ten years). They have been the cause of jaundice by crawling into the ductus choledochus, and may infest the larger hepatic ducts. Enormous numbers may be present in the intestine at one time.

b. Oxyuris vermicularis. The thread-worm, or seat-worm, inhabits the large intestine, and is often present in the stool as a white, thread-like body ; the male 5 mm. and the female 10 mm. long They often wander out of the anus and into the vagina. The head has a number of small lips, and is covered with a thick skin. The female has one vagina and two uteri. The eggs are unsymmetrical, have a laminated shell and a diameter of about 4 mm.

B. *Strongylides. Ankylostomum duodenale.* This is a round worm, reaching a length of 6 to 10 mm. in the male and 10 to 18 mm. in the female, and can, therefore, be seen easily, though the eggs are

much more frequently found in the stool than is the worm itself. With the eggs there may be present in the stools large numbers of Charcot-Leyden crystals. The head is prominent, especially in the male. Four hook-like teeth surround the mouth, and by these the animal attaches itself to the intestinal wall. The tail of the male is expanded and that of the female pointed. The vulva is in the posterior third. The eggs are oval, about 0.05 mm. in diameter, and contain one to four cells—embryonic globules, which rapidly develop in a warm place outside the body, and may thus be recognized. The worm infests the small intestine, especially the jejunum. It often causes serious symptoms—bloody stools and intense anæmia.

c. *Trichotrachelides. a. Tricocephalus dispar.* The whip-worm is 4 to 5 cm. in length, the female being longer than the male. It is recognized by the contrasting form of the anterior and posterior portions. The former is thin and threadlike, the latter expanded and broad, and in the male curled up. The eggs are brownish, about 0.05 mm. long and half as broad, and have a button-like projection at either end; they are to be recognized in the stools, where large ones may be present. There may be only a few or thousands of the forms present in the body. They live chiefly in the cæcum and large intestine. They have been thought to cause beri-beri by some writers.

b. Trichina spiralis. It is the adult trichinæ which exist in the intestine and are found very infrequently in the feces. These produce the embryos, which become muscle trichinæ. The adult male is 1.5 mm. long and the female twice that length. The former has two projections from the hinder end, between which are four papillæ. The female has a tubular uterus and a tubular ovary in the posterior half of the body.

d. *Rhabdonema. Strongylides.* Under rhabdonema intestinale we now include two small nematodes, which were termed anguillula intestinalis and A. stercoralis, and which are probably one and the same. They are found in the stools of cases of endemic diarrhœa of hot countries. Usually the young embryos, which have developed in the intestinal canal, are dejected with the stools. These sexually mature embryos are 0.8 to 1.2 mm long, male and female respectively. They are round and have a cone-shaped head. There are two jaws and two teeth in each. The adult worm is about 2.2 mm. long and 0.04 mm. thick. The mouth has three lips. The vulva is at the beginning of the posterior third. The eggs might be easily confounded with those of the ankylostomum duodenale, but are somewhat more pointed, and larger. The rhabdonema infests the small intestine, and is frequently found in connection with ankylostoma.

Echinococcus hooklets and portions of the striated cyst-wall have been found in the feces. The rupture of a hydatid cyst into the intestine may be discovered when the above structures are found, pointing to a cyst in the abdominal cavity.

B. Vegetable Parasites. We find both (I) *pathogenic* and (II) *non-pathogenic* vegetable parasites in the feces. The latter we have classed as (1) moulds, (2) yeasts, and (3) fission-fungi.

1. *Moulds.* The only mould found in the stools is the thrush

fungus, when children are the subjects of thrush in the mouth. It is of very rare occurrence in the feces, and has no special clinical import.

2. *Yeasts.* In all feces, in health or disease, yeast fungi exist. They are most numerous in acid stools. They are round or ovoid, and usually occur in groups. They stain dark brown with a solution of iodine and iodide of potash, while apparently similar cells become violet or blue with the same dye.

3. *Fission-fungi.* Bacteria are found in greatest numbers in the feces, chiefly as bacilli, micrococci, and spirilla. They may be grouped as torulæ or sarcinæ. They present active movement, and may be separate or in colonies. The bacillus coli communis (*B. termo*) is the most frequent form met with, both in health and disease. It is not yet determined what relations it holds to normal and abnormal conditions, or what is the true relationship between it and certain other bacteria. B. subtilis is another bacterium found both in health and disease. As above stated, there are various organisms which stain brown with iodo-potassic-iodide solution, and others which become blue with the same dye. Von Jaksch has studied these latter closely. They take various forms, as long or short rods, and take different shades of blue or violet. One of them is the clostridium butyricum of Nothnagel. It occurs as large round cells, like yeast fungi, and stains like the tubercle bacilli with the Ziehl-Neelsen fluid. Von Jaksch finds these fungi in greater abundance in intestinal catarrh. They are present in both acid and alkaline stools.

Bacillus coli communis has been found in the blood, various organs, feces of cholera patients, in healthy feces, in the air, and in putrefying infusions; it can also be found in the peritoneal exudate in most cases of peritonitis.

Morphology. A bacillus, 4 to 6μ by 2 to 3μ, with rounded ends, sometimes in cultures a short oval. Five or more flagella have been observed attached to the organism.

Biological Properties. Aërobic; facultative anaërobic; non-liquefying; slightly non-motile.

Growth. On gelatin plates the colonies vary very much. The deep colonies are transparent, straw color to dark brown, or may be granular and opaque. The surface-colonies are large and spherical, centre dark brown, edges transparent. In stab-cultures the surface-growth is thin and dry. There is abundant growth along punctures, which is white by reflected, but amber by transmitted light; sometimes moss-like tufts are seen. On potato a soft, shining, brownish-yellow layer grows. Stains with anilines, but not by Gram's method. Injected in guinea-pigs it produces fever, diarrhœa, and collapse. Injected into the abdomen of rabbits it causes a typical peritonitis.

Pathogenic Fungi. Spirillum Choleræ Asiaticæ. See page 338.

Spirillum Cholera Nostras. *Morphology.* Longer and thicker than the spirillum of Asiatic cholera; central part thicker than ends. Stains as the true cholera spirillum.

Biological Properties. Culture. A thick, stocking-like funnel of liquefaction instead of a fine, straight funnel. (See Fig. 87, page 340.)

Typhoid Fever Bacillus. This bacillus is present in the stools of typhoid fever patients, but cannot be directly differentiated by microscopic examination alone, either when stained or unstained. It is necessary for its detection to make pure cultures according to bacteriological methods. The bacillus is about as long as the tubercle bacillus, but much thicker, being one-third as thick as it is long. The ends are rounded. It is best stained by concentrated aqueous solutions of methylene-blue, the dried preparations on the cover-slip being prepared as above. (See Plate III., Fig. 6, B; and Typhoid Fever).

Tubercle Bacillus. The bacillus of tuberculosis is frequently found in the feces of persons suffering from intestinal tuberculosis and occasionally in the feces of cases of pulmonary tuberculosis, when sputum has been swallowed. When tubercle bacilli are constantly found in the feces, and in large quantities, it points to the former condition almost to a certainty. They are detected by methods employed in the examination of sputum.

Bacilli of Booker. No less than nine bacilli have been described by Booker. They have been found by him in cases of diarrhœa in children. Seven of them resemble very closely the bacillus coli communis. Bacillus A is a bacillus with rounded ends, 3–4μ by 0.7μ. It is aërobic and facultative anaërobic, liquefying, and motile. Colonies on agar and potato are dirty brown. On gelatin they liquefy too soon to show characteristic form. The bacillus is found in the stools of cholera infantum.

Chemical Examination. The *chemical examination* of the feces is of but slight clinical value. Mucin and albumin are normally present; peptones in different diseases (Von Jaksch). Among the acids to be found are bile-acids, volatile and fatty acids, formic, acetic, butyric, and propionic acids; while phenol, indol, skatol, cholesterin, and fats are always present, according to the same author. They will not aid in diagnosis.

The normal coloring-matter of the feces is urobilin; its presence is shown by the proper tests. As before stated, bile-pigment never occurs in the feces in health; it is present when there is catarrh of the small intestine. Blood-pigment is usually in the form of hæmatin. As might be expected, ptomaïnes have been obtained from the feces of certain diseases caused by fungi.

Diseases Characterized by Pain and Flatulence.

Intestinal Indigestion. Intestinal indigestion is said to be due to alterations in or diminution of the bile, the pancreatic, or the intestinal secretion. It is almost always attended by gastric indigestion, and may not readily be distinguished from it.

Acute Intestinal Indigestion. *Acute intestinal indigestion* is due to the irritation of food not properly digested in the stomach. It is attended with colic, flatulency, and borborygmi. Some fever may develop, and diarrhœa may ensue. In the mild forms the tongue is coated, there are loss of appetite and some general pains. There is

epigastric distress or pain in the right upper quadrant. Flatulency and constipation occur. The stools are often clay-colored, or may not be changed. Slight jaundice occurs, and there is an abundance of lithates in the urine. Accompanying gastric indigestion modifies the symptoms slightly.

The symptoms are more marked and pronounced in *chronic intestinal indigestion*. The *local symptoms* are as follows: Pain which begins from two to six hours after eating. It may be complained of in the region of the liver or below the sternum. It is usually seated in the umbilical region. It is dull and continues two or three hours, or until the next meal is taken. There is some tenderness. With the pain there are tympanites, borborygmi, and a sense of fulness in the abdomen; the bowels are constipated, and the stools are hard and dry. The constipation alternates with diarrhœa, and undigested particles of food are passed. The appetite is not lost, but is variable. Hemorrhoids are often present.

The *general symptoms* are marked, and are referred to the nervous system and the condition of the blood. There are great depression and hypochondriasis. The patients sleep badly, suffer from bad dreams and tinnitus aurium; there are spots before the eyes and more or less constant headache. They complain of pain in the back and limbs, and hyperæsthesia and anæsthesia are present. There is inaptitude for mental exertion. Frequently the patient has sudden attacks, apparently due to toxins, as sudden fainting followed by collapse, or vertigo. During these attacks there are great palpitation and tachycardia. The extremities are cold, and there are cold sweats over the body. Independently of the attacks, the patient is subject to palpitation and some dyspnœa. The urine is always high-colored, acid in reaction, and full of urates and uric acid. Oxalate of lime may be present, and the albuminuria of uric acid occurs, due to the irritation. The patient early becomes anæmic, because of the auto-intoxication and poor assimilation. There is some emaciation; in some cases the emaciation is rapid. The complexion is sallow. If there is an abundance of oxalates, the patient complains of weight and heaviness about the loins. The stools may contain fat, indicating probable pancreatic disease, if fatty food has been ingested. On the other hand, with loss of appetite, furred tongue, frontal headache, and drowsiness, the stools may be clay-colored and the bowels costive; apparently the bile is at fault.

Diseases Characterized by Pain and Diarrhœa.

Acute Intestinal Catarrh. *Cause.* Exposure to cold or the direct irritation of mechanical or chemical substances within the intestine. Irritating food that is not digested, or that cannot be digested because of the quantity; spoiled meats and unripe fruit usually excite an attack. Water saturated with impurities, or such as the individual is not accustomed to, may excite an attack. Strangers in a new locality are frequently subject to a diarrhœa until accustomed to the drinking-water, which in the native does not excite catarrh. Toxic substances, as poisons or drugs, or toxic substances the result of putrefaction, as

ptomaïnes, are frequent exciting causes. Extension of inflammation from neighboring structures by infection, as in peritonitis, sets up a catarrh. Local diseases of the intestine, as ileus, intussusception, hernia, and ulcers of all forms, are attended by catarrh of the intestine. It also occurs in cachectic states of the system, as cancer, anæmia, and Bright's disease. In disease of the heart and bloodvessels, or of the liver and spleen, where the disturbance of the circulation causes a congestion, catarrhal inflammation occurs. It is of common occurrence in the infectious diseases, and particularly in septicæmia and pyæmia.

Symptoms. *Diarrhœa* is the chief symptom, varying with the cause and the extent of the catarrhal inflammation. The stools differ in frequency and in color, as has been previously indicated in the various types. They contain undigested matter; sometimes worms. Colicky pains about the umbilicus, with borborygmi and frequent desire to go to stool, attend each evacuation. The fever is of the remittent type, and is attended with some prostration. The urine is scanty and high-colored. The symptoms vary somewhat with the location of the inflammation, although the exact locality cannot be distinctly defined. The symptoms of proctitis, pain with tormina and tenesmus, do, however, enable the localization to be made to that portion of the bowel. These are more common than in inflammation apparently limited to the small intestine, while in colitis the violence of the rectal symptoms stands between enteritis and proctitis.

The *diagnosis* of acute intestinal catarrh is not difficult. It is more difficult to determine the actual cause. If the attack occurs suddenly after the eating of improper food, or the drinking of impure water, the irritation is probably due to that cause, and may be determined by the nature of the feces. If they contain undigested food, the diarrhœa is probably due to indigestion. Catarrh from cold usually follows exposure, and is generally not very severe. To estimate the cause from poison or drugs the condition of the rest of the intestinal tract must be investigated and other symptoms of the effects of drugs must be inquired for. In arsenical poisoning there is always vomiting and the discharges are of a choleraic nature. Collapse rapidly ensues. The other symptoms of arsenical poisoning must be inquired for and the history of exposure, if possible, ascertained. The intestinal catarrh due to infectious diseases is attended by the symptoms due to the respective affections, each of which is usually readily recognized. It may be necessary to resort to a bacteriological examination of the feces. The intestinal catarrh which occurs on account of local disease of the bowel, as hernia, stricture, etc., is preceded or attended by the local symptoms of these diseases. In like manner we judge of the nature of the diarrhœa that occurs in the course of tuberculosis or syphilis, and in the course of organic heart disease or of liver disease. In each instance the possible influence of morbid processes present in other structures must be very carefully estimated.

The Varieties of Acute Intestinal Catarrh. Divisions have been made in accordance with the symptoms which distinguish the various localities of the intestine in which the inflammation is most marked.

Catarrh of the Duodenum. This partakes of the nature and has the symptoms of gastro-intestinal catarrh in a mild degree, and is characterized by the occurrence of jaundice due to catarrhal inflammation of the biliary passages.

The Small Intestine. Colicky pains and rumbling are experienced. There is usually gastritis at the same time. The feces are mixed with mucus. Over the right lower quadrant there is tenderness on pressure.

Cæcum. Pain in the right lower quadrant with tumor, dulness on percussion, and tenderness are present. (See Typhlitis.)

Colitis. The large intestine is most frequently affected. Pain and tenderness occur along the course of the bowel. The evacuations contain mucus; there is tenesmus. The association with gastro-enteroptosis and with neurasthenia must be borne in mind.

The Rectum. Proctitis gives rise frequently to small stools, tenesmus, pain in the left lower quadrant, with tenderness about the anus, and spasms of the sphincter. There are considerable mucus and blood in the passages.

Cholera Infantum. This affection occurs in children during the hot season. It is promoted by bad hygienic surroundings, and is due to improper milk or food. At first there is catarrhal diarrhœa. This may continue for twenty-four hours, then vomiting and diarrhœa ensue. The stools are liquid and large in amount. At first they may contain milk-curds. The vomiting is excited by anything taken into the mouth, or by odors, or by movement of the little patient. The watery discharges are almost constant. They may be preceded by greenish or yellowish-green stools for twenty-four hours. Stools are acid in reaction, and their odor is sour. At first there is colicky pain, but when the watery discharges begin there is only a little tenesmus. The stools irritate the skin and cause eczema. The rectum may become prolapsed. The abdomen is at first distended with gas, but soon becomes retracted.

In a short time, twenty-four hours or even less, collapse ensues. Previous to the collapse the skin is hot and dry; the patient is restless. The thirst is intense, the mouth dry. The body-temperature is 103° to 104°. With collapse the extremities become cold, the skin cool. The axillary temperature is lowered and the rectal temperature increased to 105° to 106°. The restlessness continues, the fontanelles become depressed, the eyes sunken, the face pinched, the brows drawn. The urine diminishes in amount or may disappear entirely. Brain symptoms ensue. So-called hydrocephaloid symptoms follow—rolling of the head, strabismus, turning in of the thumbs, and, later, convulsions. Stupor followed by coma develops in the fatal cases. If the patient does not die in collapse, marasmus develops; ulceration of the cornea may take place; there are œdema and blood extravasation under the skin. The child emaciates and withers. On account of the weak heart and exhaustion pulmonary atelectasis or bronchopneumonia may occur. The age, the season, the presence of catarrh, with collapse and other symptoms, render the diagnosis easy.

Cholera Morbus. The attack is characterized by sudden vomiting, followed in a short time by purging. The vomiting may be preceded

by pain, or both may occur at the same time. At first the pain is seated in the epigastrium and subsequently about the navel. It is very severe and paroxysmal in character, compelling the patient to double up if lying in bed. A cold perspiration breaks out on the forehead, the extremities become cold, the face anxious; the pulse becomes rapid. At first the patient vomits undigested food, then watery, greenish-colored fluid. The latter is bitter. Purging sets in at once, or within an hour. The bowel-movements follow an attack of pain. The first passage is fecal, and may contain undigested food; the subsequent passages are watery and profuse. There are severe attacks of burning and tenesmus; the abdomen is tender around the navel and in the epigastrium. After an evacuation there is slight relief, but soon another paroxysm of pain comes on. The vomiting is excessive, and retching may be present in the intervals. Ice, or water, or anything taken into the stomach excites pain and causes the vomiting. The attack subsides in twelve to twenty-four hours, and is followed by exhaustion. In rare cases collapse ensues, and in others it is followed by gastro-intestinal catarrh.

Cholera Nostras. The symptoms are those of severe gastro-enteritis. There are sudden vomiting and diarrhœa. It usually begins in the night. The vomiting is not different from that of cholera morbus. The watery and brownish-colored stools become colorless and have the appearance of rice-water. Pain attends the attack, rapid prostration ensues, the extremities become cold, and collapse takes place. With the collapse there are cramps in the legs. Other muscles of the body may become cramped. The disease occurs in epidemics during the hot season, and may be mistaken for cholera. It can be distinguished from the milder forms of cholera which precede the occurrence of the epidemic only by the absence of the comma-bacillus. The bacillus of cholera nostras is found in the stools. (See Feces.)

Entero-colitis. In entero-colitis the more intense inflammation succeeds a mild intestinal catarrh. There are increased languor, great fretfulness, and fever. The early catarrh is attended by green acid stools, with lumps of casein. The tongue is furred and moist at first. It soon becomes red and dry; vomiting ensues. The stools are offensive and increase in frequency, and, in addition to the appearance first indicated, contain mucus and blood. Death may take place within the first week, on account of exhaustion from the vomiting and diarrhœa.

If the disease is protracted, it is attended by great wasting, symptoms of hydrocephalus, skin eruptions, hypostatic pneumonia, and extremely weak, feeble circulation.

Chronic Intestinal Catarrh. It usually follows an acute attack, or may be chronic from the start. It may follow gastric hyperacidity and dilatation of the stomach. It arises secondarily to portal congestion in disease of the liver and in chronic disease of the heart or of the lungs. It occur in malaria and in the scorbutic cachexia.

The symptom is diarrhœa alternating with constipation, or diarrhœa alone. Stools may contain undigested food, or pus, mucus, and blood in small amounts. Diarrhœa may be present in the morning only under these circumstances. If the feces are examined, the eggs of

parasites, or infusoria may be found. The local abdominal symptoms of rumbling, flatulency, and tormina are present. There are reflex symptoms of cardiac palpitation and dyspnœa (asthma). Rush of blood to the head may occur. Often these symptoms are relieved by the passage of flatus. Chronic catarrhal gastritis usually accompanies the intestinal catarrh. The general symptoms of *anæmia*, *emaciation*, and *neurasthenia* are present. Hemorrhoids are common.

Amyloid Degeneration of the Intestines. The symptoms are those of diarrhœa, persistent but mild in character, associated with symptoms of amyloid disease in other organs. With enlargement of the liver and spleen changes in the urine due to amyloid disease are present. The occurrence of these symptoms in a patient with syphilis, or especially in a child with bone disease or tuberculosis, points to the nature of the case.

Ulceration of the Intestines. Duodenal Ulcer. Ulcer of the duodenum usually occurs in young subjects in whom there are symptoms of chlorosis or anæmia. The causes are the same as those of gastric ulcer. It may follow boils, erysipelas, or pemphigus, and differs in one etiological respect from ulcer of the stomach, in that it occurs more frequently in the male sex. The symptoms are obscure, and may be wanting entirely, the patient probably complaining only of intestinal indigestion. In other cases they are like those of gastric ulcer. In typical cases the symptoms are those of pain situated below the xiphoid or to the right of the median line in the region of the pylorus. The pain occurs after eating, and may be relieved by vomiting. There is localized tenderness on pressure. Hemorrhage may take place from the stomach, or blood be found in the stools alone. It differs from gastric ulcer only in the possible difference in location of the pain, the occurrence of intestinal indigestion and hemorrhage, and the fact that the pain comes on one to two hours after eating.

Duodenal ulcer is diagnosticated by the occurrence of melæna, which may be excessive and cause syncope and vomiting with no blood in the vomitus; by pain, which may be in the right hypochondrium or between the navel and the right costal border; by gastralgic attacks; by dyspepsia, with constipation.

General Ulceration. Ulceration of the intestine may be due to a specific infection, and hence be symptomatic of typhoid fever, syphilis, and tuberculosis. It is always present in the first mentioned, and of frequent occurrence in the latter. Follicular ulceration occurs in entero-colitis in children. Ulcers due to the pressure of feces occur in typhlitis and chronic constipation. The sacculi of the colon become filled with scybalous masses, the pressure of which produces ulcers. Tenderness is experienced along the course of the colon, particularly on palpation of the fecal masses, which may be felt through the abdominal wall. A *chronic ulcerative colitis* is the form that succeeds the diarrhœas which occur during camp-life, or that are set up in communities where people are crowded and live under bad hygienic circumstances. It is the form that attends scurvy, and is frequently seen in chronic Bright's disease. It may be succeeded by dilatation of the colon, by hypertrophy of the muscular walls, or by contraction of the

bowel. The persistent diarrhœa leads to profound emaciation, extreme prostration, sallow complexion, with markedly impaired nutrition of the skin. Such forms of diarrhœa were seen during the late war, particularly in soldiers held in captivity. The diarrhœa may first be of a lienteric character, and later alternate with constipation. Stools contain blood and mucus.

Ulcers of the intestinal tract may occur from other causes, and diarrhœa may be the predominant symptom. They may be due to *cancer;* the malignant nodules may ulcerate within the lumen of the bowel. The bowel may be perforated from the exterior, on account of suppuration somewhere along its course, as in appendicitis, pancreatitis, or tuberculous peritonitis.

Symptoms. The *symptoms* of intestinal ulcer are usually those of diarrhœa. Ulceration, however, may be present without any symptoms whatsoever, particularly if the small intestine is affected. One or two small ulcers, on the other hand, in the lower portion of the colon, may set up continuous diarrhœa. The *stools* are composed of feces, mucus, pus, shreds of tissue, and blood. If pus is discharged in large amounts, an abscess has probably opened into the bowel. Moderate discharge of pus usually follows ulcers in the colon. Pus may be present in cancer. *Hemorrhage* is of frequent occurrence, and is an important diagnostic symptom, especially if profuse and occurring without symptoms of obstruction, of gastric ulcer, or of hemorrhoids. The fragments of tissue found in the stools may point to the nature of the process. Large amounts attend the dysenteric process. The fragments may be composed of the mucosa, connective tissue, and the muscular coat. *Pain* occurs in many of the cases. It may be general and colicky, or circumscribed in cases of ulcer of the colon. Perforation of the intestine is followed by localized or general peritonitis. The occurrence of the latter depends largely upon the situation and the rapidity of the ulceration. If the perforation is in the posterior wall of the colon, a circumscribed abscess may develop. When it is situated in the upper zone the pus may accumulate underneath the diaphragm, or in the lesser peritoneal cavity. The signs of pyo-pneumothorax subphrenicus occur when the latter accident takes place, as both pus and air accumulate in the abscess-cavity. In such instances the ulceration usually takes place at the splenic flexure. Perforation of an ulcer of the cæcum may simulate appendicitis.

Tuberculosis of the Intestine. The disease is usually secondary to chronic tuberculosis, but may be primary, especially in children. The symptoms are usually those of *diarrhœa,* and in the primary form this is associated with general emaciation, which advances rapidly, and with anæmia. Fever of the intermittent or remittent type is present. There is meteorism; the abdomen is much distended, but eventually becomes contracted. The mesenteric glands can be made out along the spinal column, and the intestines may become bunched into a mass, yielding a dull tympany on percussion in the centre of the abdomen. The diarrhœa is attended with colicky pains. The diagnosis is based upon the rapid emaciation, irregular fever, enlargement of the mesenteric glands in a patient, usually a child, who had probably been ex-

posed to tuberculous infection. In one of my cases, the child, aged four years, ate of the same food, using the same utensils, as a brother, a young man of twenty-two years, dying of pulmonary tuberculosis. The child was constantly with the brother. The remainder of the family, eight in number, remained in perfect health, and were all of good physique. The elder brother became infected by association with tuberculous subjects in improper quarters away from home.

Intestinal Obstruction.

Intestinal obstruction may be acute or chronic. Acute obstruction may set in in the course of chronic obstruction due to stricture of the bowel, to occlusion due to external pressure, or to accumulations within the bowel.

Causes. *Acute intestinal obstruction* is due, first, to constriction by bands or strangulation of the bowel through apertures; second, to volvulus of the colon; third, to acute intussusception.

In the first instance the type of the obstruction is seen in strangulated hernia, but similar *strangulations* occur in apertures within the peritoneal cavity. Thus loops of the intestine are caught and constricted in the duodeno-jejunal fossa, the so-called Treitz' retroperitoneal hernia, or in the foramen of Winslow, also known as inter-sigmoid hernia; finally, diaphragmatic hernia, in which protrusions of the intestine through the diaphragm, along with other abdominal viscera, may take place. The above-mentioned forms of hernia may exist without symptoms, or may lead to constriction or twisting of the loop of the intestine, with occurrence of acute obstruction. Moreover, lacerations in the omentum may give rise to internal constrictions. External constrictions, however, take place, most commonly in the regions of hernias, on account of the gut being constricted by dense fibrous adhesion; or about the uterus or Fallopian tubes, which had previously been the seat of inflammation. The constricting bands that follow the local peritonitis may gradually occlude the gut, or be in such position that the latter becomes twisted about it. In other forms of peritonitis similar constricting bands may form, which are liable to produce this accident. Disease about the vermiform appendix, with secondary adhesions, has been observed to cause constriction. A frequent form of intestinal obstruction is due to the tangling of the intestines in the fœtal remains of the omphalomesenteric duct, Meckel's diverticulum, which is situated a short distance above the ileo-cæcal valve.

Volvulus is a form of acute obstruction due to twisting or knotting of the intestine. The condition is not common. It occurs most frequently at the sigmoid flexure of the colon. The mesentery of the latter is often congenitally narrowed, on account of which the colon is unduly dragged upon, and, if filled with masses of feces, cannot restore itself; the twisting becomes permanent, and obstruction takes place. Peristalsis is set up and other portions of the intestine wind about the pedicle of the loops, so as to form a regular knot. Abnormal peristalsis, on account of diarrhœa, often precedes the appearance of

the obstruction. *External injury* is said also to give rise to the formation of an obstruction.

Intussusception (Plate XXXIX., Fig. 1), as a cause of intestinal obstruction, occurs most frequently in children, and is due to a portion of the bowel being pushed into the lumen of that which lies next below it. A circumscribed portion of the intestine may be paralyzed. In the portion above, the peristaltic action continues and the energetic movements push it into the paralyzed part. Intussusception is found frequently after death in the bodies of children dying from exhaustion. In such cases it occurs just before death. Intussusception also occurs when intestinal polypi drag one portion of the bowel into the lower portion. Large portions of the intestine may be involved. The invagination usually takes place at the lower portion of the ileum, or into the cæcum ; sometimes the invaginated portion may reach the rectum and project externally. Intense inflammation and adhesion are set up. The internal portion becomes gangrenous, on account of constriction of the afferent vessels. This portion may slough and pass with the dejections, followed by spontaneous cure.

Chronic intestinal obstruction may be due to occlusion by external pressure, or by the excessive accumulation of material within the bowels, or to stricture. The various causes are specified below.

Intestinal obstruction, to view it from another stand-point, may be due to (*a*) disease outside of the intestines ; (*b*) to disease of the intestinal walls ; (*c*) to accumulation within the intestine.

The obstruction takes place under the same circumstances as obstruction in other channels.

A. *Diseases Outside of the Intestines.* 1. Pressure of *tumors*, chiefly ovarian tumors, uterine tumors, tumors of the omentum, and pelvic abscess, or abscess about the cæcum. The obstruction may be *acute* or *chronic*. The symptoms of obstruction develop gradually, although in some instances they may take place suddenly, especially if aided by the accidental occurrence of fecal impaction.

2. *Constricting bands*, hernial openings, the remains of fœtal structures, cause constriction of the intestine. In this class of cases there is usually pain, and the history preceding the obstruction is that of peritonitis, general or local, of old hernia, of appendicitis, of pyosalpinx, or of inflammation about the gall-bladder and gall-ducts. The onset may be *acute* or *chronic*. If the constriction is due to protrusion into hernial openings, the onset is usually sudden and without previous symptoms.

3. *Peritonitis* is a most common cause of *acute* intestinal obstruction. It may be due to overdistention by gas and paresis of the bowel, or to pressure by external exudation.

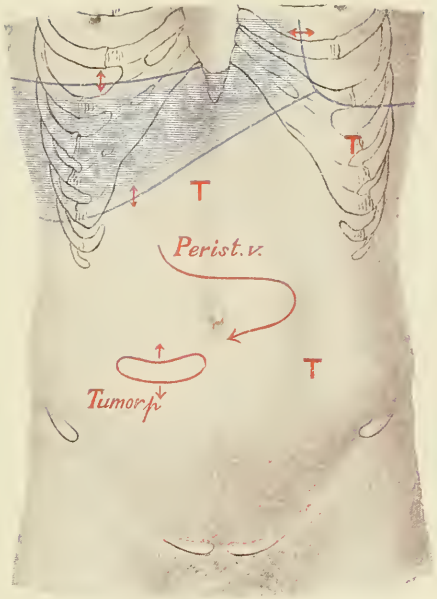
4. Knots and twists of the intestines, usually seated about the sigmoid flexure, causing *volvulus*, are a common cause of *acute* constriction.

B. *Disease of the Intestinal Walls.* 1. Invagination, or intussusception. The attack is *acute*, although the affection may continue over a long period of time.

2. Cancer and other tumors of the intestine generally lead to stricture and *chronic* obstruction.

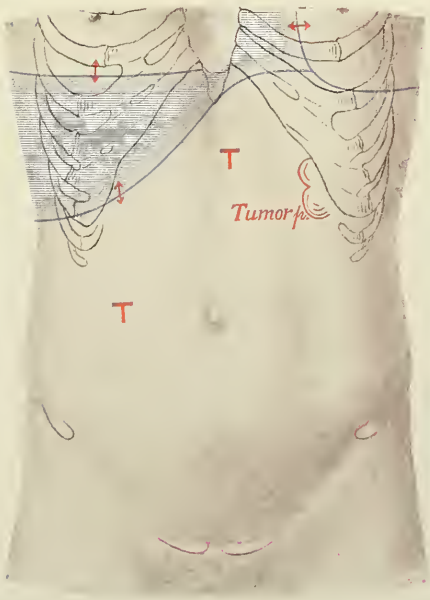
PLATE XXXIX.

FIG. 1.



Invagination of the Ileum.

FIG. 2.



Carcinoma of the Colon.

3. The healing of ulcers, which are syphilitic in the larger number of cases, rarely tuberculous, will lead to stricture. The obstruction belongs to the *chronic* variety. It is seated, in the larger number of instances, in the rectum or sigmoid flexure of the colon.

C. *Accumulations within the Intestines.* 1. Feces. The obstruction takes place gradually, occurs in weak and debilitated people in the course of constipation, especially the constipation of acute disease.

2. Accumulations of improper food or foreign materials. The seeds of fruits or the husks of grain accumulate and cause obstruction. Magnesia, iron, and other articles taken as medicines, from their accumulation lead to obstruction of the intestine. In both of the above mentioned varieties obstruction is *chronic.*

3. Impaction of gallstone within the intestine is followed by acute obstruction.

THE SYMPTOMS. When symptoms of intestinal obstruction occur it is important to ascertain, in addition, first, the duration of the obstruction and its mode of onset; second, the possible cause of the obstruction; third, the seat of the obstruction.

The Symptoms Common to Acute Obstruction. The *symptoms* of intestinal obstruction depend upon the nature and the seat of the obstruction. *Constipation.* The major symptom is stoppage of the intestinal contents. When this takes place suddenly, and there is a local injury to the bowel, the symptoms, both local and general, are severe and alarming. When the constipation is complete there is no escape of flatus. *Pain.* The pain is at the seat of obstruction or about the umbilicus. It occurs suddenly, and is intense and colicky or lancinating in character, radiating from the point of obstruction. There is tenderness over the painful part. The pain is due to the injury by the constricting agent or to violent peristalsis. It may be relieved by pressure. When intermittent, the obstruction is incomplete; when constant, it is absolute. *Tumor.* In many instances a tumor can be outlined due to single loops of intestine, thickened walls, or abnormal contents. This is particularly the case in the obstruction of invagination and the obstruction due to volvulus. *Peristalsis.* The obstruction further causes *increased peristalsis.* This takes place above the point of constriction. Sometimes the movements of the intestine can be seen through the abdominal walls. The extent of the peristalsis is an indication of the site of the obstruction. The higher the obstruction, the less the peristalsis. *Meteorism.* The obstruction causes accumulation of gas above the point, giving rise to meteorism. If the obstruction is low down, the distention and meteorismus are general. If high up, as in the small intestine, on account of constriction by Meckel's diverticulum or internal hernia, the meteorism is in the upper part of the abdomen, and may be limited in extent, or dilatation of the stomach alone may be present. *Vomiting.* Vomiting soon occurs in acute intestinal obstruction, due to decomposition of intestinal contents, to irritation of the stomach by the intestinal contents, to a trauma of the peritoneum at the seat of the obstruction, or, finally, to the occurrence of peritonitis. At first the contents of the stomach are ejected, then watery fluid, bile tinged or largely made up of bile, and later feculent matter. Although

of fecal odor, this is not true stercoraceous vomiting; the latter occurs later in the course of the disease. It must not be forgotten that any obstruction of the intestine may develop with extreme rapidity, so that fecal vomiting may occur within two hours of the commencement of an obstruction. It is recognized by the odor of the matter vomited and by its color. It is a grave symptom, indicating complete obstruction of the intestine. If the obstruction is high up, as in the jejunum, fecal vomiting does not occur. The vomiting, however, is more persistent in high obstruction. *Eructations of gas* are frequent. The *general symptoms* are those of *extreme prostration* or *shock* in its most pronounced form. The *abdominal facies* previously described develops very rapidly. The *tongue* is not changed at first, but soon becomes dry and brown. In a few instances, as in invagination, there may be *fever*, but in other cases usually at once, or very soon in its course, the temperature falls to normal or subnormal, or remains at this point if it has not risen. The extremities are cold, the features pinched, the eyes sunken, the expression anxious. The pain causes the patient to double up in bed. The pulse becomes rapid, weak, and thready in character. The respirations are proportionately hurried, but are also made more rapid and shallow by the tympany. The mind remains clear until the supervention of peritonitis and septicæmia.

The Symptoms Common to Chronic Obstruction. The symptoms are those of *chronic constipation*, with local symptoms due to the cause of the obstruction. The bowels are moved infrequently, and then in small amounts. In obstruction due to stricture from cancer, or cicatricial closure, the feces are ribbon-shaped. Reference must again be made to the occurrence of so-called spurious diarrhœa, with or without the passage of small scybalous masses, on account of impaction of feces. Some credence can be given to the oft-repeated expression of the patients that they have a sense of obstruction in the bowel and that they experience great relief when there is a free evacuation. In chronic obstruction the general symptoms are those of inanition, with the nervous train of symptoms that have been described in constipation; while the local symptoms depend upon the cause. When the local symptoms are due to the pressure of a tumor, or accumulation of pus or fluid within the abdomen, there is a history of local disease, on account of which the tumor developed; such history is obtained in fibroids or ovarian tumor, or in previous inflammation, which was followed by the occurrence of a tumor about the locality of the inflammation, as the pelvis or the appendix.

If the obstruction is due to *stricture* from *cancer* of the intestine, the symptoms of that affection are present. A tumor can be made out at some situation in the course of the bowel. The symptoms are (1) the cachexia, emaciation, and anæmia; (2) pain; (3) tumor; (4) constipation with scybalous discharge; (5) bloody discharge; (6) mucous discharge. If the cancer is seated in the rectum, we find tormina and tenesmus, and the discharge of blood and scybalous masses. Local examination reveals the presence of a malignant mass. Obstruction due to *stricture* from the healing of an *ulcer* is seated in the rectum or sigmoid flexure of the colon. Pain and a sense of obstruction are

referred to that locality. A history of syphilis may be obtained, and frequently the rectal tube, or the finger, will detect the stricture. In both instances there is a history of imperfect, irregular action of the bowels from time to time, with intervals of comparative comfort. These symptoms precede the constipation. When feces accumulate in the colon the larger accumulations take place in the sigmoid flexure and in the cæcum. Fecal tumors, described under Constipation, are felt through the abdominal walls. Obstruction from *fecal accumulation* is preceded by a history of constipation (*q. v.*). The accumulations can be easily discerned as a rule. It must not be forgotten that chronic intestinal obstruction may at any time become acute.

Chronic intestinal obstruction always occurs in *adults*. The *onset* is gradual. The pain that attends obstruction of this form is intermittent, and if there is fecal accumulation, it is not very prominent. *Vomiting* occurs late in the disease, is small in amount, and generally is not a prominent factor. Obstruction to the passage of feces may be constant, or alternate with *diarrhœa*. In fecal accumulation it becomes complete, although spurious diarrhœa may attend it. The discharges may be bloody, which points to cancer. *Tenesmus* is present in stricture low down in the large bowel. *Meteorism* is not marked when the obstruction is high up, as in acute obstruction. When the obstruction is in the large intestine it may be extreme, and in fecal obstruction gradually increases as the obstruction becomes more marked. Coils of intestine in peristaltic movement are seen only in cases in which there is marked emaciation.

The forms of *chronic obstruction* that are attended by *tumor* have been mentioned.

The Differential Diagnosis. It is essential in order to distinguish the form of acute obstruction to ascertain the *nature* of the obstruction, and to determine, if possible, its *site*.

The Nature of the Obstruction. Various factors must be considered in order to estimate the cause of the obstruction.

The Age. Obstruction from intussusception occurs early in life; from bands or through apertures, in adult life, usually prior to forty years of age; from volvulus, between forty and sixty years. Obstruction due to a gallstone occurs during the middle or later period of life — always after the fortieth year.

The Previous History. In obstruction by bands of adhesion there is a history of peritonitis, or, as Treves points out, previous attacks of obstruction more or less marked. In volvulus the patient has been subject to constipation prior to the attack, and in intussusception there has been no previous history, unless polypus was present, causing dragging, colicky pains, and occasional discharge of blood.

The Symptoms. The symptoms of the various forms of acute obstruction vary somewhat. *Pain* in strangulation, from bands or hernia, is severe and paroxysmal in character, attended by collapse. It occurs early in volvulus, though it is not so severe as in the former, and occurs at long intervals, becoming constant with exacerbations. In acute intussusception the pain occurs early, and is steady. It increases, and then may suddenly subside. At first it is paroxysmal,

attending discharge of blood and mucus from the bowels. *Local tenderness* in the first group of cases occurs late. In volvulus it occurs early, and may be noted over distended coils. In intussusception it is usually common about a sausage-shaped tumor. *Vomiting* is marked and occurs in strangulation, soon becomes feculent, and increases the severity of the paroxysms of pain. In jejunal obstruction it is excessive and non-feculent. In volvulus it does not come on so quickly, but is severe and constant when it takes place. The relaxation that attends vomiting often affords relief to the obstruction. In intussusception it does not occur as early as in the other forms, and is not so severe. It becomes feculent in only a small number of cases. *Constipation* is continuous in all cases except intussusception. In the latter there is some constipation, but it is not absolute; diarrhœa is not uncommon, and discharge of blood in the stools occurs in 80 per cent. of the cases, according to Treves. *Prostration* is severe in all cases, although probably not so marked in volvulus. Because of its close proximity to the rectum *tenesmus* occurs in volvulus. It is of frequent occurrence in intussusception, often beginning early in the attack.

The Physical Signs. (Plate XXXIX., Figs. 1 and 2.) On palpation of the abdominal wall it is noted to be soft and flaccid in most of the cases, unless peritonitis has ensued. This occurs early in volvulus, hence rigidity is marked. In a large number of cases a *tumor* can be made out only in intussusception. It is seated in the lower right quadrant of the abdomen. Early in the attack it is oblong and of sausage-shape. When peritonitis ensues it disappears, on account of the tympany. A portion of the gut may protrude at the anus, or be felt on rectal examination. *Meteorism* occurs about the third day in a strangulation; it occurs early, is very rapid and pronounced in volvulus, and is absent in intussusception, unless constipation or peritonitis takes place. It is not marked in high obstruction.

The Site of the Obstruction. The seat of obstruction is in a measure indicated by (1) the location of the pain or abnormal sensations, (2) the character of the swelling, (3) the character of the stools, (4) the degree of meteorism, (5) the results of a rectal examination, (6) the change in the urine, (7) the general condition. The patient is often able to indicate the location of the obstruction fairly well by the sensations of obstruction or fulness and by the great relief experienced when a free evacuation of the bowels is naturally or artificially produced. On auscultation, when the bowel is irrigated, a *murmur*, like the deglutition-murmur, may be heard at the point of constriction of the gut. In obstruction high up there is but little *meteorism*, the tumor is usually not detected, and pain is seated about the umbilicus or the upper quadrants of the abdomen. Obstruction at the ileo-cæcal valve may be indicated by a tumor in the lower right quadrant over the region of the valve or just above it. It is usually at this point that invagination takes place, and hence we may look for a tumor in this situation. (Plate XXXIX., Fig. 1.) On the other hand, in volvulus of the colon, or stricture of the rectum, the obstruction, being low down, is attended by much meteorism and by pain in the left lower quadrant of the abdomen. A tumor may be detected in this

position. The position of the obstruction is sometimes indicated by the seat of *peristalsis*. This may be seen to stop at a given point, which usually indicates the position of the obstruction. The seat of obstruction may be indicated by the number of coils of intestine that are engaged in the peristaltic movement. The coils of intestine in front of the tumor are dilated and hypertrophied. In active movement they cause prominences which follow the course of the bowel. Wyllie has called them "patterns of abdominal tumidity." If the obstruction is in the jejunum, peristalsis may not be observed. If the lower end of the large intestine is obstructed, the colon is prominent; if the gut about the ileo-cæcal valve, the region about and below the umbilicus is prominent. *The Urine.* The position of the tumor, it is said, can be ascertained by changes in the urine. When the obstruction is in the small intestine, indican is much increased from the decomposition of albuminous substances and products of putrefaction. In this location the urine may be suppressed. In stenosis of the large intestine indican is not increased unless there is cancer. The value of the information derived from the character of the stools and the results of rectal examination are obvious. Obstruction in the duodenum or jejunum is followed by rapid collapse and anuria. In general, it may be said the more severe and rapid the symptoms the more likelihood that the obstruction is in the small intestine.

Intussusception (Plate XXXIX., Fig. 1), or invagination, occurs most frequently in children prior to the tenth year. It is characterized by severe colic and pain in the abdomen, first complained of about the navel. The severity increases in paroxysms, and only lessens if complete strangulation has taken place. With the onset of the pain there are one or two movements of the bowels, which contain mucus and blood. After this there may be constipation, or the stools continue to be loose, and are as frequent as fifteen or twenty in a day. Sometimes they are quite bloody, and almost always there is some tenesmus. In a short time after the attack vomiting commences. It may be constant or occur only after taking food. At first the abdomen is soft, but tender on pressure. A sausage-like tumor can be felt on the right side below the transverse umbilical line. On inspection of the rectum a portion of the intestine may be seen, dark and gangrenous in appearance, or it may be felt by palpation. If there is much tenesmus, the anus often remains open. In rare cases the bowel may slip back and the symptoms subside spontaneously. On the other hand, peritonitis may rapidly ensue, with high fever, followed by collapse and death.

Diagnosis. It must be distinguished from the *entero-colitis* of childhood or the proctitis due to a polypus. In entero-colitis there is no tumor, and the collapse and prostration do not occur so early and are not so rapid. There is greater likelihood of a number of the stools being greenish, like spinach. In a polypus of the rectum the symptoms are local. The child is worn out and restless, but great abdominal tenderness, and the tumor, meteorism, vomiting, and collapse are absent. The rectum must be examined.

Intussusception must be distinguished from *peritonitis*, in which symptoms of stenosis of the bowel from ileus paralytica may be present.

The history and sequence of events must be watched carefully. Often the commencement of the affection about hollow viscera which have previously been the seat of disease, or its onset with sudden perforation, will point to the nature of the affection. In *peritonitis* there is no active peristalsis; there is general distention of the abdomen, with general tenderness; the urine is diminished, but does not contain indican in excess. Collapse ensues rapidly. Signs of effusion within the abdomen may appear.

It must be distinguished from *embolism* or *thrombosis* of the *mesenteric artery* and *infarction* of the bowel. In the latter the symptoms take place suddenly. The patients have reached middle or late life, and have atheroma of the general arterial system. Sudden pain in the abdomen, with vomiting and symptoms of collapse, takes place. Moderate obstruction occurs, with distention of the abdomen. After the pain diarrhœa with the passage of blood follows. The age and the absence of tumor distinguish it from intussusception, the only intestinal condition for which it may be mistaken.

Hernia and Constriction by Bands. Obstruction due to these conditions occurs in adults after the fortieth year of age, in both sexes. In stricture from pressure of bands there has usually been a history of previous attacks of peritonitis or of inflammation of the structures in relation to the peritoneum. Hence, a cholecystitis or appendicitis are often found to precede the obstruction. The attacks begin suddenly, and the symptoms may from the start be most pronounced. They are the typical symptoms of intestinal obstruction. The local tenderness, however, may not be present as early as in other forms of obstruction. It is quite characteristic not to find a tumor or positive local cause for the obstruction, and also not to have meteorismus. This is due to the fact that the obstruction is usually high up in the intestinal tract.

Volvulus. Volvulus occurs most frequently in males. It occurs late in life, and is usually preceded by a history of constipation. Premonitory symptoms may have been present for a few days, but the symptoms of obstruction develop suddenly. They are the symptoms of acute obstruction, but as the lesion is in the lower portion of the bowel, meteorismus is present to a marked degree, and rectal symptoms are found. Tenesmus is present in a small proportion of the cases. Peritonitis is likely to set in early, with increase in the temperature, increased tenderness of the abdomen, and more pronounced symptoms of collapse.

Diagnosis of Intestinal Obstruction from Other Conditions. Intestinal obstruction must be distinguished from peritonitis and appendicitis. This is sometimes very difficult. Careful attention must be paid to the evolution of the case and the history of previous abdominal disease, or of lesions on account of which, on the one hand, peritonitis may occur, or, on the other, obstruction of the bowel. In peritonitis the attack follows disease in the uterine appendages, the vermiform appendix, or the gall-bladder, or perforation in some portion of the gastro-intestinal tract. *Fever* usually attends the inflammation, with or without chill. *Vomiting* will probably occur at the onset, and then subside until the

peritonitis becomes general. The first paroxysms of vomiting are apparently due to shock. The vomiting that occurs rarely becomes feculent. As the peritonitis advances the vomiting becomes passive; a simple constant regurgitation of a large amount of fluid, greenish or grayish-yellow, or watery, takes place. It pours into the mouth, and is simply discharged without the occurrence of retching. The abdomen is swollen and *tympanitic*. The symptoms due to excessive tympany are more marked than in intestinal obstruction. As the diaphragm is interfered with, breathing is hurried. The abdomen is tender on pressure and is the seat of general *pain*. The general pain and tenderness, however, can usually be found to be more marked at the possible primary focus of the disease. Further, on local examination, in these positions fulness or undue prominence or *swelling* may be observed. *On palpation* over the point of origin there may be localized *œdema*. The symptoms of collapse do not differ from those of intestinal obstruction in marked degree, although the peculiar appearance of the face and other nervous features occur more rapidly in peritonitis than in obstruction. It must be remembered that peritonitis in a large majority of cases attends obstruction.

In *appendicitis* the symptoms are somewhat like those of intestinal obstruction. There may be constipation and vomiting. The former is not pronounced, and can usually be relieved. Vomiting subsides after the first twenty-four hours, unless peritonitis supervenes; it is never stercoraceous. The local physical signs are characteristic. In appendicitis there is fixed tenderness on pressure at McBurney's point. Some swelling can almost always be observed. On light or deep percussion there is change in the note as compared with the other side. Fluctuation can often be detected in from two to four or five days. Both the tumor and fluctuation can be detected by bimanual examination of the abdomen and flank. Examination by the rectum may reveal a tumor at the brim of the pelvis in the right side. Fever attends the attack throughout. When peritonitis supervenes there is rigidity of the entire abdomen, which at first was localized to the right lower quadrant.

Intestinal obstruction must not be confounded with *enteritis*. In all forms there is diarrhœa, in many vomiting. Pain of a colicky nature, spreading from the neighborhood of the umbilicus, is marked whenever obstruction to the passage of feces or gas takes place. Vomiting is not stercoraceous, and the general symptoms, collapse, etc., do not occur. Acute *hemorrhagic pancreatitis* is also attended by symptoms similar to those of intestinal obstruction. There is sudden severe pain in the upper half of the abdomen, with vomiting and the rapid development of collapse; there may be constipation; the situation of the pain is of some significance. Vomiting never becomes stercoraceous; flatus can usually be passed and the bowels opened by an enema. Meteorismus does not take place, although the epigastrium is tympanitic. If the symptoms are not so severe, there may be increased dulness, and possibly a tumor on deep palpation in the left upper quadrant of the abdomen along the margins of the ribs, which should be dull on percussion, or, on account of its relation to the stomach, give a dull tympanitic note.

The symptoms of internal hemorrhage are present, pallor of the face and extremities, syncope, and, in addition, prostration and other symptoms of collapse.

Cancer of the Intestines. (Plate XXXIX., Fig. 2.) *Obstruction* must not be confounded with carcinoma of the intestines. The disease usually occurs late in life, and is associated with progressive *emaciation* and *cachexia*. There may not be any symptoms save general failure of health until the sudden occurrence of obstruction of the bowel. The symptoms vary with the position of the carcinoma and the direction of growth of the tumor. In some instances with the general symptoms there may be irregular *pain* in the abdomen, with irregularity of stools. The *tumor* may be detected if the small intestine is involved Its detection is facilitated by having the patient get on the hands and knees and palpating the abdomen in this position, and by clearing out the colon by a large enema. On auscultation the water may be heard to enter the dilated colon beyond the tumor, the sound resembling the deglutition-murmur at the cardiac end of the stomach. If the tumor is situated in the lower colon, pain in the sacral region, resembling sciatica, may be complained of; if the cæcum or the sigmoid flexure is the seat of disease, a tumor is usually detected. Wherever the situation, the tumor found is tender, usually lying in the axis of the intestine—movable if in the small intestine, fixed if in the cæcum or the sigmoid flexure. In the latter location the tumor may be felt per rectum. One notable characteristic is that it may be palpable some days and not be present at other times. The position and size may vary from day to day, although it is always hard and knotty, not doughy. By means of the proctoscope, with the patient in the knee-chest position, as described by Kelly, the presence of tumors of the descending colon will be disclosed. *Constipation* is characteristic of most of the cases. It may alternate with diarrhœa. Paralysis of the sphincter ani may take place, with incontinence. The stools are frequently ribbon-shaped, or they may pass in scybalous masses, and large or oftener small amounts of blood, chiefly the latter, are passed with pus or mucus; sometimes masses resembling cancer can be found in the stools. If the tumor is in the rectum, there is great difficulty in defecation; the act is attended by pain. Later the pain becomes constant, and may radiate to the hip or the genitalia. Sometimes this pain is the only symptom complained of.

The diagnostic symptoms are: (1) The general symptoms of cancer. (2) The tumor. (3) The occurrence of constipation which leads to complete obstruction, or obstipation, alternating with diarrhœa. Blood in the stools, with alteration in the shape of the feces, is significant.[1]

Diseases of the Rectum.

Consideration of rectal lesions belongs to the surgeon. It is proper, however, to insist upon the very frequent deleterious effect of such lesions in neurasthenic subjects. Indeed, the bleeding which attends

[1] Musser: "Carcinoma of the Descending Colon." Univ. Med. Mag., 1896.

hemorrhoids may be sufficient to lead to profound anæmia, upon which neurasthenia may readily develop. The local suffering due to rectal fissure, or prolapse, may aggravate any tendency to the state of neurasthenia, or aid materially, with other conditions, to fasten it more firmly upon the system. In cases of anæmia, of neurasthenia, of the gastric neuroses, of debility, or prostration, the cause of which cannot be ascertained, the rectum should be examined. The appearance of hemorrhoids and other rectal affections is described in works on surgery. Hemorrhoids, ulcers, fistula, and carcinoma are to be sought for in abdominal affections.

Inspection and palpation are necessary. The symptoms are those of local pain, tenesmus, and frequently hemorrhage. The pain follows a movement of the bowels. There may be a feeling as of a foreign body in the rectum, with some itching and burning about the anus. The pain may be so severe as to inhibit defecation. The timid subjects will not endure the act; in consequence they suffer from vertigo, headache, tympanites, and symptoms of gastro-intestinal disorder. In some instances there is chronic catarrh of the rectum, with discharge of small stools containing mucus or pus streaked with blood. Cases occur in which hemorrhage is the only symptom, the constant recurrence of which leads to grave constitutional results. Hemorrhoids are the lesions for which the rectum is most frequently examined. They, as well as other lesions, are of diagnostic significance in affections beyond the rectum. Thus in all forms of portal congestion internal hemorrhoids are of constant occurrence, and when found in a toper may be one of the first indications of cirrhosis of the liver. Rectal fissure is not of much diagnostic significance. The finding of a small cancer, the symptoms of which may be those of hemorrhoids, may explain emaciation and the development of cachexia. Ulcer of the rectum may be due to syphilis, cancer, or tuberculosis. A fistula is often tuberculous. The rectum must be examined in cases of pyæmia, particularly of the portal variety, when jaundice, enlargement of the liver, and hectic fever are present, for local rectal disease may cause pylephlebitis.

CHAPTER VI.

DISEASES OF THE LIVER, SPLEEN, AND PANCREAS.

The symptoms of disease of the liver are due to the morbid processes, to disturbance of the functions of the hepatic cells, or to obstruction of the channels for the flow of blood and of bile. As these channels extend beyond the glandular structure of the liver they may be affected by disease outside of the organ. Hepatic symptoms may, therefore, be due to diseases other than those of the liver.

The morbid process may, in time, cause alterations in function, obstruction of channels, or physical alterations in the size and shape of the liver. But the channels may be obstructed and the size and shape of the liver changed by disease outside of the liver.

Symptoms due to the Morbid Process. The *morbid processes* are the congestions, the inflammations, the degenerations, the morbid growths, and gross parasites.

In *congestion* of the liver the symptoms are (1) the symptoms of the cause, (2) enlargement of the organ from the increased amount of blood, (3) functional disturbance from the same cause. The congestion is not limited to the vessels in relation with the liver-cells, but involves the vessels of the mucous membrane also, hence the latter swell, obstruct the ducts, and produce jaundice in moderate degree. The *inflammations* are toxic and infectious. The symptoms are due to the cause (intoxication or infection), to the degree of obstruction of the vessels and ducts, to the shape and size of the liver, and to the alteration of its function. When the inflammation is diffused, as in the cirrhoses, the hepatic symptoms are more marked ; when local, as in abscess, the infectious symptoms are in preponderance. If the ducts are the seat of infection, the bile channels are obstructed—jaundice arising ; if the vessels, ascites. In *morbid growths* of the liver the symptoms are those of malignant disease in general, to which are added symptoms due to change in the size of the liver, and, more frequently than in inflammation, symptoms due to obstruction of the channels. The *degenerations* are so frequently secondary to and masked by the symptoms of their primary cause that, save in regard to change of size, there are no hepatic symptoms worth mentioning.

Symptoms due to Functional Disturbance of the Liver. The functions of the liver are to secrete bile ; to destroy the hæmoglobin of the blood ; to destroy, modify or neutralize poisons entering, or to modify and render available for nutrition the peptones absorbed by, the portal circulation ; the elaboration of glycogen. Bile is not secreted when the liver-cells are destroyed, as in acute yellow atrophy. The liver does not destroy the usual amount of hæmoglobin. On the other hand, hæmoglobin may be so much in excess that the liver cannot

destroy it; jaundice then results. (See Hæmatogenous Jaundice.) Functional disturbances of the liver are manifested clinically by symptoms due to the entrance into the circulation of imperfect products of digestion, or poisons not destroyed by the liver.

Lithæmia is a common toxic condition, and is believed to be due to functional liver-disturbance. There is an excess of uric acid and urates, or of other metabolic compounds in the blood. It may be a convenient term for the *auto-intoxication* which takes place in disease of the gastro-intestinal tract. The *symptoms* are, first, symptoms of excess of lithic acid in the system; second, the effects of the lithic acid upon the nervous system. Lithæmia may be acute or chronic.

Acute Lithæmia; Biliousness. When acute the local disturbances are: furred tongue, a bitter taste in the mouth, anorexia, nausea, disgust at the sight of food, with possible morning vomiting. There is some tenderness in the upper mid-abdomen, and, after eating, weight and fulness and distress in that region. Flatulency occurs. Symptoms of intestinal dyspepsia may arise secondarily. Slight fever or feverishness may attend the attack. The skin is hot and burning; or cold perspirations may break out at irregular times, alternating with flashes of heat. The bowels are constipated, the stools are clay-colored. The symptoms may be attended by slight obstruction to the ducts, causing a moderate degree of jaundice. In some instances the liver is slightly enlarged. The urine is loaded with urates and uric acid. It is scanty and high-colored, and there may be painful micturition. The *nervous symptoms* are usually those of depression, as headache, some dulness, or stupor; the patient may be unusually drowsy. The headaches may be the most prominent feature of the attack. They are frontal, attended by slight vertigo, flashes of light or spots before the eyes, and ringing in the ears.

The same group of symptoms is seen in *acute gastro-duodenal catarrh*.

Chronic Lithæmia. In *chronic lithæmia* the symptoms are variable, and are characterized by disturbance of function in nearly all the organs of the body. They have been classically described by Murchison, Da Costa, and others, and while the theory is fairly satisfactory to work upon for lines of treatment, the same group of symptoms may be met with in forms of chronic indigestion, particularly the forms in which there is inability to digest sugars and starches. The symptoms are attributed by some to *chronic intestinal catarrh*.

Symptoms. The patients are in ill health and subject to chronic indigestion. They may be under weight or corpulent. The skin is harsh and dry, its nutrition poor. It is subject to erythema; or local inflammations, as eczema, may arise. Irregular sweats occur, alternating with intervals when the skin is hot and dry. The extremities are cold and clammy, and tingling and numbness are often complained of.

Gastro-intestinal Symptoms. The symptoms are those of chronic indigestion. There is constantly a furred tongue with local dyspeptic symptoms. The bowels are irregular or constipated; sometimes mucus is passed. Flatulency is excessive, both gastric and intestinal. An icteric tinge may be seen on account of a slight local catarrh of the

ducts, or of hepatic congestion. It recurs at frequent periods, while a sallow complexion is more or less constant.

Respiratory Symptoms. The patient is liable to attacks of catarrh of the upper air-passages, and especially to pharyngitis. In lithæmic states tonsillitis is not uncommon. Chronic pharyngitis is present. On the other hand, some persons, particularly those over fifty years, have chronic bronchitis, and attacks of asthma are common. The bronchitis cannot be distinguished from that due to other causes, except by the fact that the subject is lithæmic. Emphysema of the lungs develops on account of bronchitis and tissue degeneration.

Cardiac Symptoms. Palpitation is a constant accompaniment of many forms of lithæmia ; in others there may be unduly rapid action of the heart, or, during exacerbations, slowness of the heart's action. In the later stages pseudo-angina pectoris is of common occurrence. In the earlier stages pain about the heart or in the left side is frequently complained of.

Nervous Symptoms. Constant headache, worse in the morning, relieved toward the end of the day. Some vertigo may be present. Depression of spirits and inaptitude for mental exertion exist. The memory is dull, the faculties blunted. The patient is subject to backache, chiefly in the loins. Pain in the right shoulder is of frequent occurrence. In addition, pains along the course of the nerves (neuritis), and myalgias, are of common occurrence. The nerve-trunks may be tender. There is tenderness in the sheaths of the muscles, or at the insertions of fasciæ and tendons. Peripheral nerve-sensations are common. Numbness and tingling are frequently complained of. Paræsthesiæ of all forms, variously distributed, are a source of annoyance. Local sensations of heat or burning alternate with areas of coldness. Tingling, pricking of needles, and other forms of paræsthesia occur.

The Urine. The urine is high-colored and contains an abundance of uric acid and urates. The amount is scanty, the specific gravity high. There may be albumin, small in amount, depending upon the irritation of the urates in their passage through the kidneys. Cylindroids are present ; casts are not common, although at times, when the uric acid is passed in excess, there may be a secondary nephritis, with albumin, blood, and casts. As an ultimate result of such condition we may have gallstones, or calculi in the kidneys and bladder. Lithæmic patients are subject to attacks of hepatic or renal colic.

As part of the same process or an accompaniment we may have *gout* or *rheumatism*. Acute inflammatory rheumatism (rheumatic fever) does not belong to this category, but muscular rheumatism, subacute inflammation of the joints with moderate fever, true gout, and gout with its modifications when seated in the various joints, are the ultimate results of this process in the patient. Attacks of gout may occur in a patient who has not shown any symptoms of lithæmia, but those who have symptoms of lithæmia are more susceptible to causes which produce attacks of gout. The gouty and rheumatic manifestations are due to the deposition of uric acid and urates in tissues which are not highly vitalized, and in which, therefore, the circulation is sluggish.

Lithæmia later assumes the *gouty aspect.* Tophi are seen in the situations natural to them. The appearance of the face is characteristic, with capillary congestions and stases. The patients usually become more or less obese and are subject to attacks of glycosuria. Early in their life degenerations of vessels take place. The kidneys are always under an excessive strain. A good deal of material is not discharged; its effects upon peripheral vessels are such as to cause vasomotor spasm and heightened tension, leading to low-grade inflammations, with the development of atheroma. For the same reason chronic interstitial nephritis is set up, and, because of heightened strain in the vascular system, chronic sclerotic valvulitis.

Functional symptoms from disorder of the liver are otherwise not marked, unless we include a group of cases in which sudden coma and convulsions take place, presumably because material has been absorbed from the gastro-intestinal tract and enters the general circulation through the temporary cessation of the function of the liver, the office of which is to destroy the material. Such symptoms may arise in organic disease of the liver, as cirrhosis.

Symptoms due to Obstruction of the Channels. (1) Obstruction of the *bile-ducts*, either from disease or external pressure, causes jaundice, pain, and fever. The three symptoms may occur singly or combined. Jaundice may occur alone in obstruction by *gallstones;* pain may occur with it; or jaundice, pain, and fever may occur together; rarely, pain or fever may be present alone. Each symptom will be described later. (2) Obstruction of the *blood-channels* causes *congestion* of the liver, which may be active or passive, or *portal obstruction.* The symptoms of each will be discussed; suffice it to say that here again the symptoms are modified by the process. Thus in portal obstruction from pressure the symptoms are quite different from those in portal obstruction due to suppurative inflammation of the vein.

Congestion of the Liver. In the *congestions* the liver is enlarged. If the hyperæmia is active, painful distention may be complained of, and the organ may be the seat of some tenderness. There may be, in addition, weight and fulness in the liver-region. Active hyperæmia may follow a chill or suppression of the menses, but more frequently occurs after indiscretions of diet, the free use of alcohol, or stimulating food, followed by an attack of acute gastro-intestinal catarrh. It is more common in the tropics, and is due in that climate to suppression of the perspiration. It is recognized by the occurrence of symptoms of acute gastritis with enlargement, pain, and tenderness of the liver. Slight jaundice may attend the attack.

Passive congestion is also attended by enlargement of the liver. The enlargement may cause a sense of weight or fulness, but pain is not complained of. The organ is not tender, the edges are smooth and indurated. The liver may pulsate. This is detected by placing the hand over the surface of the liver, when, with each impulse of the heart, the organ can be felt to expand. The symptoms of the cause of the passive congestion combine with those just enumerated as due to enlargement of the organ. In addition we have symptoms due to obstruction of the flow of blood in the portal circuit.

Passive congestion occurs in organic heart disease after compensation has failed and the right heart is dilated. The organ rapidly becomes congested because of its close proximity to this chamber. In emphysema of the lungs, in fibroid phthisis, in intrathoracic tumors pressing upon the vena cava, mechanical congestion also takes place. The recognition of passive congestion is not difficult. The symptoms due to enlargement (see Objective Symptoms) and the symptoms due to portal obstruction point to the true nature of the morbid process.

Portal Obstruction. Disease of the portal vein or occlusion of its branches in the liver, obstructs the flow of blood. The diseases of the portal vein are *thrombosis,* and *adhesive* and *suppurative inflammation.* Obstruction of the terminal venous radicles in the liver is caused by cirrhosis.

Thrombosis of the portal vein attends cirrhosis of the liver, or may occur secondarily to pressure upon the vein by a tumor. Disease of the pancreas was the cause of the pressure in a patient under my observation. As a result of thrombosis *adhesive inflammation* of the vein takes place, with or without the establishment of a collateral circulation to replace its function.

The symptoms of *disease of the trunk* of the portal vein are the same as those of obstruction of the terminal branches, and are known as the symptoms of portal congestion. (See below.) In one respect only do they differ. While we have *ascites* in both, in thrombosis of the portal vein it occurs suddenly, and is characterized by rapid recurrence after tapping.

Suppurative inflammation of the portal vein is attended by symptoms resembling pyæmia, and is also called *portal pyæmia.* The inflammation is secondary, and depends upon inflammation in the portal area. It may follow appendicitis, infectious inflammation of the hemorrhoidal veins, or of the veins anywhere in the gastro-intestinal tract. Pus is carried into the liver by the portal current. In consequence thereof, multiple hepatic abscesses arise. Three pathological affections are therefore seen : (1) Suppuration in the portal area ; (2) inflammation of the vein ; (3) multiple abscesses of the liver (for the symptoms of which see Abscess).

Occlusion or overfilling of the branches in the liver occurs in passive congestion, and most typically in cirrhosis of the liver. The circulation in the liver is interfered with ; the blood is thrown back into the portal vein, and overfills the vessels of the portal area. As a result we have (1) congestion of the mucous membrane of the stomach and bowels, with the symptoms of gastro-intestinal catarrh. (2) Dilatation of the veins, chiefly the hemorrhoidal, giving rise to hemorrhoids. (3) Ascites. (4) Hemorrhages. The *hemorrhages* may occur in any part of the gastro-intestinal tract. Hæmatemesis and intestinal hemorrhage are seen singly or combined. The vomited blood may be small in amount, often with mucus. In some cases large, sometimes fatal, hemorrhages take place either from the mucous membrane of the stomach or from the veins about the œsophagus, which often become varicosed in cirrhosis. Hemorrhages from the intestine may be from enlarged hemorrhoidal veins, from an intestinal ulcer, or from the intact mucous

membrane. (5) Enlargement of the spleen. (6) Changes due to the collateral circulation. If complete collateral circulation is established, the above symptoms may not ensue. The *collateral circulation* may be through deep-seated or through superficial veins. If the latter, the *external veins* of the abdomen are enlarged. The epigastric and mammary veins become prominent. The veins about the umbilicus may become so enlarged and prominent as to form a swelling, to which the term *caput Medusæ* has been applied. The venules along the line of attachment of the diaphragm in the lower thoracic zone are overdistended. They may be the seat of pulsation.[1]

In consequence of the portal overfilling the enlarged terminal branches of the vein press upon contiguous structures, interfere with the circulation of blood in the major vascular system of the liver, and invite catarrh of the terminal ducts, with obstruction, and hence *jaundice*. This is seen quite frequently in passive congestion of the liver, rarely in cirrhosis.

Symptoms due to the Changes in Shape and Size. The liver may be enlarged, contracted, or irregular. (See Objective Symptoms.) When the liver is contracted symptoms of portal obstruction usually occur; when enlarged they occur occasionally.

The Data Obtained by Inquiry.

A knowledge of etiological factors is of aid in the diagnosis of hepatic affections. In disease of the liver more than in any other organ of the body we find the affection secondary to disease elsewhere. Moreover, diseases of the liver are almost always associated with definite causes, the presence or absence of which is of great diagnostic significance. In the study of hepatic disease we consider, therefore, among etiological factors, the age, the sex, the habits of life, the climate, and the presence or absence of disease in other portions of the body. Primary liver disease is comparatively rare. Secondary liver disease, on the other hand, is of common occurrence. There are but few general diseases or states of the system that do not in some way influence the liver. The above remarks refer to organic disease. Functional disorders of the liver, as previously remarked, are so difficult to separate from functional disorders of the stomach and intestines, that, practically, from an etiological and clinical stand-point, they go hand-in-hand

The Social History. *The Age.* Diseases of the liver usually occur late in life, because the causes upon which they depend are operative only at that period. In a case, therefore, of ill health in a young subject, when the cause cannot well be determined, the liver is not so likely to be the seat of disease as in older subjects. Late in life we have gallstones with their multiple consequences, inflammation, cirrhosis, and cancer. We may, however, have the congestions and the degenerations in early life, although not so frequently.

The Sex. The sex is not of much significance from a diagnostic stand-point. Cancer may be more frequent in the female sex, because

[1] Musser: Trans. Phil. Path. Soc., vol. xi. p. 20.

cancer of the uterus and other organs is more common. Cancer of the biliary passages is more frequent in females, because in that sex gallstones, which are etiological factors in cancer, are more common. Cirrhosis, also, is said to be relatively more frequent in females.

The Habits. It is always necessary to inquire into the habits. Alcoholism points to cirrhosis; the excessive use of stimulating foods to hyperæmia; sedentary habits and the use of starches and fats to gallstones. The occupation has but little influence in the development of hepatic disease. With regard to the climate, it may be said that in tropical countries hyperæmias and abscess of the liver are more frequent.

The Family History. But little avails in the study of the family history for diagnosis, as most of the morbid processes are secondary to disease elsewhere. This does not apply to biliary calculi, the formation of which appears to be confined to members of special families.

Previous Disease. It is absolutely essential to inquire into this to establish a diagnosis, as liver disease is usually secondary. The occurrence of heart disease or obstructive lung disease points to a congestion; infectious diseases to cirrhosis when that is not otherwise accounted for; dysentery to abscess; ulceration or suppuration in the portal area to multiple abscess; syphilis to syphilitic diseases; tuberculosis, suppurations, bone disease, and syphilis to amyloid disease; pyæmia to multiple abscesses; tuberculosis to fatty liver.

The Subjective Symptoms.

The subjective symptoms are such as belong to functional disorder of the liver, conspicuous among which are gastro-intestinal symptoms and toxæmia. (See Functional Disturbance and Lithæmia.)

Pain is a frequent symptom of liver disease. When sudden in onset, acute, and increased by pressure or movement, it is due to *perihepatitis.* Acute paroxysmal pain below the ribs or in the epigastrium points to *gallstones.* It may be in the seventh or eighth interspace. Pain with distention occurs in *congestion.* Stabbing or darting pains belong to *cancer.* The pain of perihepatitis may attend *abscess.*

Pain in the liver must not be confounded with pleurisy. In pneumonia there is often congestion of the liver and perhaps perihepatitis. The associated pain has been mistaken for the pain of hepatic colic.

The Data Obtained by Observation. The Objective Symptoms.

Topographical Anatomy. (See Plates XIII., XIV., and XXXV.) The right lobe of the liver is applied to the concavity formed by the lower lobe of the right lung, being separated from it by the diaphragm. The thin lower edge of the right lung overlaps the liver at its upper part, but the greater portion of the anterior surface of the right lobe of the liver is in contact with the ribs. The under surface of the liver is in relation with the stomach, transverse colon, duodenum, right kidney, and right suprarenal capsule. "The highest part of its con-

vexity on the right side is about one inch below the nipple, or nearly on a level with the external and inferior angle of the pectoralis major. Posteriorly the liver comes to the surface below the base of the right lung, about the level of the tenth dorsal spine." (Holden.)

A needle thrust into the right side, between the sixth and seventh ribs, would traverse the lung, and then go through the diaphragm at its central attachment, into the liver. The lower border of the liver extends in the median line, one-third of the distance from the tip of the xiphoid cartilage to the umbilicus. In the right mammary line it extends to the lower border of the ribs; and in the mid-axillary line to the tenth rib. The upper border is opposite the upper border of the sixth rib in the mammary line, and extends horizontally in the axilla to the ninth rib behind.

The attachments of the liver permit of a certain amount of movement. Hence, the liver can be depressed by deep inspiration, emphysema of the lungs, or right pleural effusion. If the patient lie upon his left side, the left lobe of the liver rises higher and the right extends lower, and *vice versa* if the patient lie upon the right side, the liver turning upon the suspensory ligament as an axis. (Gerhardt.)

Inspection. Inspection is not of very great assistance in the diagnosis of diseases of the liver. Frequently there is a *swelling* or *tumor* in the right upper quadrant, which may or may not be produced by an enlargement of the liver, but which should direct attention to that organ. The lower right zone of the thorax may also be distinctly prominent. Such a swelling may be observed in amyloid disease, hydatid tumor, cancer, abscess, and, less frequently, in fatty liver. In amyloid and fatty livers the projection in the right upper quadrant, which may extend to the left beyond the median line, presents a smooth surface, whereas in *hydatid tumor* there is frequently a rounded projection at some part of the prominent area, and, in *cancer*, several nodules may be large enough to cause slight rounded projections, which the eye is more apt to detect after the sense of touch has first directed attention to their presence.

Enlargement and occasionally pulsation of the superficial abdominal *veins* are accompaniments of cirrhosis.

Jaundice. *The Symptoms.* The color of the skin and of the mucous membranes in jaundice has been described. (See page 121.) In addition to the yellow discoloration we find: 1. *Irritations of the skin.* Pruritus is common and intense, and may cause great distress. An attack of jaundice may be preceded by general itching. It occurs in all forms, but is more marked in obstructive jaundice of long duration. Scratch-marks are seen on the surface of the skin, and erythematous eruptions and boils frequently occur. *Xanthelasma* is a peculiar affection occurring on the tongue, on the skin of the eyelids, and about the ears. (See page 92.) 2. *Discoloration of the secretions.* All the secretions of the body are changed in color, as previously described. 3. *Bile absent in the feces.* The stools are ashy or gray in color. 4. *Slowness of the pulse.* The heart's action falls to 40 or 30 to the minute, or even lower. 5. *Hemorrhages.* In the later

stages of all forms of jaundice hemorrhages are of common occurrence. In acute malignant jaundice they are seen underneath the skin, and come from the mucous membranes. 6. *Cerebral symptoms.* Irritability and depression of spirits are marked. As the disease advances the mind grows sluggish ; the patient is dull, and sleeping most of the time. Gradually the symptoms of the typhoid state develop. In the acute febrile forms coma and convulsions are of common occurrence. In the affection known as *acute yellow atrophy* the cerebral symptoms are marked, and occur soon after the onset of the disease. Within the first twenty-four hours there may be convulsions, with delirium in the intervals, and subsequently coma.

Causes. Jaundice is of two varieties, the hepatogenous and the hæmatogenous.

Hepatogenous Jaundice. Jaundice is *hepatogenous* when there is obstruction of the ducts. The obstruction may take place in the large ducts or in the smaller terminal ducts. The obstruction may be due to disease outside of the ducts ; to disease of the ducts, or to obstruction within the ducts.

1. Jaundice from disease outside of the ducts. *External pressure.* External pressure by tumors of the stomach, kidney, pancreas, or omentum ; by tumors of the liver itself, or enlarged glands in the fissure of the liver ; by accumulated feces in the colon ; by an abdominal aneurism ; and by the pregnant uterus, in rare instances, may cause jaundice. Jaundice due to *disease outside of the ducts* is gradual in onset, varies in degree with the amount of pressure, and becomes chronic, except in pregnancy and from fecal accumulation ; it may cause death, or persist until such termination results from the primary disease. It is recognized by the absence of pain ; the presence of disease in other localities, indicated by its peculiar symptoms and signs ; the absence of a history of gallstones ; and, finally, by the patient's age. Its nature must be inferred from the symptoms and physical signs of disease in neighboring structures. If the jaundice is due to enlargement of the lymphatic glands, its nature may be inferred from the presence of primary carcinoma in other organs of the body, or from the condition of the lymphatic glands in other parts. If they are the seat of malignant disease, it can usually be recognized. Cancer of the liver must be excluded by its symptoms—enlargement with jaundice, with moderate fever, rapid emaciation, and short duration of the disease. In the large majority of cases this form of jaundice is due to *disease of the pancreas,* particularly carcinoma.

2. Jaundice from disease of the ducts themselves. Catarrhal inflammation, suppurative inflammation, or adhesive inflammation of the ducts ; and cancer or other tumors of the duct cause jaundice.

Jaundice due to *disease of the ducts* presents various features. The most common form is that due to *catarrhal inflammation* of the ducts. The jaundice comes on suddenly, at least within forty-eight hours after the onset of the symptoms ; there is no pain, but it is attended by vomiting and other symptoms of mild gastritis, and is usually accompanied by itching. It follows indiscretions in diet, and occurs in young subjects. A definite cause for the gastritis can usually be found.

The *diagnosis* is based upon the age, the association of the jaundice with gastritis, for which a definite cause can often be assigned; the absence of organic heart disease, or any lesion within the body, on account of which jaundice might arise; the moderate degree of jaundice, the absence of emaciation and symptoms of portal obstruction, the occurrence of moderate enlargement without pain. It must not be forgotten that jaundice due to obstruction from gallstones, or to pressure from tumors outside of the duct, is characterized in its onset by symptoms similar to those just mentioned. It is often necessary to wait before giving an opinion; a history of previous attacks of jaundice and the age of the patient, over forty years, also lead to caution in the diagnosis.

If the jaundice is due to *suppurative inflammation of the ducts*, cholangitis, the infection is usually associated with a previous history of gallstones. It must not be forgotten, however, that other lesions, which cause jaundice, may invite an infectious inflammation of the ducts also, such as obstruction by external pressure. The course of the jaundice is chronic. Fever and other symptoms of an infection attend it. In *adhesive inflammation* there is a history of trauma from gallstones, and the affection is chronic. In *cancer of the gall-ducts* the advent of jaundice is slow, the course protracted; the symptoms are the symptoms of carcinoma, to which are often added the physical signs of an enlarged gall-bladder. (See Diseases of the Gall-ducts.)

3. Jaundice from obstruction within the ducts. Foreign bodies within the ducts, as inspissated mucus, gallstones, or parasites, such as round worms or hydatid cysts, are the common causes of the occlusion of the ducts which may cause jaundice.

Foreign bodies within the ducts cause jaundice by direct obstruction, or by the catarrhal inflammation which their presence excites. The symptoms occur suddenly in the former instance, gradually in the latter. The characteristic symptoms of *gallstones* precede the jaundice. The patient is usually a woman past forty years, with habits of life which predispose to the formation of calculi. Colicky pains occurring in paroxysms, intermittent jaundice varying in intensity, and an intermittent fever, point to this form of obstruction.

Jaundice due to lowering of the blood-pressure in the liver, so that the tension between the bile-ducts and the blood-passages is altered, occurs suddenly, is light in degree, and is not attended by marked symptoms; it is due usually to shock or emotions.

Hæmatogenous Jaundice. Jaundice is *hæmatogenous* or *non-obstructive* when (1) the function of the liver-cells has been suppressed, as in acute yellow atrophy of the liver; (2) when blood-destruction is in excess of the capacity of the liver to remove the product of destruction—the urobilin, as in certain forms of malaria, in pernicious anæmia, in certain fevers, and other toxæmias. The onset of the jaundice is rapid, the general symptoms are more pronounced, particularly the cerebral symptoms. They occur simultaneously with the jaundice. They are *infectious*, as in acute yellow atrophy of the liver and in Weil's disease. The *toxic* forms of hæmatogenous jaundice are not severe; the discoloration of the skin is light yellow, and may not

even be observed by the patient, nor cause pronounced symptoms. The blood is destroyed rapidly in these cases, and, as it cannot be disposed of by the liver, spleen, or kidneys, the transformed hæmoglobin is deposited in the tissues. In this class of cases the urine contains but little bile-pigment, but there is a large amount of urobilin and indican. The stools are not clay-colored.

Malignant or Infectious Jaundice. *Acute Yellow Atrophy of the Liver.* Acute diffuse inflammation of the liver, with necrosis of the cells, characterized by jaundice and cholæmia. Many of the cases occur during pregnancy. It is most common prior to the thirtieth year. It is said to follow fright. The symptoms are local and general. Jaundice is at first noticed after an attack of gastroduodenal catarrh. It is light, occasionally extends over the entire body, and is not usually attended by itching. After a continuance of these mild symptoms for from two days to two weeks, the patient complains of headache ; delirium sets in with stupor and convulsions. The headache is attended with vomiting. Fever of moderate degree begins at the same time, although in some cases it is absent.

Although the jaundice is not intense, the effects upon the blood are early seen ; hemorrhages underneath the skin and from the mucous membrane take place. In pregnant women abortion follows, the hemorrhage from which may be very excessive. The stupor and delirium are followed by coma, and death takes place in the first week ; or coma may be preceded by the typhoid state, and the disease lasts longer than a week. The urine is bile-stained, and contains albumin and casts. It diminishes in amount, and is soon passed involuntarily. Leucin and tyrosin are always present. The latter may be seen in the sediment, although it is more marked when a few drops are evaporated on a cover-glass. The bowels are loose and the stools involuntary and clay colored.

On examination the liver is found to be diminished in size ; this may not be appreciated by percussion in the anterior region, but in the axillary region the width is reduced one to two inches. There may be some tenderness over the liver and over the ducts.

Diagnosis. The data upon which a diagnosis is based are the age, sex, pregnancy, the rapidity of onset of cerebral symptoms following jaundice, diminution in the size of the liver, with leucin and tyrosin in the urine. It must be distinguished from the jaundice of hypertrophic cirrhosis of the liver, which at times becomes malignant. Some observers have thought that acute yellow atrophy may supervene upon this form of cirrhosis, thereby causing malignant jaundice ; but there is more fever than in atrophy, while leucin and tyrosin are not found in the urine. It must not be forgotten that all cases of jaundice may terminate suddenly with delirium, followed by coma, or by the development of the typhoid state.

In *phosphorus-poisoning* the hemorrhages, the jaundice, and diminution in the size of the liver are the same as in acute yellow atrophy. Gastric symptoms are more marked, and leucin and tyrosin are not present in the urine.

Weil's Disease. This infection, in which *jaundice* is the chief symptom, is considered in the chapter on Infectious Diseases.

Yellow Fever. The account of the jaundice attending this infection is found in the chapter on Infectious Diseases.

Infantile Jaundice. Jaundice in infants is due to two causes: First, *congenital obliteration* of the ducts; and, second, *catarrhal inflammation*. It must not be confounded with the yellow discoloration of the skin, due to the excess of coloring-matter in the blood, which is not disposed of by the liver.

In *congenital obliteration* of the gall-ducts jaundice rapidly ensues and deepens to an intense degree; hemorrhages occur, the child becomes stupid or comatose, may have convulsions, and death takes place in coma. There is rapid emaciation, and the liver and spleen are enlarged. The child may live many months.

Simple *catarrhal jaundice* in infants is associated with moderate gastric disorder. The jaundice is light; the conjunctivæ alone may be discolored. In infants malignant or infectious jaundice may be due to inflammation of the portal veins, secondary to umbilical phlebitis. The jaundice develops after suppurative inflammation about the umbilicus, and is attended by fever. There may be some tenderness over the liver; frequently peritonitis develops at the same time. Pyæmic symptoms may set in, and pus may be found in other situations. If death does not ensue early the jaundice becomes more pronounced and causes cutaneous and mucous hemorrhages. Convulsions and coma are apt to supervene before death. Jaundice in infants also occurs in interstitial hepatitis of syphilitic origin. The evidences of hereditary syphilis are seen in the skin and mucous membranes. The liver is enlarged, and there may be tenderness from perihepatitis.

Fever. *Hepatic Fever.* The occurrence of fever may be of diagnostic importance in distinguishing the various forms of obstructive jaundice. Fever occurs frequently in jaundice; but is significant in certain forms only. In *catarrhal jaundice* it is present for three or four days only, disappearing as the severe gastric symptoms subside. It is probably toxic. In *hepatic colic*, with jaundice, it is transitory and associated with chills and sweats. In jaundice from *obstruction* it occurs when an infectious cholangitis, primary or secondary, arises. A peculiar type known as *intermittent hepatic fever* (see page 202) is often seen. The intermittent fever is associated with gallstones in the following groups: First, with each paroxysm of hepatic colic moderate fever and jaundice are present. The latter becomes more intense after each paroxysm, but disappears in a short time. The paroxysmal attacks may recur at intervals for years. Second, the hepatic colic is attended by distinct ague-like paroxysms of chill, fever, and sweat, after each of which the jaundice, which continues to the end, is more intense. Third, hepatic colic and gastric disturbance occur with fever, but without jaundice. The symptoms occur in distinct paroxysms. *Gallstones* are probably the cause in all these conditions, leading in some cases to chronic obstruction of the duct without infection.

If an *infectious cholangitis*, with or without gallstones, is present, the symptoms are somewhat different, although the fever is of the same

type. Thus (1) there is more tenderness in the hepatic region, with enlargement of the gall-bladder; (2) the paroxysms are more frequent; (3) jaundice is not so intense and not influenced by paroxysms; (4) the patient is ill in the intervals, and there is wasting. There are no periods of improvement locally or in the general condition. The most important point in cases of gallstone is the subsidence of all symptoms between the paroxysm of fever.

Intermitting fever of this character must be distinguished from *malaria*. The history of gallstones, with pain in the region of the liver, and the negative appearance of the blood, are sufficient to establish the diagnosis.

Hepatic fever also occurs in cancer when the neoplasms grow rapidly, in certain forms of cirrhosis, and in obstruction from other causes than gallstones. It is particularly common in suppurative inflammation of hydatid cysts, or after they rupture and discharge into the biliary vessels. Without previous knowledge of the hydatid cyst the diagnosis is almost impossible, save that the pain is less when the obstruction is due to this cause than in obstruction from the passage of gallstones.

Palpation. By palpation the lower border of the liver can be determined in thin subjects, or in those in whom the liver is greatly enlarged. It may be difficult to determine the border when the abdomen is distended on account of flatulency. Careful palpation must be made with the tips of the fingers, pressing them firmly inward along the margin of the ribs, at the same time securing relaxation of the abdominal muscles by having the patient take a full breath, and having the legs drawn up and the shoulders elevated. The pressure should be made in the intervals following the act of inspiration. By care and patience the fingers can be pushed deeply inward and be made to feel the border of the liver, even in health. Care must be taken not to cause contraction of the right rectus muscle, for if this takes place the indurated mass may simulate tumor or enlargement of the liver. The left lobe of the liver, below the ensiform cartilage, extends half-way to the umbilicus. Here it is most accessible to palpation. By palpation we also determine the size of the gall-bladder and the degree of movement of the liver in respiration. On full inspiration the liver descends, and during the act of expiration rises again. This movability is of service in distinguishing the liver from other organs that are fixed within the abdomen.

In *amyloid* disease the lower edge is smooth, rounded, the tissue dense and unyielding to pressure, and the anterior surface perfectly smooth, as a rule; but when the liver is also cirrhotic or syphilitic the surface may be irregular and fissured.[1]

The *fatty* liver has also a rounded smooth border, but its tissue is not so dense and resistant, except when cirrhosis coexists. Its surface is smooth.

In single *abscess* the liver is enlarged, but not uniformly, and not invariably. If the abscess is located in the right lobe, and nearer the anterior than the posterior surface, palpation may be able to detect not only enlargement, but also deep-seated obscure fluctuation, surrounded

[1] See Musser: "Amyloid Disease of Liver," Penna. State Medical Journal, 1899.

by a zone of hard tissue. The tumor is round, smooth, tense, tender, and painful.

In *multiple abscesses* the liver is enlarged uniformly, and usually none of the abscesses are large enough to be felt as a distinct prominence. The liver is tender and painful.

In *hydatid tumor* the degree of enlargement depends very much upon the situation of the cyst, upon its stage of development, and upon the activity of the echinococci. Sometimes the cyst is so small that its existence remains unsuspected; at other times the enlargement is so great as to fill the abdominal cavity. As in abscess, the possibility of detecting the tense, globular, fluctuating, painless tumor characteristic of the disease depends upon its situation. If it is on the anterior surface or lower border, it is easily detected, especially if the tumor is at all large; but if it projects from the posterior surface or from the upper or lateral borders, detection is difficult, and may be impossible.

In *congestion of the liver* the enlargement is not so great as in abscess, nor are pain and tenderness so pronounced. Moreover, the enlargement is usually not permanent. The lower border, if it projects below the edge of the ribs, is smooth.

In *hypertrophic cirrhosis* the enlargement is moderate, the surface smooth, or but slightly roughened, denser than normal, and somewhat tender.

In *cancer* the enlargement resembles that of single abscess and hydatid tumor in that it is irregular. But, unlike hydatid tumor, the irregularities are due to knobs or bosses which project from the surface of the liver, are usually entirely free from any fluctuation, and are tender on palpation. There may be a single large mass, or a number of knobs or nodules. The part projecting below the ribs may be free from any nodules.

Palpation of the liver may discover a *friction* from perihepatitis, and *pain* or *tenderness* from that cause, or from cancer or abscess. *Pulsation* of the liver may be a transmitted impulse from the abdominal aorta or a venous pulse, such as occurs also in the jugulars, from tricuspid regurgitation.

Floating liver is diagnosticated by feeling in the lower, most frequently the right portion of the belly, a large tumor, which may, however, easily be confounded with tumors of other organs. It can be distinguished as liver: (1) By recognizing the notch; (2) by the presence of a tympanitic note in the proper region of the liver, as loops of intestine lie between the diaphragm and liver; (3) by the excessive movability of the tumor; and (4) by the fact that it is possible to replace the liver; (5) by its size and consistency. It occurs almost exclusively in women, possibly as the result of a congenital lengthening of the suspensory ligament, although more likely from relaxed abdominal walls. It may be confounded with ovarian cyst, appendicitis with tumor, and movable right kidney with hydronephrosis.

Constriction of the liver from tight lacing (*Schnurleber*) occurs chiefly in women. Tight corsets, and, still more, tight waist-straps or bands, squeeze the liver downward, especially the right lobe, so that it can be palpated. In more pronounced cases a furrow, often palpable, is pro-

duced, and, below this, a constricted lobe which may extend as far down as the anterior superior spine of the ilium and carry the gall-bladder with it. In other instances, the right lobe is elongated, extending even to the crest of the ilium.[1]

Lobes so depressed are usually thin and easily movable, and can be grasped with the hand and moved to and fro. If the lobe does not reach so far downward, it is more rounded and blunt in shape. It is not always easy to demonstrate its connection with the liver, because coils of intestine lying over the liver in the furrow make palpation difficult, and cause a tympanitic note between the liver-dulness and the dulness of the constricted lobe.

Confusion with tumors of other kinds can be avoided usually by deep palpation or percussion.

Gall-bladder. When the gall-bladder has a certain degree of fulness, it may, according to Gerhardt, be not only felt in healthy persons, if the stomach and bowel are empty, as a smooth, round, fluctuating tumor at the lower border of the liver, but be even visible and be outlined by percussion. If a line is drawn from the right acromion process to the umbilicus, it will bisect the gall-bladder at a point where it passes over the margin of the ribs. The fundus is situated below the edge of the liver, at about the ninth costal cartilage, just outside the edge of the right rectus muscle. Palpation is easy when, owing to closure of the cystic duct, the gall-bladder is distended with bile or with inflammatory exudate, or enlarged by thickening of its walls or by an accumulation of gallstones. A pear-shaped tumor is then felt which, if not adherent to the border of the liver, is movable with it. In simple stasis, hydrops vesicæ felleæ, and purulent inflammation the tumor is tense and elastic; in inflammatory or carcinomatous thickening of the wall, dense and irregular. Calculi can often be recognized by the form or hardness or by the sound made by rubbing them together.

Aspiration. We are warranted in determining the nature of an obscure enlargement of the liver or of the gall-bladder by aspiration. In abscess, pus; in hydatid disease, the characteristic fluid, may be withdrawn.

In a case of local enlargement the apex of the swelling should be aspirated. If aspiration is performed near the upper border, the needle should be thrust downward; if near the lower border, upward. The left lobe should be aspirated with care, in order that the stomach be not pierced. (See Aspiration in Diagnosis.)

Auscultation. By auscultation we may detect a *friction*-sound in perihepatitis; a *grating* or *rubbing* when the gall-bladder contains calculi if it is palpated; a continuous murmur in tricuspid regurgitation.

Percussion. The Size and Shape of the Liver. (See Plate XVI., Fig. 1.) *Diminution in size* can only be recognized by percussion. The normal extent of hepatic dulness is diminished. This

[1] Musser: Transactions Philadelphia Pathological Society, vol. x.

is usually more marked in the anterior and lateral regions. The diminution is due to simple or acute yellow *atrophy* of the liver or *cirrhosis*. It must not be confounded with the apparent diminution that takes place in emphysema, or that which occurs from distention of the bowels with flatus, as in peritonitis. Absence of hepatic dulness may occur when there is gas in the peritoneal cavity. When there is considerable distention of the intestines by gas, the anterior and lateral hepatic areas may be tympanitic.

Enlargement of the liver is determined by inspection, palpation, and percussion. By *percussion* the size of the liver is accurately made out. Any marked increase of hepatic dulness beyond the normal limits (see p. 861) usually means increase in size of the liver. Both superficial and deep percussion must be performed. Palpatory percussion is of great advantage.

The upper border is determined by percussing from a point above the liver-area toward the liver—anteriorly from the third interspace downward, laterally from the fourth, and posteriorly from the angle of the scapula. In health the upper border of the liver is found at the fifth interspace; in the axilla, at the sixth; and in the back, at the ninth interspace. Thence downward hepatic dulness should continue to the margin of the ribs. It falls short of this position by at least an inch in the aged, and in deep-chested persons it may not be more than two inches in width in front. The width of the liver-dulness in the right mid-clavicular line is about four inches, in the mid-axillary line six inches, and in the mid-scapular line three inches.

Extent and *direction* of enlargement. The entire liver may be enlarged and of normal shape, or its outline may be irregular; again, the enlargement may be limited to one lobe. Hence, the area of dulness may be increased in all directions, or the increase may be above or below the normal limit, if the normal shape is preserved. By percussion it may be found that the enlargement is regular from increase in size upward or downward, or increase in the area of dulness in both directions. On the other hand, if the enlargement is irregular, the liver-dulness may begin higher in the anterior region than in the axillary region, or may extend beyond the margin of the ribs in a limited area. When the enlargement is limited to the left lobe it is revealed by increase in the dulness from the xiphoid cartilage downward as far as the umbilicus. The entire middle region to the navel may be filled up by the enlarged liver.

Uniform enlargement of the liver is due to congestion, hypertrophic cirrhosis, fatty degeneration, amyloid disease, leukæmia, cancer, and sometimes to hydatid disease and abscess. *Enlargement of one lobe* of the liver is due to hydatid disease, to abscess, or to cancer, in nearly all cases. Either the right or the left lobe may be the seat of such enlargement.

Enlargement in one direction is due also to the three conditions just indicated. Although in abscess or hydatid disease enlargement *downward* is the more common one, it may be directly upward, the lower border of the liver occupying the normal position. When enlargement of the liver extends *upward* it is due to a cyst, or an abscess in the convex surface of the right lobe.

Irregularity in the shape of the liver-dulness occurs in cancer, in abscess, and hydatid disease. Notwithstanding the apparent irregularity, enlargements of the liver conform to its usual outline, with but moderate variations, and always occupy the *normal site* of the organ.

Diagnosis. Enlargement of the liver must be distinguished from enlargement of organs in contiguity with the liver, and from structures usually containing air, which have become solid or non-resonant. The enlargement must, therefore, be distinguished from pleural effusion, from disease of the lungs which causes dulness on percussion, or from disease of the abdominal organs causing increased dulness near the hepatic region. Hence, in renal tumors, in tumors of the large intestine or stomach, in ovarian tumors, in tumors due to accumulation of feces, the physical signs on percussion may simulate enlargement of the liver.

Simulated Enlargement. It is well to bear in mind the conditions which simulate enlargement of the liver. Of these we have :

1. *Congenital* malformation : the liver may be of abnormal shape, on account of which the area of dulness will be increased in a particular direction. It may be quadrangular or rounded. The liver may be found in the right pleural sac in congenital diaphragmatic hernia. The increase of dulness upward will simulate enlargement of the liver. Congenital malformations may be suspected in the absence of any symptoms of hepatic disease, or of conditions which may cause other forms of spurious enlargement. Moreover, the increased dulness will have existed from early life.

2. In *rhachitis*, on account of the malformation of the chest, the position of the liver may be such that its area will be increased. For the same reason the liver may be felt below the margin of the ribs.

3. Disease of the *spinal column* causes dislocation, on account of which the liver may apparently be increased in size.

4. Enlargement of the liver must be distinguished from *pleural effusions.* This is sometimes difficult. The symptoms of the pulmonary affection must be considered. The general conditions which cause hydrothorax must be borne in mind. The difficulty in distinguishing the two arises because the dulness of each is continuous. In pleural effusion, however, there is uniform bulging of the affected side. The liver is not movable, the chest-expansion is lessened. The upper border of dulness of the fluid may be movable if the effusion is not large, while the line of dulness is S-shaped—that is, high behind and high in front. If the effusion is large, the upper limit of dulness is horizontal. The upper limit of dulness in the pleural effusion changes its position in many instances. In enlargement of the liver the lower ribs are often everted, but in pleural effusion a depression may be seen between the lower margin of the ribs and the upper surface of the liver, if the latter is dislocated by pressure of the fluid. Sometimes enlargements of the liver give rise to secondary pleural effusion, so that too often, after finding pleural effusion, the size of the liver is not estimated.

5. *Pericardial effusion* and dilated heart are said to simulate enlargement of the liver. The history of the case, the origin and mode of

development of the symptoms, the physical signs of cardiac disease, point to its true nature.

6. Enlargement of the liver may be due apparently to *subdiaphragmatic abscess.* The history of the case is generally essential to a diagnosis. The accumulation between the liver and diaphragm causes the latter to be pushed downward. It is very difficult to distinguish the spurious from the false enlargement in these instances. *Aspiration* may help in the diagnosis.

7. *Abnormal Condition of the Abdominal Parietes.* Increased tension or spasm of the recti muscles, giving rise to phantom tumors of the abdomen, simulate enlargement of the liver. They occur in young girls, and are associated with gastro-intestinal catarrh and symptoms of hysteria. Anæsthesia must often be employed to disperse the swelling.

8. *Tight Lacing.* This may displace the liver upward or downward, according to the direction of the pressure. It may also, by exerting lateral compression, bring more of the liver into contact with the anterior abdominal wall. And finally, if the constriction has been by a strap or tight cord, a portion of the liver may be more or less detached and appear as a movable tumor.

9. Some enlargements of the *abdominal contents* cause spurious enlargement of the liver. In the same way increased abdominal pressure (ascites, tympanites, etc.) causes the liver to rise higher than normal.

a. The accumulation of feces in the colon. This causes continuance of liver-dulness downward, on account of which it may be thought that the patient has liver disease. A purgative must be given.

b. An ovarian cyst.

c. The presence of ascites. Exclusion of the latter is sometimes difficult, because the ascites may be loculated and situated in the hepatic region. It may give rise to symptoms of hepatic enlargement. Probably aspiration alone can establish the diagnosis. Ordinary ascites should be easily distinguished by the physical signs and the result of exploratory puncture.

d. Tumors of the omentum, chiefly tuberculous, may occupy such relation to the liver as to increase the dulness downward. The history, the occurrence of the omental tumor, with symptoms of tuberculosis, may aid in determining the true condition.

e. In tumors of the kidney, which simulate enlarged liver, it is found that the edge of the liver cannot well be felt, but Murchison thinks the fingers can usually be inserted between the ribs and the upper part of the renal tumor. The renal tumor, however, is not fixed. It is rounded on every side; it has the shape of a kidney. It may be associated with changes in the urine.

f. Enlargements of the liver must be distinguished from pancreatic cyst, or effusion in the lesser peritoneal cavity. This can usually be accomplished with ease, except in hydatid disease of the left lobe near the suspensory ligament. In effusion in the lesser peritoneal cavity the tumor occupies the left upper quadrant, and may extend as low as the transverse umbilical line. It causes dislocation of the heart, so that the apex is as high as the third interspace, and beyond the mid-

clavicular line. It is accompanied by an increase in the dulness posteriorly, so that the upper limit may extend to the angle of the left scapula. Puncture may furnish the necessary information.

The presence or absence of pain may sometimes furnish a clue to the nature of the enlargement of the liver. Murchison considers this a reliable distinction. *Painless enlargements* of the liver are due to passive congestion, to hydatid disease, to fatty and amyloid disease of the liver. *Painful enlargements* of the liver are seen in abscess, cancer, and syphilitic disease, with perihepatitis.

In children the lower border of the liver is normally lower than in adults, because the liver is itself proportionately larger. For the same reason the upper border is at a higher level.

Enlargement of the Liver. Enlargement of the liver occurs in the *congestions;* the acute *inflammations*, except acute yellow atrophy; the chronic inflammations, except cirrhosis; the degenerations, the morbid growths, and in hydatid disease. The physical signs have been considered *seriatim* in the pages immediately preceding. It must be remembered that the disease may occur without great changes in the size of the liver. The *congestions* have been considered in the previous pages.

The remaining diseases of the liver will be considered in accordance with their pathological classification. After the congestions, we have the inflammations, then the morbid growths, then the degenerations, and, finally, hydatid disease.

Abscess of the Liver.

Two forms are seen: tropical abscess, so-called, in which one or two abscesses are found; and multiple abscesses, found throughout the liver-structure. The single or solitary abscess usually occurs in the course of dysentery, and, in all probability, in the amœbic form only. A single abscess may also be due to traumatism, particularly in children. Multiple abscesses occur secondarily to inflammation somewhere in the portal area. Inflammation and abscess about the rectum, inflammation of the appendix, ulceration anywhere in the gastro-intestinal tract may be followed by multiple hepatic abscesses. The abscesses, however, do not occur directly by means of emboli, as in the case of amœbic abscess, but after inflammation of the portal vein or *suppurative pylephlebitis*. Multiple abscesses of the liver also follow obstruction and infectious inflammation of the biliary passages (*suppurative cholangitis*).

Tropical abscess or amœbic abscess varies in its clinical course. In a typical case the clinical picture is that of the general symptoms of suppuration setting in in the course of, or soon after, an exacerbation of amœbic dysentery, with local symptoms referred to the liver.

Symptoms. The *general symptoms* are those of intermittent fever, paroxysms of which may occur daily or only every second day, attended by chill, fever, and sweat. The fever may be remittent or continuous.

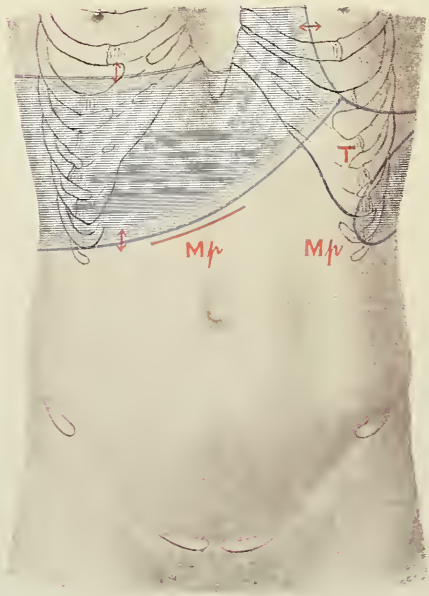
The complexion in tropical abscess of the liver is peculiar, as all writers upon tropical disease agree. The skin is sallow, the complex-

PLATE XL.

FIG. 1.

Abscess of the Liver.

FIG. 2.



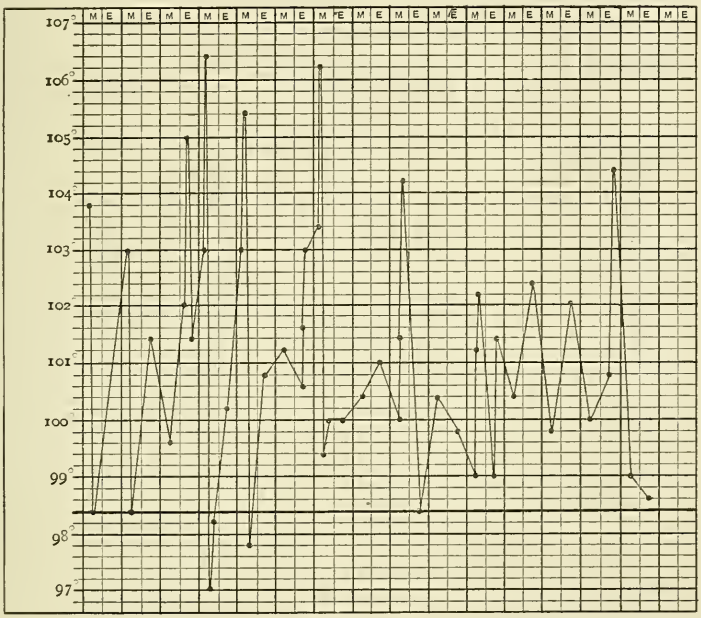
Hypertrophic Cirrhosis of the Liver with Enlargement of the Spleen.

ion muddy, the face pale. Through this a slightly icteroid tint may be seen, and the conjunctivæ are bile-tinged. Distinct jaundice is rare.

The *local symptoms.* Pain in the region of the liver; this may be referred to the region of the right or left lobe. It may be seated in the fifth or sixth interspaces anteriorly, or behind at the ninth and tenth ribs. There may be pain in the right shoulder. The pain may be paroxysmal, or it may be intense and persistent.

The patient complains of weight and fulness in the region of the liver; the enlargement causes some dyspnœa, and may cause cough and some vomiting.

FIG. 201.



Intermittent fever in abscess of the liver.

Physical Examination. (Plate XL., Fig. 1.) The liver is enlarged. The enlargement may be uniform; if the abscess is central, the entire organ takes part in the swelling; on the other hand, it may be an enlargement upward in the anterior, the axillary, or the posterior region. If the convex surface of the right lobe of the liver is affected, the enlargement is usually upward. If the lower portion of the right lobe is affected, enlargement extends downward, and the lobe of the liver can readily be detected on palpation. The mass may extend outward from the liver-edge. At first it is hard; ultimately it softens and may fluctuate. If the abscess is limited to the left lobe of the liver, and is situated about the suspensory ligament, the enlargement may be seen below the xiphoid cartilage. It may extend to the umbilicus and project forward. Sometimes it may be so large as to cause eversion of the ribs of each side, and render the entire epigastrium unusually

prominent. The surface may become reddened. Over the tumor there is tenderness on palpation, and there may be, as in other situations, fluctuation. Œdema of the surface is frequently seen.

The irregular enlargement above mentioned is made out by percussion. The enlargement may be difficult to ascertain, on account of secondary pleural effusion, or secondary pleural inflammation, with the development of a hepato-pulmonary fistula, causing dulness posteriorly. If the case has been seen from the first, a friction-sound may be heard, followed by the physical signs of effusion.

The appetite is lost, and nausea at the sight of food is pronounced. The condition of the bowels may vary with the state of the intestinal tract at the time of the hepatic complication. The dysenteric symptoms may subside entirely or they may continue. Often there is only constipation, with the passage of mucus and hardened feces. In an obscure case the study of the stools should be made. The detection of amœbæ in the mucus or in the feces may point to the true conclusion.

Atypical cases are characterized by the absence of general symptoms, or the absence of local signs. Fever may be absent entirely, exhaustion alone being present, which could probably be ascribed to the previous dysentery. Pronounced anæmia due to the dysentery may be associated, and even be the most marked symptom, as well as inflammation of the joints, or neuritis. In a case under my care the only symptom for a long time, with the exception of anæmia and loss of appetite, was severe pain in the sixth interspace. In other instances there are no liver-symptoms whatsoever. General symptoms of infection, or an irregular, or even a continued fever, the cause of which cannot be ascertained, may alone be present. In one of my cases there was moderate continued fever, with loss of appetite and dyspeptic symptoms. There was no diarrhœa. No cause could be given for the fever, although it was noted that there was slight enlargement of the liver. The patient slipped out of the ward and went down to the yard to smoke ; on his return he was seized with an intestinal hemorrhage which could not be checked and which resulted fatally. At the autopsy a large abscess of the liver was found, and there was ulceration of the rectum from which the intestinal hemorrhage took place.

The *diagnosis* is usually not difficult in the typical cases. Under all circumstances attention must be paid to the facts bearing upon the etiology and the association of general and local symptoms. If the general symptoms of suppuration are present, malarial abscess may be mistaken for an intermittent fever. The result of an examination of the blood and of treatment by quinine would establish a diagnosis of malarial fever. It is difficult sometimes to determine whether the abscess is in the abdominal wall or in the liver proper, or whether it is situated beneath the diaphragm. If the liver is movable with respiration, the two former conditions may be excluded. An abscess in the abdominal wall is not influenced by respiration, and in subdiaphragmatic abscess the movement is impaired. Suppuration of a hydatid cyst cannot be distinguished unless it has been known beforehand that a simple hydatid was present in the liver. Under such circumstances, if suppuration occurs, it is likely to be confined to the cyst. Abscess

of the liver must be distinguished from gallstones, attended by intermitting fever without suppuration. While the distinction is difficult in many cases, yet the history of the case, the association of jaundice which deepens after each paroxysm, and the good general nutrition of the patient point to gallstones. Abscess of the liver is of shorter duration than cholelithiasis, and its primary cause can usually be ascertained by examination of the rectum or the discovery of suppuration in other parts of the body.

Exploratory puncture must be employed in many cases, and it can usually be done with safety. Puncture must be made over the region in which the enlargement is greatest, or at which the swelling is most prominent. In abscess secondary to dysentery a brownish-colored pus will be withdrawn, resembling anchovy sauce. It may be of a peculiar odor, and, on examination, amœbæ common to this form of dysentery may be found. If there is no point of election, the needle may be introduced in the lowest interspace in the anterior axillary, or the seventh interspace in the mid-axillary line. A fairly large-sized aspirator should be used. Suppuration may be present, and yet not be reached by aspiration.

Suppurative Pylephlebitis. Abscess of the liver may be due to *pyæmia*. It may be a part of general pyæmia, or of portal pyæmia. Parasites and foreign bodies, as well as gallstones, may excite an abscess. The echinococcus cyst may suppurate, or round-worms may penetrate to the liver and cause suppuration.

The symptoms of *suppurative pylephlebitis* and of *pyæmic abscess* are general and local. Jaundice is more common than in solitary abscess, and there are greater pain and tenderness over the liver, which is uniformly enlarged and tender. With the enlargement of the liver and jaundice we have the symptoms of pyæmia. They are not peculiar. Sometimes the fever is distinctly intermitting, or it may be irregular and septic in character.

The symptoms of solitary abscess of the liver, as has been previously stated, may be obscure, and attention be called to the liver only when symptoms arise due to a rupture into the neighboring organs. If perforation takes place into the peritoneum, it is not likely that the cause can be established during life. The perforation frequently extends through the diaphragm to the pleura, and then to the lung. An empyema may be set up, the true source of which may not be ascertained unless the pus is examined. The physical signs are those of empyema —dulness or diminished resonance, absence of fremitus and vocal resonance, diminished breath-sounds, and impaired movement, together with symptoms of cough and dyspnœa. When the lung is infected the physical signs may resemble those of consolidation. We find dulness, bronchial breathing, and increased tactile fremitus. A harassing, convulsive cough occurs, and, sooner or later, expectoration of a reddish-brown, brickdust-colored material which resembles anchovy sauce. This characteristic expectoration is decisive. It contains amœbæ, and, in addition to blood-pigment and corpuscles, orange-red crystals of hæmatoidin, cholesterin-plates, and leucin and tyrosin. When the abscess perforates into the stomach or bowel the discharge from either

cavity may be of the above-mentioned nature. Perforation into the pericardium is usually followed by immediate death.

Cirrhosis of the Liver.

A diffuse interstitial inflammation of the liver, frequently with atrophy of the organ, is caused, in the large majority of cases, by irritants which enter the portal circulation through the stomach. Of the irritants alcohol is the most common, and particularly the stronger liquors, as gin and whiskey. Other irritants, as spices used to excess, may likewise cause the diffuse inflammation. Cirrhosis of the liver may, however, be a sequel to the infectious diseases, notably scarlatina, and may be incited by malaria. The infectious forms of cirrhosis usually lead to atrophy of the liver.

Another form is due to obstruction of the bile-ducts, with secondary overgrowth of the connective tissue. It is known as hypertrophic or biliary cirrhosis. In addition, cirrhosis of the liver may arise in the course of syphilis; the histological characteristics are different from those of true cirrhosis. A secondary cirrhosis of the liver arises in the course of passive congestion of that organ, producing the so-called nutmeg-liver.

Cirrhosis of the liver of the atrophic form, due to alcohol, presents various clinical features. In the first place, it may exist without causing any symptoms whatever during life. It may be found after death from other causes, or it may not present symptoms until an accident occurs in the course of the disease, as hemorrhage from some portion of the collateral circulation. In both cases the symptoms are absent because the collateral circulation is complete. If this is incomplete, however, grave symptoms, local and general, ensue.

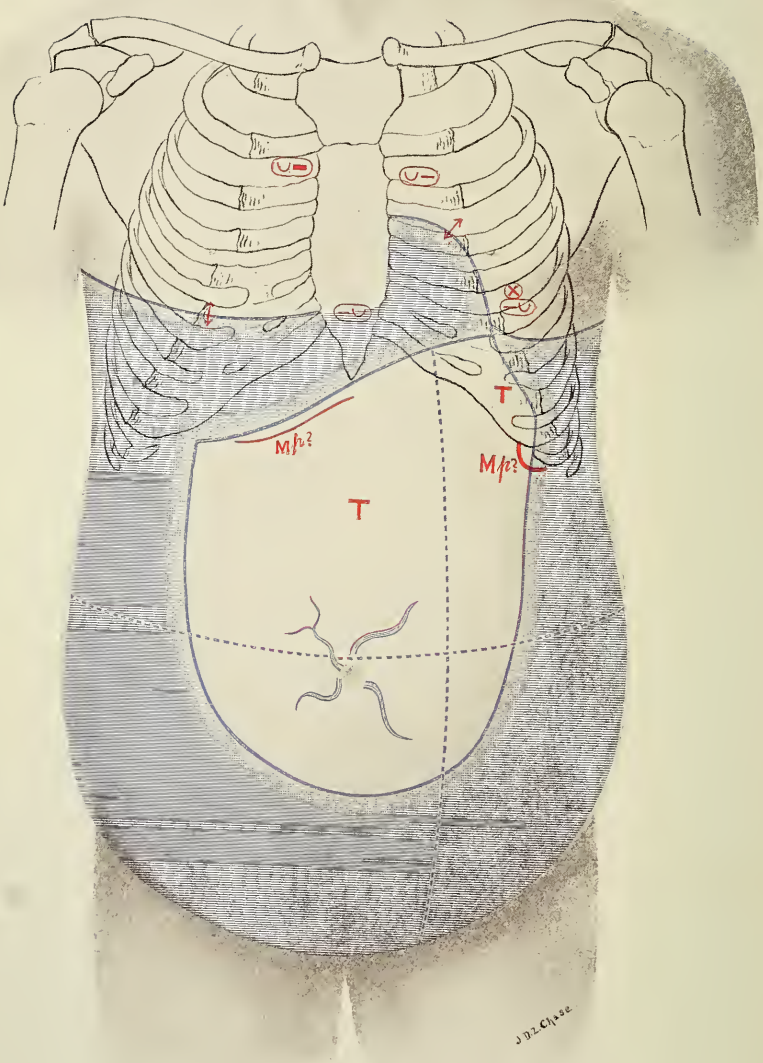
Before detailing them it may be well to state that the occurrence of one symptom, which we have termed accidental, may lead to the inference that cirrhosis of the liver is present, particularly if the patient has been an alcoholic. This symptom is hemorrhage. It may be of the stomach, causing death at once or after repeated hemorrhages; it may also take place from the intestine.

The Symptoms of Cirrhosis. The symptoms are general, due to interference with the nutrition of the patient; and local, their extent depending upon the degree of obstruction to the portal circulation. General symptoms rarely occur unless the local symptoms are present, as the latter cause malnutrition and mal-assimilation from interference with the gastro-intestinal digestion.

The symptoms have been divided into those of the first stage, or stage of enlargement, and those of the second stage, or contraction. The so-called first stage is not always observed.

During the *first* stage the symptoms are those of gastritis, with enlargement of the liver. The gastric symptoms are: morning retching or vomiting, with discharge of mucus, associated with other symptoms of gastric catarrh, as loss of appetite, nausea, tenderness in the epigastrium, eructations, and constipation, with loss of flesh and strength. The liver is *enlarged*, but the outline is regular.

PLATE XLI.



Cirrhosis of the Liver with Ascites.

During the *second* stage more severe symptoms arise, due to obstruction of the portal capillaries. The abdomen becomes distended and a sensation of weight and pressure is complained of. On examination *ascites* is detected. This may be enormous, causing monstrous distention, with pouting of the umbilicus. The *spleen* is found to be enlarged, extending over twice or three times the normal area of percussion. If ascites does not interfere, the edge of the spleen can be readily made out The portal obstruction causes secondary gastro-intestinal catarrh, if it was not already present, on account of the alcoholism. Although constipation is usually present, there may be persistent diarrhœa, usually lienteric and occurring in the morning only. Hemorrhages may take place from the gastro-intestinal tract at any time, either from the stomach or the intestine. Not infrequently they occur from the œsophagus, due to varicosity of the veins at the junction of the œsophagus and the cardiac end of the stomach. Hemorrhoids are always present and may bleed at each stool. Jaundice is not the rule, and, if present, is usually light and due to the duodenal catarrh. The skin has a yellowish tinge or only a grayish earthen color.

Physical Examination. (Plate XL., Fig. 2, and Plate XLI.) This may be rendered difficult before paracentesis is performed by the extensive ascites. The enlarged liver of the first stage will be found to have undergone contraction, although diminution in the area of dulness is not by any means as absolutely confirmative of contraction as the opposite condition is of hypertrophy. Percussion should be performed several times, because the distended intestinal coils may affect the results.

With the distention of the abdomen enlargement of the superficial veins is also observed. This may be very pronounced, particularly about the umbilicus. The enlarged, swollen mass of veins in this situation has been called, from its appearance, the *caput Medusæ.*

The general symptoms of cirrhosis, and particularly the symptoms of the later stages, are striking and diagnostic. The nutrition is much impaired. The patient, who, in the large majority of cases, had been corpulent, becomes emaciated. The skin changes in color and becomes of an earthy-gray or dirty sallow hue. The capillary venules of the face are dilated; the distended capillaries on the nose are distinct. Later, ecchymoses may occur in the skin, and hemorrhages take place from the mucous membrane and into the retina. Debility ensues; œdema of the ankles is almost sure to occur, and sometimes general anasarca may take place. It is extremely rare to have fever unless complications occur. The pulse is small and becomes more rapid than normal; the heart-sounds grow weaker. The skin may be the seat of eruptions, and chronic skin diseases of various kinds develop.

The *urine* throughout the disease presents no characteristics; as ascites develops, it becomes scanty and dark, and loaded with urates and uric acid. In rare instances it may contain sugar, and, if the uric acid is in excess, albumin.

Collateral Circulation. The collateral circulation that develops in order that the portal blood may reach the right heart takes place in various ways. First, communication may be formed between the veins

of the mesentery and those of the posterior abdominal walls; second between the coronary veins of the stomach and the veins of Glisson's capsule and the phrenic veins; third, between the hemorrhoidal and the inferior mesenteric veins; fourth, between enlarged veins occupying the position of the obliterated umbilical vein in the ligamentum teres, and the epigastric and mammary vein.

In the study of a case of cirrhosis of the liver a judgment as to its nature may be, in a measure, confirmed by the presence of other phenomena due to the same cause. Very frequently we have, at the same time, cirrhosis of the kidneys and sclerosis of the arteries, with secondary atheroma, both of which have led to hypertrophy of the heart. Strümpell refers to the association of cirrhosis and chronic tubercular peritonitis. He thinks the former is the primary lesion which predisposes to the development of the latter. The course of the disease is prolonged.

The duration cannot be determined accurately, as the onset is usually insidious. After the ascites appears the duration may vary from six to eighteen months. Of course, this depends largely upon the completeness of the compensatory circulation. Death usually occurs from intercurrent disease or progressive exhaustion. In not a few cases cerebral symptoms occur. In addition to the cirrhotic cachexia, the sudden occurrence of coma and convulsions, preceded by delirium, may ensue; the cause of this is not fully known. It must be borne in mind that the occurrence of these symptoms in an alcoholic subject may be due to a cirrhosis, the presence of which had not been suspected during life.

Diagnosis. The diagnosis is usually not difficult if the complete picture of the case is presented. It cannot be established positively without definite knowledge of the cause. If the patient comes under observation after ascites has developed, the diagnosis is more difficult. It must, in the majority of cases, be based upon exclusion of heart, lung, and kidney disease. A history of alcoholism and the presence of other symptoms of liver disease point to the hepatic origin of ascites. *Ascites* may be due to other causes within the abdomen, notably *chronic peritonitis*, exclusion of which is sometimes difficult. The general tenderness, the less marked distention of the abdomen, and the absence of enlargement of the spleen point to peritonitis. The *fatty cirrhotic liver* may present symptoms similar to those of the atrophic form, except that it is enlarged.

Hypertrophic cirrhosis, or so-called biliary cirrhosis, presents a somewhat different picture. In the first place, the cause is different. There is a history of gallstones, or obstruction of the duct from other causes. The liver is uniformly enlarged, and the surface is smooth and strikingly indurated. There are weakness and loss of appetite. Jaundice ensues very early, or may be the first symptom. It increases and persists throughout the course of the disease. Ascites is very slight or absent altogether. The enlargement and jaundice may continue for months or even years without the development of grave symptoms.

Fever may, however, set in at any time, being in all probability due to the biliary obstruction. It is continuous; the temperature rises

to from 102° to 104°; the tongue becomes dry and brown, the pulse rapid. All the symptoms of febrile jaundice ensue. The patient may be seized with convulsions in the course of the disease, followed by coma and death. Most authorities state that the enlargement persists throughout the course of the disease, but some observers say that after a long period of enlargement, with jaundice, contraction of the liver takes place, with symptoms of portal obstruction. Then the spleen may become enlarged and ascites take place, while the symptoms of digestive disturbances become more prominent. There may be nervous symptoms, due to acute, diffuse necrosis (acute yellow atrophy), setting in in the course of the disease.

The *diagnosis* is often difficult. Gradual and persistent jaundice without cause, continuing for a long time, associated with persistent enlargement of the liver without symptoms of portal obstruction in the non-alcoholic subject, points pretty certainly to hypertrophic cirrhosis of the liver.

Syphilitic Disease of the Liver.

Syphilitic disease of the liver may result in cirrhosis, or in the development of gummata. *Syphilitic cirrhosis* presents the same symptoms as the alcoholic form. The history, the marked irregularity on the surface of the liver, and the existence of syphilis elsewhere may lead to a diagnosis of the true condition.

In *congenital syphilitic disease* of the liver the inflammation is diffuse; the liver is enlarged and hard; the surface is smooth; there are usually syphilitic lesions in other organs; the patient presents syphilitic eruptions, and has the well-known wizened appearance that belongs to this affection.

Syphilitic gummata in the liver may exist without presenting any symptoms whatsoever, or they may reveal their presence by pain and a localized swelling and discomfort, which call the patient's attention to the region, particularly if his general health is reduced at the same time. Tumors are situated in the left lobe, in the median line, or along the margin of the ribs. The pain is usually localized in this region, but may extend more or less over the entire liver, particularly if there is general perihepatitis along with other evidences of syphilis; the latter are not always present, however. If the temperature is taken frequently, a moderate febrile range will be observed. It may not rise above 100½°, but in the absence of other causes it is a valuable diagnostic symptom.[1] In other instances the gummata may grow in such situation as to interfere with the portal circulation, or press upon the gall-ducts. The latter is very rare. If the gummata are felt, they appear as enlarged bosses which give the sensation of flattened hemispheres. Sometimes several separate elevations can be made out on the surface of the enlarged organ. To determine the exact nature of the lesion is often very difficult. The symptoms may conclusively point to hepatic disease. Knowledge of the presence of

[1] "The Diagnostic Importance of Fever in Late Syphilis." Musser: University Medical Magazine, October, 1892.

syphilis aids in the diagnosis. If without a syphilitic history there are scars in the throat, nodes on the bones, or other signs of syphilis, the diagnosis will be tolerably certain. Severe pain is more prominent in syphilis than in cirrhosis, and the nodules of syphilis are very different from the granular surface of cirrhosis.

The Fatty Liver.

The symptoms of fatty liver are not marked. The physical sign is a uniform enlargement extending in all directions. On palpation the edges can be felt; they are rounded and smooth. They are soft at first, but later become indurated. Fatty liver may be followed by cirrhosis after a period of alcoholism. The general symptoms are those of the primary disease. Fatty liver occurs in gouty subjects, but is notably present in wasting diseases, in tuberculosis, in chronic hip-joint disease, and in amyloid disease of the liver.

Fatty liver sometimes follows the congestion of the liver which is present in the course of organic heart disease. It is not a true fatty liver, but a fatty cirrhosis. There is increased fatty degeneration with an overgrowth of connective tissue. This form is associated with heart and kidney disease. On palpation the edges of the liver are indurated. The liver may undergo diminution in size later, and the symptoms of cirrhosis ensue.

Amyloid Disease of the Liver.

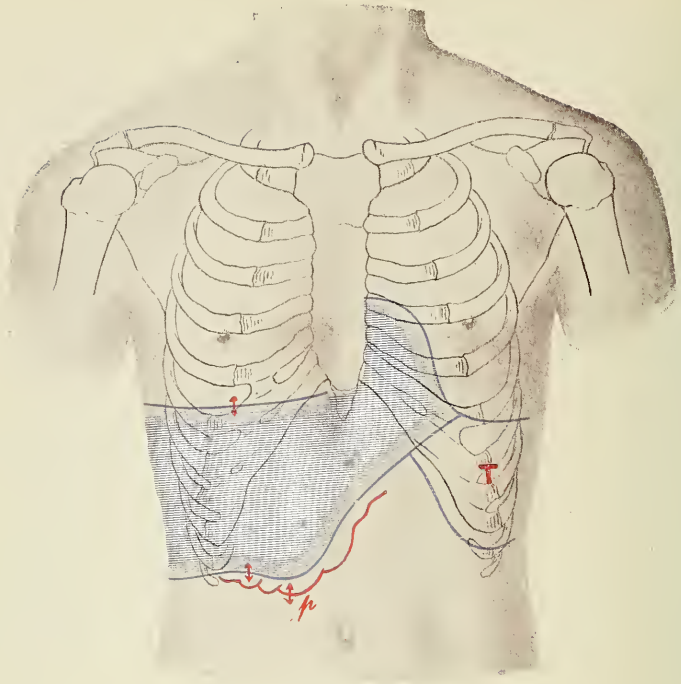
Enlargement of the liver without pain is often due to amyloid disease. Similar disease is found in other organs, and there is present, to point to the nature of the enlargement, syphilis, bone disease, prolonged suppuration, or tuberculosis. In amyloid disease the pallor of the patient is great; the face may be swollen, and the ankles slightly œdematous. The spleen is enlarged, the urine albuminous and abundant, but of moderate specific gravity. A history of syphilis is an important point in establishing the diagnosis. Fatty liver can readily be distinguished from amyloid disease by palpation. In the latter the surface is smooth, but very much indurated.

Cancer of the Liver.

The etiological factors upon which the diagnosis of cancer is based are: the age of the patient—most frequently between the fortieth and sixtieth year; the female sex, in a measure; and heredity. The disease is nearly always secondary to cancer in some other situation; consequently, in cases in which symptoms point to cancer of the liver, search must be made for the primary lesion elsewhere. The most frequent seat is the rectum, the uterus, the stomach, the remainder of the gastro-intestinal tract, the eye. The eye has been removed for obscure disease, and symptoms of carcinoma of the liver have subsequently developed. The nature of the hepatic symptoms was obscure during life, but at the post-mortem examination melanotic sarcoma was found; the primary lesion undoubtedly had been in the eye. Further etiological influences that may bear upon the diagnosis are: (1) The occur-

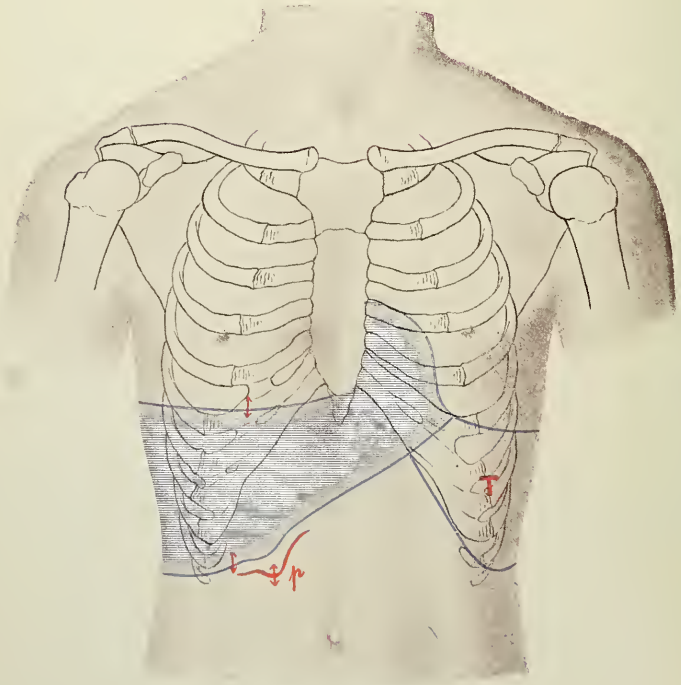
PLATE XLII.

FIG. 1.



Carcinoma of the Gall Bladder with Involvement of the Liver.

FIG. 2.



Enlargement of the Gall Bladder.

rence of gallstones, which act as the exciting cause in the development of primary cancer of the ducts, thence spreading to the liver; (2) the occurrence of trauma.

The *symptoms* of cancer of the liver may be due to (1) increase in the size of the liver; (2) to pressure of the growths upon the ducts or terminal portal vessels; and (3) to the general effects of carcinoma upon the system—the *cachexia*.

Physical Signs. (Plate XXXVIII., Fig. 2, Plate XL., Fig. 2, and Plate XLII., Fig. 1.) The liver is enlarged and its surface irregular. The organ can be made out, by palpation, extending below the margin of the ribs. The edges are irregular, and, on the surface, bosses can be distinctly felt. In rare cases one or two masses only may be present, growing out of the substance of the left lobe of the liver, causing a large tumor below the sternum. The nodules are usually hard, but sometimes may be soft and even fluctuate. After emaciation becomes marked the nodules can be seen as well as felt near the surface of the skin, and their number distinctly made out. The abdomen is distended.

The liver is movable with inspiration. Progressive enlargement can be noted while under observation. The enlargement can be well defined by percussion, and, while the surface is irregular, the general shape of the dulness corresponds to that of the liver. The increased size and inflammation of the capsule cause a sensation of weight in the hepatic region and pain which may be intermitting in character. The nodules may be tender on palpation. The superficial veins are enlarged.

In not every instance do we find enlargement. In some cases the cancer is associated with cirrhosis of the liver, or may itself be of a nodular type, and in the course of the disease undergo shrinkage. The liver is then normal or diminished in size, as indicated by percussion.

The symptoms that attend cancer are: 1. Jaundice, which is not very deep unless the common duct is affected. 2. Ascites, which is always present in the atrophic forms, but may be absent when the liver is enlarged. 3. The general symptoms are those of rapid emaciation, prostration, and, in some instances, moderate fever. Fever attends the rapidly-growing cases. It is usually continuous, but may be intermittent, especially if there is suppuration or suppurative inflammation of the ducts. It is a well-known fact that gallstones are of common occurrence in patients suffering from cancer in any location whatever. The symptoms of biliary calculus or of obstruction may attend those of secondary cancer of the liver, and the stone has an etiological significance.

In many instances secondary cancer of the liver may be present without symptoms to attract attention to this organ during life. If cancer in certain other regions has continued for the usual period of time, it is almost certain that at the autopsy cancer of the liver will be found to be present.

Diagnosis. The diagnosis of cancer of the liver is not difficult when the changes in the liver can be made out on palpation and percussion. In rare instances, in which the liver is smooth, it may be mistaken for fatty or amyloid liver. A definite cause can usually be assigned for

the latter, while the occurrence of jaundice, the rapid increase in size of the liver, and the general symptoms of the cancerous cachexia indicate cancer of the liver. The *syphilitic liver* with irregular gummata may cause serious doubt; the history of the case and other signs of syphilis aid in the diagnosis. Locally the condition may exactly simulate carcinoma. The jaundice, however, is not so frequent in occurrence, or so deep in syphilitic gummata; the cachexia does not ensue, but the therapeutic test may be essential in order to make a diagnosis.

In *hypertrophic cirrhosis* of the liver the jaundice is deep and the liver enlarged; but there is little wasting or anæmia. The surface of the liver is smooth; there are certainly no bosses, and the organ is painless. Ascites is more common in cirrhosis; the patient is usually affected earlier in life than in cancer.

In a large growing cancer one or two of the nodules may suppurate and simulate *abscess of the liver*. Abscess follows a definite cause usually, and occurs in middle life; cancer is secondary to disease in other organs and occurs usually in late life. The results of aspiration differ in each. Moreover, a history of dysentery, the occurrence of pain, of profound anæmia, of pronounced hectic fever with irregular enlargement of the liver, but without jaundice or cachexia, point to abscess.

Cancer of the liver may be simulated by cancer of organs in close proximity to the liver, as the *pancreas*, the *pyloric* end of the stomach, or the *colon*. In addition to the usual symptoms of pyloric cancer, it will be found that jaundice occurs late. Cancer of the pyloric end is not movable with respiration unless it becomes adherent to the liver. *Cancer of the omentum* and *colon* are not modified by respiration. The percussion-note over them is different; they frequently extend beyond the liver-confines and are associated with symptoms of obstruction of the bowels. *Fecal accumulation* in the transverse colon must not be mistaken for cancer of the liver. The large masses adjacent to the liver may closely simulate cancerous nodules. In doubtful cases the colon should be emptied. Cancer of the liver and hydatid disease must not be confounded. The tumor in *hydatid disease* is usually single; it is large, and may fluctuate or yield the hydatid fremitus. It causes irregular enlargement of the liver, when the tumor presents in the epigastrium or along the margin of the ribs. It is painless. Aspiration yields the characteristic hydatid fluid.

Cancer of the bile-ducts cannot always be distinguished from cancer of the liver. Jaundice early in the course of the disease, in a person who has had gallstones, followed by enlargement of the liver and gall-bladder, in the absence of primary disease elsewhere, suggests cancer of the gall-bladder or ducts. This is more or less confirmed if the smooth and painless gall-bladder becomes hard, irregular, and tender on pressure. *Cancer of the pancreas* also presents difficulties; a tumor in the mid-costal region, however, with vomiting and the early development of jaundice, *before* the liver has become enlarged or nodular, and associated with other characteristic symptoms, such as intestinal dyspepsia and fatty stools, points to the pancreas as the primary seat of the disease.

Hydatid Disease of the Liver.

Hydatid disease is comparatively rare in this country, but, in my own experience at least, it is undoubtedly increasing in frequency. Without any increase in the opportunities for observation, I have seen seven cases within the last two years, compared to the same number during the five previous years. The disease occurs in people who live with dogs. It may occur at any age, but is most common in adult life. It is very rare before the fifth year.

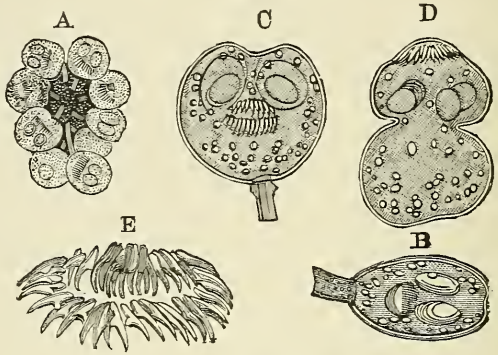
The symptoms are *local*, depending upon the size of the *tumor*. Small cysts may be present without any disturbance. Large and growing cysts cause signs of tumor, with great increase in the size of the liver. The physical signs depend upon the situation of the tumor. It may be found in the median line above the umbilicus, causing a distinct prominence, tense and firm, which sometimes yields fluctuation. Quite often the tumor grows at the suspensory ligament, pushing the diaphragm upward, dislocating the heart, and causing an increased area of dulness in the left upper quadrant. In this position it may simulate a pancreatic cyst or effusion in the lesser peritoneal cavity. If the tumor is in the right lobe, the enlargement of the liver may be upward or downward. The upper border of liver-dulness may begin two or three interspaces higher than normal posteriorly or in the axillary region. If the cysts are superficial, when palpated with the fingers of the left hand and percussed with the right, a vibration or trembling movement is felt, which may continue for a certain time. It is known as the *hydatid fremitus*. It is not always present. The enlargement is painless. Local sensations of weight and dragging may be complained of. If suppuration sets in, there may be a good deal of pain.

The *general symptoms* are negative; the nutrition does not suffer unless the enlarged mass interferes, by its pressure, with physiological acts of digestion and assimilation. If suppuration sets in, the general symptoms of abscess of the liver arise. Jaundice is more common than in tropical abscess. The abscess may perforate into one of the adjacent hollow viscera, or into the pleura and bronchi. It may perforate externally. It may perforate into the pericardium or vena cava, and cause death. If perforation takes place in the biliary passages, obstructive jaundice arises, with secondary suppurative cholangitis. When the cysts rupture, or if they are aspirated, an eruption of urticaria may break out. This is not of diagnostic significance, except that it may point to rupture of the cyst.

Diagnosis. The diagnosis is not difficult. The occurrence of irregular, painless enlargement of the liver without general symptoms is significant. If fluctuation is detected, or the fremitus, a more positive conclusion can be reached. When suppuration takes place the symptoms are like those of abscess of the liver. Hydatid disease is to be distinguished from *syphilitic hepatitis*, in which the enlargement is hard and irregular, and does not fluctuate. Sometimes the symptoms resemble cancer, but the age of the patient, the presence of jaundice, and the extreme emaciation and cachexia indicate that affection rather than hydatid disease. *Enlargement of the gall-bladder* containing a mucoid

fluid, in which fluctuation can be detected, may simulate hydatid disease. The enlargement, however, may be preceded by conditions which cause obstruction of the cystic duct. The gall-bladder is movable. In some instances there may be resonance between it and the liver. It is usually of a pyriform or oblong shape. In *hydronephrosis* the symptoms

FIG. 202.



Human echinococci. (From FINLAYSON, after DAVAINE.)
A, a group of echinococci, still adhering to the germinal membrane by their pedicles. × 40.
B, an echinococcus with head invaginated in the body. × 107.
C, the same compressed, showing the suckers and hooks of the retracted head.
D, echinococcus with head protruded.
E, crown of hooks, showing the two circles. × 350.

of a localized cyst are present. It does not move with respiration, as in hydatid disease; it is attended by symptoms of renal disease; exploratory puncture is sometimes necessary to establish a diagnosis. A hydatid cyst is frequently confounded with *pleural effusion* of the right side, for there may be all the physical signs of effusion at the right base. The

FIG. 203.

Hooks from tænia echinococcus. × 350.

distinction can be made by the character of the line of dulness. In hydatid cyst, as Frerichs points out, it is a curved line, the greatest height of which is found in the scapular region. It is not difficult usually to distinguish hydatid cyst from other forms of painless enlargement. In *fatty* and *amyloid* disease the enlargement is uniform. Both

occur more commonly in individuals of previous ill health, whereas hydatid disease occurs in healthy individuals.

An absolute diagnosis of hydatid disease is based upon the results of *exploratory puncture*. When this is made over a tumor, or the centre of dulness, if it is due to hydatid disease, a clear fluid, slightly opalescent, is withdrawn. The fluid is of a specific gravity of 1005 to 1009; it is of neutral reaction, does not contain albumin, but contains chlorides and sometimes traces of sugar. Hooklets may be found in the clear fluid.

Diseases of the Gall-ducts.

Pain and *jaundice* are symptoms of disease of the biliary passages. *Pain* may be constant or paroxysmal. If it occurs in mild degree, with tenderness and with jaundice, it is probably due to *catarrh of the biliary passages*. If severe, and in paroxysms with or without jaundice, it is due to *gallstones*.

Inflammation of the Bile-ducts. This is due to inflammation and obstruction of the terminal portions of the common bile-duct. But few words are necessary, as it has been referred to frequently in speaking of jaundice. The symptoms are those of moderate jaundice, occurring coincidently with or following in a few days upon an attack of acute gastritis. The disease may occur in *epidemic* form.

Gallstones. Gallstones form in the biliary passages, and may remain there without creating symptoms, or they may, by the efforts to pass them, cause attacks of pain called *hepatic* or *biliary colic*, after which the stone may pass into the intestinal tract without further hepatic symptoms. It may become *impacted* in the biliary canal and set up catarrhal or suppurative inflammation, which in turn may be followed by stricture. Gallstones usually form or at least show signs of their presence after the age of forty years, most frequently in women and in people who have led a sedentary life and partaken of rich and indigestible food. Individuals in different generations of the same family may be predisposed to them.

Hepatic Colic. The passage of gallstones may be attended by a slight amount of pain only, which, unless in the right upper quadrant, would pass for an attack of simple indigestion. In the large majority of cases the pain is severe. The attack may be preceded by biliousness or indigestion for twenty-four hours, and moderate pains or a sense of weight and fulness in the liver. It frequently follows the taking of food. Ringing in the ears, disturbance of vision, or undue flushings are said to precede it in some instances.

The attack may be sudden. The patient is seized with *pain* along the margin of the ribs of the right side, or there may be pain above the ribs, over the liver, and in the right shoulder at the same time. From the hepatic region it extends to the median line. Very frequently the pain begins and continues in the epigastrium. It may be most pronounced in this locality from the first. The pain is intense and paroxysmal. The patient is doubled up in agony. It causes more or less *collapse*. The pulse increases. *Vomiting* usually occurs at the same time, consisting first of the contents of the stomach, and then of a

yellowish, bile-stained fluid. The vomiting may be extreme, so that the patient is tormented by the pain, the retching, and vomiting. The attack sometimes disappears as suddenly as it occurred, or wears off gradually. When most severe, symptoms of shock follow. The bowels are not disturbed during the attack. The urine may become suppressed; it is usually high-colored, and after the attack may contain bile.

At the time of the attack there is considerable tenderness below the xiphoid cartilage and in the hepatic region. The *tenderness* is more marked on deep pressure in the gall-bladder region and to the right of the mid-clavicular line, at the margin of the ribs. The epigastrium may be slightly swollen. The tenderness persists after the attack, and the stomach may be weak or irritable for some time; pain, however, usually disappears at once. The attack may recur frequently until the stone has been passed, so that in twenty-four hours the patient may have a dozen or more paroxysms. After the attacks have subsided light *jaundice* may supervene, which usually does not continue more than a week at the furthest, during which there are also symptoms of mild gastritis. (See Intestinal Colic.)

In some instances a chill precedes or immediately follows the pain, after which the temperature rises. After the paroxysm subsides the fever disappears rapidly, being followed by profuse perspiration. If the gallstones have set up catarrhal inflammation, moderate fever may continue for a few days. (See Fever in Obstruction.)

During any paroxysm of hepatic colic it is desirable to determine whether or not a gallstone has been passed. This can only be done by placing the feces in a sieve and pouring water upon them until they dissolve. Instead of gallstones, dark-colored granular bile, which has become inspissated, is sometimes seen in the movements. Bile in this form gives rise to as much pain, according to Harley, as true biliary concretions. If the stone is not passed, it may fall back into the gall-bladder and cause no further symptoms for a time, or become impacted in the ducts. The impaction may be such that no obstruction is caused by its position, the bile being forced through or around it; or complete obstruction may take place. (See Jaundice.)

Obstruction of the Common Duct by Gallstones. (*a*) In addition to jaundice paroxysms of chill, fever, and sweat occur, with catarrhal inflammation of the biliary passages. (1) The paroxysms resemble intermittent fever; (2) the jaundice may continue for years and deepen after each paroxysm; (3) hepatic colic may occur with the paroxysm; (4) the health fails but slightly. The paroxysms may occur daily or only once a week, or they may be tertian and quartan in type. The pain is referred to other situations than the gall-bladder or the epigastrium. It is often relieved by vomiting or by certain positions of the body. The jaundice may be intermittent or remittent. On account of the obstruction in this situation the liver becomes enlarged. It is firm and smooth on palpation. The enlargement, as determined by percussion, is uniform. The gall-bladder is not enlarged. Fenger's thorough studies show that the *intermittent phenomena* are due to ball-valve action of a single stone. He also points out that emaciation is of common occurrence. (*b*) Gallstones may cause *suppurative inflammation*

of the biliary ducts, just as suppuration of the gall-bladder may ensue. The symptoms, both general and local, are pronounced. The fever may be intermittent, but is more likely to be remittent; jaundice is present, but it is constant in its intensity. The local signs of enlargement and tenderness are made out. The patients die of exhaustion or septicæmia. Sometimes the gall-bladder ruptures into the stomach or colon, and temporary abeyance of the symptoms may result.

The Accidents of Gallstones. While these effects of the presence of stones in the biliary passages may rightly be considered as accidents, nevertheless their occurrence is so common as to be part and parcel of the history of gallstones. As accidents, we have most commonly the occurrence of localized peritonitis, which leads to dislocation of the gall-bladder, constriction of the duodenum, with secondary dilatation of the stomach; we also have the formation of biliary fistula, with passage of the gallstone into the contiguous organs or channels. The stone may ulcerate into the gall-bladder from one of the ducts, may perforate the portal vein, or may perforate into the abdominal cavity—the most frequent accident. Perforation also takes place into the duodenum, into the colon, and, rarely, into the stomach. Such perforation can only be inferred from its secondary effects: (1) An attack of gallstones; (2) local inflammation with fever; (3) the occurrence of peritonitis, or the discharge of pus by the bowels, or by vomiting. That it is due to gallstones is proved in those rare instances in which the stone is passed per rectum. Often it may be impacted in the intestinal canal, causing symptoms of acute obstruction, or in the rectum, causing local tormina and tenesmus. The perforation, however, occurs in other directions. Sometimes fistulous connection is formed between the gall-bladder and the urinary passages, calculi and pus being discharged in the urine. In other instances fistulæ between the bile-passages and the lungs are formed. The bile is coughed up and expectorated, sometimes with small calculi. In the most common form ulceration proceeds toward the surface, with formation of cutaneous fistula. After the fistula has opened externally gallstones in large numbers may be passed. If not, the cause of the fistula must be determined by the history and the results of investigation by probe, due attention being given to the condition of other organs.

Enlargement of the Gall-bladder. (Plate XLII., Fig. 2.) *Enlargement* of the gall-bladder may be due to obstruction in the *cystic duct.* The liver is not secondarily affected. The enlargement is noted at the edge of the liver in the usual situation, and may gradually increase to an enormous extent, so that it has been mistaken for an ovarian cyst. The gall-bladder is often quite movable, and on account of its location and movability, as well as its long shape, has been mistaken for a floating or movable kidney. If the gall-bladder is not too large, it can be felt as a rounded or pyriform mass when the hand is placed along the margin of the liver, becoming more marked when the patient takes a full breath. The enlargement is not attended by any other symptoms except mechanical ones, unless the contents of the gall-bladder are purulent. In obstruction with simple enlargement the fluid of the gall-bladder, should aspiration be performed, is thin, of a mucoid nature,

and alkaline in reaction. It may contain cholesterin-plates, and sometimes blood. It must be distinguished from the fluid of a hydatid cyst.

Simple *enlargement* of the gall-bladder must be distinguished from enlargements due to inflammation. (1) *Acute phlegmonous inflammation* of the gall-bladder may take place, attended by localized pain and tenderness, by high temperature, extreme prostration, and the rapid development of the typhoid state. Peritonitis rapidly ensues. It cannot be distinguished from other forms of acute inflammation in the same region, unless there was (*a*) a history of gallstones; (*b*) tumor of the gall-bladder before the attack developed. (2) *Suppurative inflammation* of the gall-bladder may occur from gallstones and in infectious diseases. The colon bacillus, the diplococcus of pneumonia, and the typhoid bacillus give rise to infectious inflammation of the gall-bladder. The enlargement takes place suddenly and may increase, the tumor becoming tender and painful on palpation. The direction of growth is toward the umbilicus. The general symptoms are those of suppuration. Hectic fever or markedly remittent fever occurs, and, unless surgical relief is given, peritonitis ensues from infection or from rupture. This complication may be suspected from the occurrence of collapse and increase of the local symptoms.

Either of the above forms of cholecystitis is attended by pain in the region of the gall-bladder or in the epigastrium or even as low down as the region of the appendix. The pain is severe and paroxysmal. The symptoms of bacterial infection, of which vomiting and fever are the most prominent, rapidly follow. The symptoms simulate appendicitis, intestinal obstruction, and pancreatitis.

Enlargement, or tumors of the *gall-bladder*, usually due to cystic obstruction, as previously mentioned, may be mistaken for floating kidney, for tumor of the pylorus, and for ovarian cyst.

Tumors of the gall-bladder from any of the above-mentioned causes are recognized by their *position* and *shape*, and by the *character* of the tumor. The *position* varies. The usual site is in the gall-bladder region, but it may extend as low as the groin, or may be so large as to distend the ribs and fill almost the entire abdominal cavity. If, however, the case has been under observation from the beginning, the tumor must have been found originally in the gall-bladder region. This region corresponds to the point of intersection of the border of the ribs by a line drawn from the acromion process of the right shoulder to the umbilicus, or in the direction of the foramen of Winslow. The tumor grows from this point toward the umbilicus in nearly all the cases. It can be recognized by its shape, which is pyriform, globular, or conical. The *character* of the tumor varies. It is usually tender and firm, but elastic on pressure, and movable. Fluctuation may often be detected. The septic gall-bladder is symmetrical and resistant to the touch. If the enlarged gall-bladder contains calculi, they may be felt as small, hard masses, which cause a grating sensation, to be transmitted to the finger. On aspiration, if the cystic duct is obstructed, the mucoid fluid previously mentioned, or pus, is withdrawn. If the common duct is obstructed, bile will pass through the trocar.

The enlargement must be distinguished from tumors of the liver,

stomach, duodenum, pancreas, or lymphatic glands. Tumors of the liver are usually due to *carcinoma*. They are multiple, associated with enlargement of the liver, with jaundice, ascites, enlargement of the spleen, and emaciation. Tumors of the *stomach*, *duodenum*, and *pancreas* are in a different position, and are attended by functional disturbance of the respective organs from which they spring. An *abscess* of the liver, if purulent, may simulate enlargement of the gall-bladder. If the abscess can be palpated, an area of induration is first felt, followed afterward by softening and fluctuation of the swelling. In judging of the true nature of the tumor we must bear in mind the causes of abscess. In *hydatid* disease the tumor develops slowly; it is painless; it may yield fremitus, and, if movable, the course is slow and not attended by general symptoms. *Multilocular hydatid disease* can rarely be distinguished save by the difference in position of the tumor. It is nodulated, hard, and tender, but is associated with jaundice, ascites, œdema of the legs, enlarged spleen, and great emaciation and prostration, with rapid decline. A *syphilitic gumma* in the liver may occupy the region of the gall-bladder. It can usually be made out as continuous with the liver-structure. It is tender and painful, but irregular; other signs of syphilis, or a history of the infection and of symptoms of a primary and secondary period, will aid in the distinction of the disease.

Floating Kidney. The gall-bladder is larger and fixed at one end, whereas the entire kidney is movable. The gall-bladder may fluctuate, and is associated with symptoms of hepatic disease. On the other hand, the well-known symptoms of floating kidney, the shape of the tumor, the sensation of nausea induced by palpation, point to the renal origin of the mass. *Tumors* of the *kidney* must be distinguished, such as sarcoma, hydronephrosis, and pyonephrosis. 1. There may be changes in the urine. 2. In renal tumors the intestine is in front of some portion of them, or a zone of resonance is found between the liver-dulness and the tumor. 3. Renal tumors are fixed. They may, as in hydronephrosis, come and go, preceded by attacks of renal colic and attended by *anuria*. From *ovarian* or *uterine tumors* the diagnosis must be made by examination of the genital organs, although with the former there is often difficulty.

Enlargement of the gall-bladder on account of calculous obstruction must be distinguished from enlargement due to *cancer* of that organ. This is often difficult and cannot be done without having the patient under observation for a long period of time. Cancer of the gall-bladder is usually primary. It may begin in the gall-ducts. In the larger number of cases it occurs in patients who have had gallstones. It is found most frequently in females, and after the fiftieth year. Tight-lacing or pressure around the abdomen may predispose to it. The symptoms are pain, jaundice, emaciation, cachexia, and the presence of a tumor. The pain is localized and lancinating in character. Jaundice occurs in 70 per cent. of the cases, and gradually increases in intensity. The tumor is situated in the gall-bladder region, to the right of the umbilicus. It is hard or firm, painful, and the seat of tenderness. The tumor is fixed. Sometimes the disease is found in the cystic duct,

and then the gall-bladder is enlarged. As the history of gallstones is of frequent occurrence in both instances, it is impossible to distinguish the two forms of obstruction causing enlargement, save that in carcinoma the emaciation and cachexia may point to the true nature of the case. In tumor of the gall-bladder due to cancer the secondary effects on the liver are usually more marked than in tumor from other causes. The liver enlarges and its surface becomes irregular or nodular.[1]

Diseases of the Spleen.

Topography of Spleen. (Plate XXXV.) The spleen lies in the left upper quadrant beneath, and in contact with the diaphragm above, and below with the tail of the pancreas, cardiac end of the stomach, and suprarenal capsule. It extends transversely between the upper border of the ninth rib and the lower border of the eleventh rib, and from the middle axillary line posteriorly toward the spine.

Palpation. An enlarged spleen usually retains the normal shape. The direction of the enlargement is downward and inward. It is accessible to *palpation* in proportion to the degree of enlargement and of relaxation of the abdominal walls. It is movable with respiration. It cannot be said to be enlarged unless the edge is palpable at the end of deep inspiration, notwithstanding there may be increased dulness in the lower axillary region. When moderately enlarged, the smooth, blunt, rounded anterior surface and sharp edge of the spleen can be felt at the margin of the ribs, in deep inspiration; when the enlargement is great, as in *leukæmia*, the organ can be grasped with both hands, and its hilus clearly mapped out. The same thing can be done in the rare instances of *floating spleen*, but here a knee-chest position will favor successful palpation. The posterior border of an enlarged spleen can usually be made out by passing the hand backward over the resisting organ. At its posterior border a non-resisting space can be detected between the border and the mass of lumbar muscle. In children it is always easy to define this border. No such space exists in renal enlargements. The existence of this space and the direction of enlargement of the spleen are due to the costo-colic fold of peritoneum (Jenner). In splenic leukæmia the spleen may be larger after a meal, yield a creaking fremitus on palpation, a murmur on auscultation, and may even pulsate. The spleen may also lessen in size after diarrhœa or free hemorrhage. As it lies entirely behind the ribs, it does not, of course, admit of palpation when the size is normal.

Percussion. (Plate XVI., Fig. 2.) Being a solid body it gives a dull sound on percussion, contrasting with pulmonary resonance above, intestinal tympany below, and stomach tympany anteriorly. Posteriorly and below its dulness merges into that of the lumbar region and kidney. The upper posterior portion is hidden behind the diaphragm and overlapping lung, and hence is not accessible to percussion. Practically, therefore, the normal splenic dulness extends between the ninth and eleventh ribs, in the middle and posterior axillary lines, the spleen being there in contact with the ribs.

[1] Musser: Trans. Assoc. Amer. Physicians, vol. iv., 1889.

In percussion of the spleen the patient should lie on his right side. Beginning from above downward we percuss gently until pulmonary resonance is succeeded by dulness; then anteriorly, proceeding toward the axilla, until stomach tympany yields to dulness. In the same way, percussing from below upward, the line is reached where intestinal tympany gives way to dulness.

Splenic dulness may be encroached upon by the stomach or colon distended with gas, or its dulness may appear increased through distention of the stomach and colon with solid matter, or by a left pleural effusion, or left basal pneumonia. The spleen may also be pressed upward by ascites or by a large abdominal tumor, so that its normal dulness is much lessened.

If the ligament which holds it in place becomes relaxed, the spleen may become floating. According to Stintzing, a floating spleen is increased in density, is generally enlarged, and is recognized by its form (notch, etc.), by being movable to and fro, and by the absence of splenic dulness in the normal position, and its reappearance when the spleen is replaced.

Enlargement of the Spleen. *Enlargement* of the spleen may be acute or chronic. *Acute enlargement* occurs in certain infectious diseases, particularly typhoid fever, typhus, smallpox, relapsing fever, scarlet fever, diphtheria, epidemic cerebro-spinal meningitis, the malarial fevers and meningitis, and in diseases with blood-poisoning, as septicæmia, puerperal fever, and erysipelas.

A rare cause of enlargement is *acute splenitis*. Generally, it is the result of emboli lodged in the spleen and starting from an endocarditis. The area of splenic dulness extends rapidly, and there is local pain and tenderness on pressure, increased by coughing and deep inspiration; other symptoms are fever, nausea and vomiting, and occasionally delirium. If, as frequently happens in splenitis, emboli lodge in the kidneys also, the urine will be albuminous and bloody. If suppuration ensues, the fever becomes hectic, and the spleen continues to increase in size. *Splenic abscess* may, however, remain latent until rupture occurs.

Chronic enlargement of the spleen occurs as hypertrophy and as the result of amyloid disease, leukæmia and pseudoleukæmia, chronic malarial poisoning (ague-cake), syphilis, hydatid tumor, and cancer. Enlargement is greatest in leukæmia and in ague-cake. The spleen in well-marked cases of these affections may reach to the umbilicus and even beyond, filling up the hypogastrium and extending to the right iliac region, measuring thirteen or fourteen inches in length and half as much in breadth, and proportionately increased in thickness.

Primary splenic enlargement may occur (1) without local or general symptoms; (2) anæmia, profuse hemorrhages, and brown pigmentation of the skin may be present with the enlargement. Hemorrhages are usually limited to the gastro-intestinal tract. The anæmia is of a chloritic type, and there is no change in the leucocytes. (3) Enlargement may be associated with cirrhosis of the liver and jaundice, with gastro-intestinal hemorrhages and with ascites. This affection is commonly known as Banti's disease. The blood changes are almost a counterpart

of those in progressive pernicious anæmia. It may be confounded with chronic inflammation of the peritoneum, giving rise to ascites and associated with mediastinal pericarditis.

Diagnosis of Enlargement of the Spleen. (Plate XLIII., Figs. 1 and 2.) Enlargement of the spleen can be distinguished from enlargement of the left *kidney* by the greater movability of the spleen. 1. The spleen does not extend as far back toward the spine as the kidney, so that the fingers can be thrust behind its posterior border, and, if the other hand grasps the anterior edge, the organ can be moved backward and forward. Splenic dulness extends to the ninth rib or higher. Kidney-dulness has no thoracic area, but reaches to the spine (lumbar). 2. Again, the spleen is more movable with respiration than the kidney is. 3. The spleen falls further toward the median line, when the patient is in the knee-chest position, than does the kidney. 4. An enlarged kidney has the colon in front of it, and hence its dulness is obscured by the tympany of the bowel. 5. The shape of an enlarged kidney is more globular than that of the spleen. The anterior surface of the latter is smooth and rounded, but at its junction with the flat posterior surface there is a sharp edge. 6. Pain in renal disease often shoots down the ureters and into the testicles. In diseases of the spleen the pain is generally localized to the splenic region, and may shoot into the left shoulder. 7. Result of examination of the urine will often make clear that the disease is renal, or, by its negative result, will point to the splenic origin of the tumor.

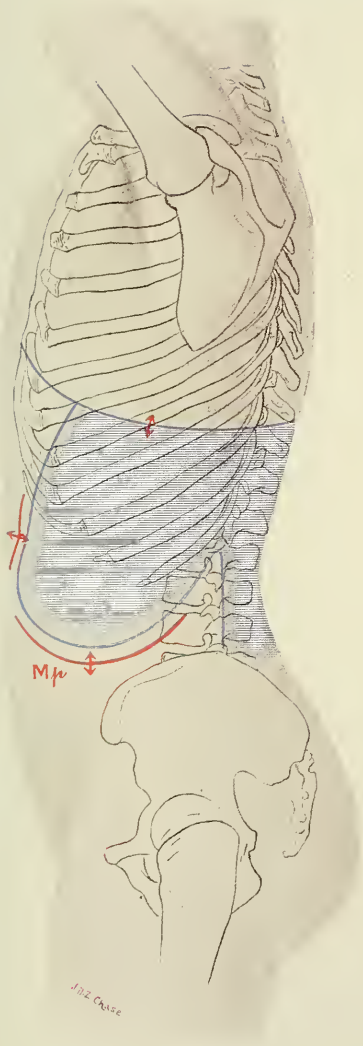
It is sometimes difficult to demonstrate enlargement of the spleen when the liver, and particularly the left lobe, are enlarged. Careful palpation reveals the edge of the spleen, which descends further than the liver in full inspiration. Having found the anterior edge, pressure with the other hand posteriorly will bring the spleen forward, which would not occur if the suspected enlargement was the left lobe of the liver.

The diagnosis of *splenic leukæmia* (Plate XLIV., Fig. 1) rests principally upon the blood-condition, particularly upon the existence of a marked increase of white blood-cells. Red cells are decreased, and altered forms are present. In addition to characteristic blood-changes there is a great disposition to hemorrhages; dropsies and priapism are common; and, in later stages, fever, diarrhœa, great weakness, and grave complications, such as pneumonia. Hemorrhage in splenic leukæmia occurs from the nose, bowel, stomach, gums, or kidney. It may also be subcutaneous, intermuscular, cerebral, or retinal.

Regarding the diagnosis of *splenic hypertrophy* (ague-cake) in *chronic malarial affections*, Osler says: "The history of malarial cachexia, the absence of lymphatic enlargement, and the blood-condition will usually be sufficient for the purpose of a diagnosis. Great increase in the white blood-corpuscles is not often seen in the chronic splenic tumor of malaria; indeed, they may be much diminished in number. Toward the end in very chronic cases the clinical picture may be very similar; the large abdomen, possibly ascites, dropsy of the feet, and irregular fever may resemble closely splenic leukæmia, and the absence of an increase in the colorless corpuscles may be the only marked difference."

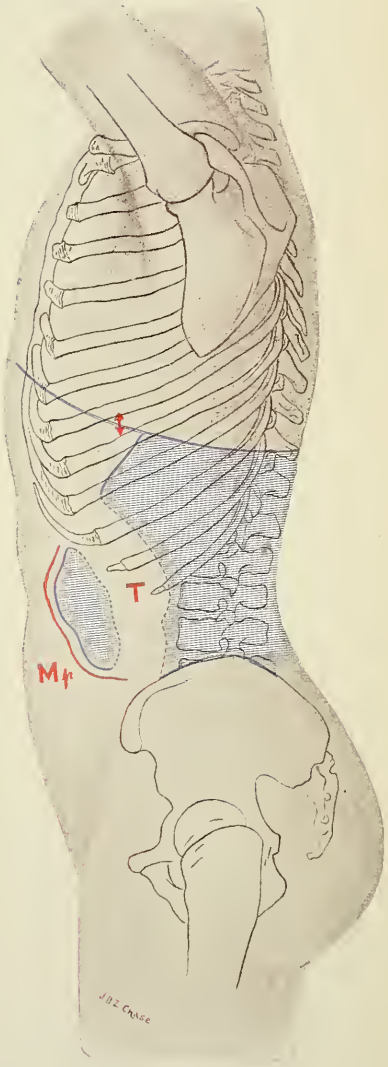
PLATE XLIII.

FIG. 1.



Enlargement of the Spleen.

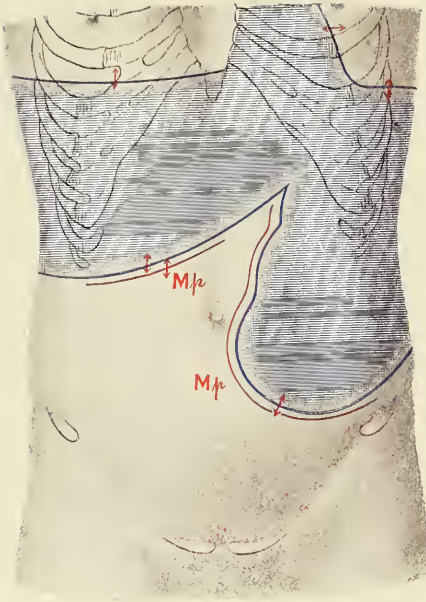
FIG. 2.



Tumor of the Left Kidney.

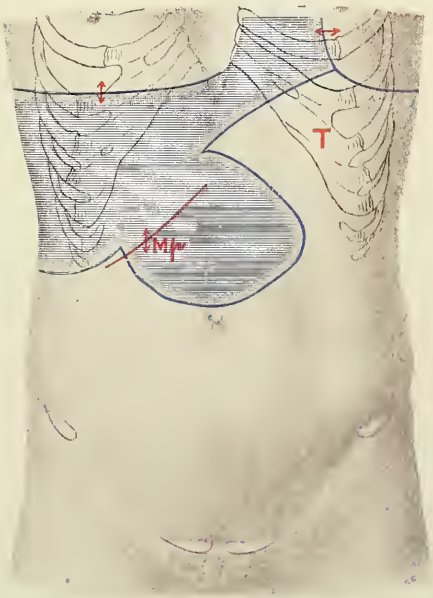
PLATE XLIV.

FIG. 1.



Leukæmia—Enlarged Liver and Spleen.

FIG. 2.



Cyst of the Pancreas.

Amyloid spleen, with enlargement of the organ, occurs in conditions of prolonged suppuration, especially when the bones are involved, and in chronic phthisis and syphilis. The spleen is enlarged, hard, and painless. The enlargement is rarely great enough to produce distress on that account, and it is so commonly associated with a similar condition of the liver and kidneys, if not of other organs, that any constitutional symptoms produced by the spleen are apt to be masked by those produced by other organs.

Hydatid tumor of the spleen rarely causes any symptoms except when it becomes very large; then it may give rise to discomfort and a dragging pain in the left hypochondrium. But hydatid tumors of the spleen are only exceptionally very large; when large enough to admit of palpation, and when the tumor is situated anteriorly or projects from the lower border or from beneath the organ, the detection of fluctuation, the withdrawal of the characteristic fluid by aspiration, and possibly the hydatid fremitus, will establish the diagnosis, when taken in connection with the gradual development of the tumor and exposure to possible infection. In the absence of physical signs of a cyst the nature of the tumor can only be suspected from the habits of the patient or his place of residence. Suppuration of the sac may be brought about by injury or rupture into the adjacent cavities, with grave if not fatal results.

Inherited syphilis and *chronic syphilis* are accompanied by enlargement of the spleen. They cause a chronic interstitial inflammation. The enlargement is not very great, and does not present characteristic features.

Malignant tumors of the spleen are very rarely primary. The diagnosis must be made by noting malignant disease elsewhere, the very rapid enlargement of the spleen, with possibly nodules scattered over its surface, and the presence of cachexia and the usual constitutional signs of a malignant disease.

In young children enlargement of the spleen is not uncommon. It is found associated most frequently with rickets, syphilis, and malarial poisoning, and has been attributed to each of these diseases. In the London *Lancet*, April 30, 1892, Dr. J. W. Carr analyzes thirty cases, and comes to the conclusion that the enlargement of the spleen is due to *splenic anæmia*, the essential cause being unknown. Rickets, syphilis, and ague are found as passing causes only, since the disease is found in some cases where these causes can be excluded. According to this author, the disease is extremely rare in children older than two and one-half years. The spleen is more readily palpated in children than in adults. It is also more movable, and hence by bimanual palpation it can be more easily brought forward to the median line.

Diseases of the Pancreas.

Just as the functional activity of the pancreas is separated with difficulty from that of other functionally related organs, so the aberration of such activity is discerned with the greatest difficulty. As the physiology and pathology are blended so the symptoms are intermingled.

The pancreatic secretion aids in intestinal digestion, particularly in emulsifying fats, hence symptoms due to disturbance of this function are looked for, and it is, in a measure, true of all cases of pancreatic disease that there is some *intestinal indigestion*. For the purpose of determining whether the function of digestion of fats has been modified the patient with suspected pancreatic disease is given fats in some form, and the stools are watched. If fat is passed in the stool in the amount taken by the mouth, without being broken up, or emulsified, it is held as proof that disease of the pancreas is present. While *fatty stools* may be indicative of pancreatic disease, the absence of fat in the stools, in patients who are fed upon it, cannot be considered to exclude disease of this organ, for, notwithstanding its absence in a large number of instances in which the experiment was tried, the pancreas was found to be the seat of grave disease. Sugar has been observed in the urine in many cases in which the pancreas was the seat of the disease. In fact, *glycosuria* has been attributed to pancreatic disease in cases of grave diabetes. This symptom, however, is not constant in pancreatic lesions. Three classes of symptoms—intestinal indigestion, fatty stools, and glycosuria—are, therefore, not diagnostic of pancreatic disease, but only afford presumptive evidence of its presence.

Tumor of the Pancreas. The most striking symptoms of disease of the pancreas, apart from those due to the morbid process, as suppuration or cancer, are those due to a *tumor* pressing upon surrounding structures. It may press upon the gall-duct, causing jaundice. From its situation in the epigastric region it may resemble an aneurism, or a tumor of the pylorus or of the transverse colon. Tumors of the pancreas are usually due to *cancer*. This is usually of the scirrhous variety, and generally primary. The enlargement cannot be distinctly made out unless the patient is very much emaciated. When it has advanced considerably it may simulate aneurism, but is distinguished by the difference in the character of the pulsation. In aneurism the pulsation is distensile, in disease of the pancreas it is an up-and-down movement; the hand is lifted with each pulsation of the aorta. Tumor of the pylorus is excluded largely because of the more superficial position of the mass, because of its association with pyloric obstruction, and with less frequent jaundice than occurs in disease of the pancreas. A pyloric tumor is more movable and may change position after the stomach is inflated by gas or distended by fluid. Examination with the patient on the hands and knees may aid in the distinction between the two. In a tumor of the transverse colon its nearness to the surface and its movability, its association with more or less constipation, and the occurrence of intestinal hemorrhage, are of diagnostic significance.

The general symptoms of the cancerous cachexia; the occurrence of intestinal indigestion, or of fatty stools; the gradual onset of jaundice; the occurrence of deep-seated epigastric pain; an immovable tumor, with glycosuria, make a symptom-group very characteristic of *cancer of the pancreas*.

When the patient is on a milk-diet an examination of the feces will show that an excess of the ingested fat is lost—in short, that there is deficient pancreatic digestion with lessened absorption of fat.

Hemorrhage. We owe to F. W. Draper and Prince our knowledge of hemorrhage into the pancreas. Since they have published the results of their labors the affection has been frequently recognized. The attack comes on suddenly in perfect health, and usually terminates life in a short period. Nothing in the occupation or conduct of the patient at the time is known to favor the development of the hemorrhage. He is seized with severe pain, which is localized in the upper part of the abdomen. It increases in severity, and may intermit like colic. Nausea and vomiting take place almost at the same time. The vomiting becomes obstinate. Extreme depression rapidly sets in and the patient becomes anxious and restless. Collapse ensues in a short time. The extremities become cold and the forehead is covered with sweat. The pulse increases in frequency, and rapidly diminishes in strength. It soon becomes imperceptible. The pain and vomiting call attention to the upper abdomen. It is tender on pressure; the tenderness may extend throughout the entire upper half of the abdomen. Tympanites may develop. There is constipation in many cases. The temperature remains normal, or becomes subnormal. The pain, the vomiting, the anxious and restless state continue without relief.

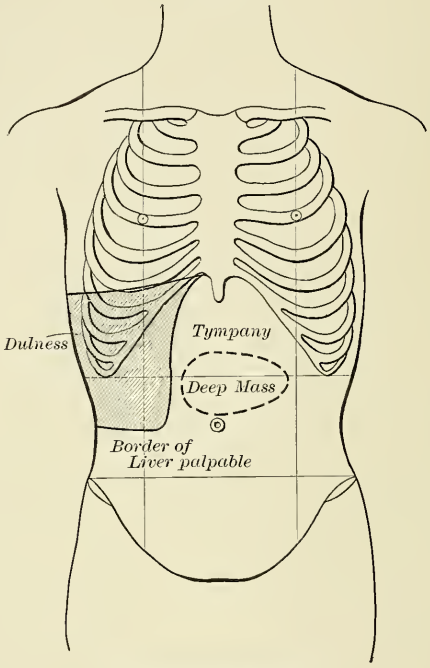
From the above group of symptoms it can readily be seen that the diagnosis is obscure. The disease can be taken for perforation of the stomach by ulcer, although the vomiting may not be so persistent and frequent. Intestinal obstruction in the upper portion of the tract presents allied symptoms. The hemorrhagic symptoms, however, are more pronounced in pancreatic hemorrhage. Pallor of the face is sure to ensue. The vomiting is not fecal in character. Constipation can be relieved. It is, however, difficult, and in many cases impossible, to establish a diagnosis. The rapidity of development of the symptoms is of importance. The pain and collapse may be due to rupture of an aneurism of the aorta.

Acute Hemorrhagic Pancreatitis. For our knowledge of this disease we are indebted to Fitz. He collated the facts from the literature, and, adding the results of his own valuable observations, has enabled us to recognize this affection during life. It usually occurs after the middle period of life, although it may occur in early childhood, the youngest patient known to the writer being eight months of age. It is more common in males; in those addicted to alcohol, and in fat subjects. The patient has often been the subject of attacks of indigestion or of epigastric pain or of biliary colic. A blow on the abdomen or injury in the lumbar region appears to have been the exciting cause in a number of cases.

The attack develops suddenly, resembling somewhat hemorrhage of the pancreas. There is violent pain which is at first complained of in the upper abdomen, although it is sometimes general. Nausea and vomiting are present in all the cases, constipation in most of them. The abdomen is frequently the seat of tympanitic distention. In many instances an obscure tumor can be made out in the lower epigastric region. Collapse-symptoms supervene, although fever may occur, the temperature rising to 102°. The cases terminate by the fourth day, even earlier in some cases. The pain and collapse are

probably due to pressure of the effused blood upon the cœliac plexus. The fever is due to a colon-bacillus infection. Violent delirium resembling acute mania and not unlike that seen in atropine-poisoning, occurs in some instances. Symptoms of localized peritonitis arise, and if the patient lives the tumor increases to a considerable size.

FIG. 204.



Tumor of the pancreas.

The symptoms resemble acute *intestinal obstruction*, an irritant *poison*, or *perforation* of the gastro-intestinal or biliary tract. In several instances laparotomy has been performed for the relief of supposed obstruction. The intense pain in the epigastrium, with violent vomiting and distention of the upper abdomen, without a possible cause for obstruction, are favorable to acute pancreatitis. The difficulty of diagnosis, however, is so great that resort to laparotomy is justifiable, in order to determine exactly the nature of the condition. In a most interesting case reported by W. S. Thayer, the diagnosis of acute pancreatitis (confirmed by laparotomy) was based upon the history of previous attacks of pancreatic pain, with fever, vomiting, and collapse, occurring in an adult, who was over-fat and an alcoholic; the exclusion of disease in other organs and the absence of a history of gallstones or gastric ulcer or abscess from other causes; the occurrence of pain; the presence of a deep-seated tumor which gave indistinct signs of fluctuation, which was not movable with respiration, and the dulness of which was not continuous with or of the same character as that of adjacent

solid organs. Epigastric tympany was also a point in favor of pancreatic disease. The accompanying figure indicates the site of the tumor in Dr. Thayer's case.

Suppurative Pancreatitis. Fitz has found that this affection occurs in adults under forty years, more frequently in males. Symptoms continue during several weeks, and may persist for a year. Pain in the epigastrium is complained of, associated with irregular vomiting, the latter persisting in spite of care as to feeding. Fever is irregular in type, and exhaustion ensues. Jaundice, fatty diarrhœa, and glycosuria have been met with in some cases. In a case under my observation obstruction of the portal vein took place, with ascites. The latter was large, and recurred rapidly after tapping. In this patient pain and gastric disturbance were absent. There was no fever. Emaciation, constipation, and a tumor above the umbilicus were present; the emaciation was extreme. The tumor was ill-defined, painless, apparently superficial. Many other symptoms of pancreatic disease pointed out by Roberts were present. Apathy and despondency were marked; bronzing of the face was also present. The patient was a middle-aged man, forty-two years old, addicted to the use of alcohol. He was thought to have cirrhosis of the liver. As happened in this case, the pus may accumulate in the duodeno-jejunal fossa and fill up the cavity of the lesser peritoneum, with more pronounced symptoms of tumor than occur in similar fluid accumulations in the above-mentioned cavity.

Gangrenous Pancreatitis. This may follow later upon hemorrhages into the pancreas. The symptoms are extremely obscure. Symptoms of collapse may occur, following pain, which is of longer duration than in the acute form, or vomiting, which is not so persistent. A patient of mine, upward of sixty years old, suffering from dyspepsia, vomited blood in the course of an illness which was characterized by loss of flesh and weakness. The anæmia became very profound after the gastric hemorrhage, and exhaustion was extreme. There was no marked tumor, but only resistance in the region below the xiphoid. There were dulness and tubular breathing at the base of the left lung. Fever was absent. Death ensued from exhaustion. A small, flat carcinoma was found in the pyloric end of the stomach, but there was no perforation. Gangrenous pancreatitis, with signs of an ante-mortem hemorrhage, was found. The accumulation took place behind the stomach and colon, but in front of the kidney; its outer wall was bounded by the spleen. It was circumscribed above by the diaphragm. Pleuritis and small pulmonary abscesses at the base of the left lung were found.

In some instances the pancreas has sloughed into the bowel, and in two such cases recovery took place after its discharge from the rectum.

Chronic pancreatitis is not recognized during life, although its possible presence must be considered in all cases of diabetes, and in jaundice not otherwise explained.

Cyst of the Pancreas. (Plate XLIV., Fig. 2.) Cysts of the pancreas follow impaction of calculi in the pancreatic duct; sometimes the biliary calculi obstruct the orifice. The symptoms are those of tumor in the upper abdomen which occupies the median position, or is

chiefly on the left side in the upper quadrant. It may fill the abdominal cavity and simulate ovarian tumor. It usually develops slowly, but cases of rapid onset have been described. Fatty diarrhœa is not present. There is a sense of weight and fulness in the epigastrium. The cysts are not really true cysts, but accumulations of pancreatic fluid in the lesser peritoneal cavity.

The signs are those of tumor to the left of the median line, encroaching upon the left lobe of the liver above, and extending almost to the transverse umbilical line. Körte, in a series of sixteen cases, observed that the greatest prominence of the mass was below the navel. The tumor is smooth, and may fluctuate; it is not hard and lobulated. On account of its presence the diaphragm may be arched so that the heart is dislocated upward to the left; the apex is found in the third interspace. It also causes increased dulness behind on the left side, the upper border approaching the angle of the scapula. Exploratory puncture in either instance determines the nature of the fluid, and may determine the diagnosis. Boas does not think the chemical character of the fluid is sufficient to establish a diagnosis. (See Examination of Cyst Fluid, page 367.)

Senn has pointed out that in cysts of the pancreas the complexion is peculiar; it is described as an unhealthy yellow, dirty, or earthy hue. The writer also considers that, in the diagnosis of pancreatic cyst, the history of the case, the location of the tumor, and its relation to other organs are to be considered. The disease occurs in adults, and usually follows traumatism. A blow in the epigastrium is a prominent exciting cause. In some instances it occurs after an attack of so-called biliary colic or colicky pains in the upper abdomen, with vomiting, but without jaundice—a condition characteristic of calculus in the pancreatic ducts. The growth of the tumor in some cases is unusually rapid—a point in favor of its pancreatic origin. It may attain an enormous size, as previously mentioned.

In contrast to cancer, pain is absent. Fatty stools are absent. Previous gastro-intestinal derangement may be ascertained upon inquiry. Diabetes, in this as well as other affections of the pancreas, may be present. The cyst is always found at first in the region occupied by the pancreas, depending somewhat upon the portion of the pancreas from which it originated. It may be below the right lobe of the liver, below the xiphoid, or in the left upper quadrant. In the great majority of cases it occupies the last situation. It displaces the stomach forward and to the right, the transverse colon downward, the diaphragm and the contents of the chest upward. The cyst may be movable in respiration.

Diagnosis. It must be distinguished from cancer of the pancreas or adjacent organs, aneurism, hydatid cyst of the liver, the spleen, or the peritoneum, affections of the retroperitoneal glands, hydronephrosis, cystic disease of the suprarenal capsule, circumscribed peritonitis with exudation, ascites, cystic disease of the ovary. Pain is an important symptom of disease of the pancreas in its more acute manifestations; it must be distinguished from the pain of intestinal obstruction and the pain of perforative peritonitis. The pain is always localized in the

region below the xiphoid, or, in general, is confined to the upper half of the abdomen. It exactly simulates the pain of the affections just described. This resemblance is more pronounced because of the association of vomiting and collapse in obstruction and perforative peritonitis. Pain, although not so intense, but of a colicky nature, attended by diarrhœa or constipation, in some instances with intestinal hemorrhage, may be due to *calculous disease* of the pancreas. Frequently this form of pain can be recognized if other symptoms of pancreatic disease, such as glycosuria, steatorrhœa, and intestinal indigestion, are present.

CHAPTER VII.

DISEASES OF THE KIDNEYS.

The kidneys are affected by disease from several sources. First, the great vascular supply is subject to the alteration which takes place in any large arterial area, either from direct *hyperæmia*, through the influence of the vasomotor nerves (see Hyperæmia), or from passive hyperæmia or *congestion* through the central organ of the circulation. Second, the bloodvessels are the seat of *thrombosis* and *embolism*, particularly the latter, causing renal infarction. Third, *infectious* material, as micro-organisms or toxins, is carried to the kidney, and, in passing through the structure, gives rise to *inflammation* either of an infective or of an irritative character. Similarly, poisons that are ingested, and the products of metabolism, which, if modified in character or increased in amount, excite irritation and lead to inflammatory changes.

But the kidney is open to attack from sources lower down in the urinary tract. Through the bladder and ureter infection may extend upward, causing the consecutive inflammatory processes which are often seen after disease of the urethra, bladder, or ureter. The kidney is at the apex of a system of tubes or channels. Any alteration of them, whether mechanical or functional, has a secondary effect upon the kidney. Obstruction of the ureter, or obstruction in the conduits beyond, leads to consecutive hypertrophy, inflammation, and atrophy. (See Morbid Processes.) If the urine is abnormal, one of these three causal conditions obviously may be present.

The morbid processes which may take place in the kidney are such as are common to all organs—congestion, inflammation, degeneration, and morbid growths. The symptoms that attend the morbid processes are such as accompany similar processes elsewhere. The *general* symptoms of the morbid processes are not marked except in the case of infectious inflammation or of morbid growths, as carcinoma. There are fever and emaciation. *Fever* occurs in acute nephritis, perinephritic abscess, suppurative and tuberculous nephritis, pyelitis, and, with twists of the ureter, in floating kidney. *Emaciation* occurs in chronic, suppurative, and tuberculous nephritis and carcinoma. Other general symptoms in renal disease are due to the interference with the function of the organ which usually results. *Pain* is the only *local* symptom due to the morbid process; a *swelling* the only physical sign.

The symptoms of renal disease are also due to the functional or anatomical alteration of the kidney. But the structure is so closely interwoven with the function that morbid changes in one imply morbid changes in the other. As the anatomical alterations are usually beyond the pale of physical investigation, we find that functional symptoms alone are apparent. Hence, we look for *changes in the urine*, which is

the product of renal function, and for symptoms resulting from abeyance or cessation of the function. Rarely we have *enlargements* due to tumor, as cancer or abscess, or to obstruction of the channels, causing hydronephrosis, or to parasitic disease.

The symptoms due to the alteration of function are: 1. *Uræmia.* 2. *Cardio-vascular symptoms.* 3. *Anæmia.* 4. *Dropsy.* 5. Alterations of the *urine.* 6. Alterations in *micturition.* The symptoms of renal disease are, therefore, both subjective and objective.

The urine is not simply an index of the condition of the kidneys. It varies, within the bounds of health, in color, quantity, and quality. Food, exercise, and other conditions modify the secretion. It can readily be seen, therefore, that any general disease and many local diseases cause alterations in the character of the urine. Any abnormal urine, therefore, may be symptomatic of renal disease or of disease beyond the point at which the urine passes out of the body. Usually abnormal changes in the urine, due to the general condition, do not give rise to local renal symptoms or to abnormal renal function. The exception is seen when an excess of uric acid, or of urates, or of oxalates is passed. They may give rise to local pain and may set up sufficient irritation to cause albuminuria.

Classification. The best classification of diseases of the kidneys is that based upon the propositions of Delafield, who, in a paper entitled "On the Diseases of the Kidneys Popularly Called 'Bright's Disease,'"[1] submitted a classification dependent upon the nature of the morbid process. The morbid processes included congestions, degenerations, and inflammations of the renal structure. In addition to these affections we must also include in the nosology of renal disease tumors (cancer, abscess, and hydronephrosis), and anomalies of growth or position (floating kidney, horseshoe kidney), affections due to invasion of the kidney by parasites, and affections due to obstruction of the tubes through which the offices of the kidney are carried on (renal calculus, hydronephrosis, and pyonephrosis).

The Data Obtained by Inquiry. The Subjective Symptoms.

The subjective symptoms are due to morbid processes within the kidney or to alterations of its function. The class of nervous symptoms which belong to uræmia are subjective in character, as are also the symptoms of movable kidney.

Pain. Pain in the kidneys is referred to the loins. It is complained of as a dull aching, sometimes increased by movement, often attended by a sense of weight or pressure. Pain of this character extends over the entire lumbar region and is due to disease of both kidneys, as in acute nephritis. It is bilateral. We have also unilateral renal pain, referred to one kidney. The pain may be seated in the region of the kidney behind, opposite the two lower dorsal and two upper lumbar vertebral spines, or deep-seated in the abdomen, to the right or left of

[1] Trans. Amer. Physicians, vol. vi., 1891, p. 124.

the spinal column below the level of the umbilicus. It is not generally mistaken for pain due to other causes, as myalgia, or disease of the vertebræ. If myalgic, it may follow exposure to cold and be associated with pain in other muscles. Neuralgia of the kidneys no doubt occurs. It may be due to malaria, lead-poisoning, gout, or anæmia. It partakes of the character of neuralgia elsewhere. It must not be forgotten that in a case of disease of one kidney the pain is frequently *referred* to its healthy fellow.

Unilateral pain may be constant or paroxysmal. *Constant* pain is usually due to organic disease of the kidney, as *carcinoma* or *tuberculosis*. (See Palpation.) It may, however, be due to the impaction of a *calculus* in the pelvis of the kidney. It may also be due to a displaced or *movable kidney*. In *tumors* the pain may follow the course of the sciatic nerve, simulating sciatica. In *pyelitis* and *hydronephrosis* the pain is of a tearing character, whereas in movable kidney it is variable.

Paroxysmal and *lancinating* pain, the paroxysms occurring at long intervals, is usually due to *renal calculus*, or to the presence of a foreign substance, as blood, in the pelvis of the kidney. The pain is seated not only in the regions just indicated, but extends along the ureter, from the loin to the front of the abdomen. It may persist for some time, at a point on either side of the umbilicus above or below it, or at a point on the surface of the abdomen opposite the brim of the pelvis. Thence the pain extends into the bladder, either above the pubis (the hypogastric region), or into the testicle, or down the inside of the thigh. It may be in the loin and at the end of the penis at the same time, or lancinate along the whole urinary tract. In rare cases the pain is in the kidney of the healthy side. The pain of renal colic is always associated with frequency of micturition, with or without pain during the passage of the urine. The character of the urine often points to the cause of the pain. The urine is usually bloody, and at first scanty; when the obstruction is removed, it becomes copious. It sometimes contains pus. Between the paroxysms the urine may contain blood, pus, and pelvic epithelium. Renal pain or colic located in front of the abdomen must not be confounded with the pain of hepatic or intestinal colic. The pain is usually lower than in hepatic colic, extends along the course of the ureter, and is attended by symptoms referable to the urinary and not to the hepatic system.

Nephrolithiasis (Renal Calculus).

Renal calculi vary in size from "sand," through "gravel," to "stones." The latter may be from the size of a cherry to one large enough to fill the pelvis of the kidney. They consist usually of uric acid, and are hard, brownish-red or blackish, crystalline, and the larger ones are arranged in distinct layers. More rarely we have calculi of calcium oxalate, extremely hard and nodular. Some stones have alternate layers of the two salts; others consist of phosphates, but usually the inside is of uric acid or calcium oxalate, the phosphates having been deposited after the urine became alkaline. Very rare forms are of cystin, xanthin, indigo, etc.

A consideration of the frequency of the affection and some etiological data aid in the diagnosis. It is not a common affection. I have had twenty-nine cases in private practice and eleven in hospital practice. Thirteen cases only have been treated in the Presbyterian Hospital in twenty-five years, during which time over 8000 cases of all kinds were treated.[1] It is a disease of the middle and upper classes. This is particularly true of uric-acid calculous disease. It is not a disease of the old or the very young, in my experience. The youngest subject was twenty-five years of age; the oldest sixty-nine. The ages ranged from thirty-five to fifty-five. Twelve of my private patients were of the female sex, seventeen of the male sex. There does not seem to be much difference of frequency in the two sexes. Most authorities, however, hold to the preponderance in women, the ratio being as 3 to 1. Sedentary occupation and an in-door life are predisposing.

Symptoms. Symptoms may be wanting or they may be divided into three classes :

(*a*) Calculi may remain in the pelvis of the kidney, and not cause any renal symptoms. They may cause gastric disturbance or catarrh of the bladder or renal pelvis. There may be occasional pain in the lumbar region, the cause of which is unsuspected.

(*b*) They may excite pain, hæmaturia, and frequent micturition.

(*c*) They may attempt to pass from the pelvis of the kidney into the ureter. They then cause renal colic, the symptoms of which have been described above. In the intervals of the attacks of colic the patient may be free from symptoms.

The symptoms ascribed to the presence of a calculus in the pelvis of the kidney are *pain, intermittent hæmaturia, pyuria, pyelitis, renal intermitting fever, acute orchitis, frequent micturition,* and *renal colic.*

Pain. Pain of the affected organ is the most constant symptom, and this pain is increased by movement, by jolting, and by pressure. Indeed, pain induced by pressure is of as great significance as spontaneous pain. It frequently is persistent, and even continues in any position assumed by the patient.

Pain in the region of the kidney occurs from renal hyperæmia, nephritis, pyelitis, tumors, and malignant disease, or from myalgia of rheumatic or other causation. Indeed, we have seen renal pain and hæmaturia in a case of commencing appendicitis. The pain of renal calculus (not renal colic) comes and goes, and is more commonly intermitting and paroxysmal. Very frequently, however, it is constant and localized, either in the region over the kidney, or anteriorly in the region mentioned. In my experience it comes on during the day, and particularly the after part of the day, and not, as Jacobson would have us believe, at night. That it may occur spontaneously is not so much a peculiarity of renal calculus as that it can be excited by pressure, movement, etc.

Pain is of more diagnostic significance in renal calculus than in any other renal affection. Every attribute that has been applied to pain belongs to the pain of renal calculus. Its very vagaries render its

[1] J. H. Musser: "Renal Calculus," Philadelphia Medical Journal, 1898.

presence one of the most valuable signs of renal calculus. Its behavior, however, is often like the flitting nerve-aches of hysteria, and we must see to it that this counterfeit is not passed upon us. Urinary phenomena do not serve for the distinction; other neurotic manifestations or the stigmata of hysteria aid in the diagnosis. The pain may be aggravated by the function of menstruation and even bear close relationship to it.

Hæmaturia. Hemorrhage from the kidney is the classical symptom of stone. It is the most constant and positive symptom of renal calculus. Prior to the use of the centrifugal machine, blood no doubt escaped the eye of the observer when in small amounts, partly because it was destroyed as the urine advanced in decomposition during the period it was set aside for the deposition of its solid elements, and partly because the fewness of corpuscles rendered them difficult to find. Excluding all causes outside of the kidney—*i. e.*, of vesical and ureteral origin—renal hæmaturia may be due to congestion and inflammation, to infarctions, to new-growths, to tuberculosis, to renal calculus, and to parasites. The fevers and infections, and scurvy, purpura, leukæmia, and hæmophilia are responsible for a number of cases. In six years 2923 samples of the urine of 1997 persons were critically examined in my laboratory. Blood was present in 364 cases detected by microscopic examination alone.

The hæmaturia resulted from congestions or hyperæmias (pregnancy, goitre, heart-disease, the fevers, infections, and jaundice) in fifty-six cases. In forty-two cases the hæmaturia occurred in the course of acute and chronic Bright's disease, and in nineteen more in arterio-capillary fibrosis, being either of renal or cardiac origin. Gastric disorders, rheumatism in many forms, gout, neurasthenia, and anæmia account for eighty-one of the cases, conditions always associated with the copious discharge of urinary salts, which are irritating. Vesical disease accounts for seventeen cases, renal calculus for twenty-eight, and in twenty the diagnosis was not noted at the time and is forgotten.

All the cases of renal calculus had hæmaturia. It is not an intermittent phenomena alone, but one that is constantly persistent.

It is necessary to eliminate all sources of urethral, vesical, and ureteral hemorrhage before coming to a conclusion that the hemorrhage is of renal origin. Cystoscopy must be resorted to, of course, and possibly in the right hands, ureteral catheterization. If the hemorrhage is free the time of its passage in the act of urination must be determined. The reaction of the urine must be borne in mind. It is true, catheterization alone can avail to pronounce from which kidney the hemorrhage comes.

Blood-cylinders are rare, if present at all, in renal calculus. They denote hemorrhage from the renal substance.

In a person of middle life with uric or oxalic acid tendencies, by virtue of heredity, occupation and habits, in whom no cause for the hemorrhage can exist in the urethra, bladder, or ureter, the chances are that it is of pelvic origin, due to the irritation of gravel or of urine densely loaded with salts.

Klemperer[1] has recently called attention to hæmaturia from healthy

[1] Deutsche med. Wochenschrift, March 4, 1897.

kidneys, as the result of overexertion, in one case from horseback riding, in another from the bicycle. He also reports four cases of hæmophilia and a group due to an angioneurosis. Hyaline casts were not present, although blood-cylindroids were. General symptoms of neurasthenia support the diagnosis in the angioneurotic cases.

PYURIA. Pus in the urine is looked upon by all authorities as almost essential to the diagnosis of renal calculus, but in my experience this product of inflammation is usually absent. Of the twenty-eight cases which I examined, in fifteen there was no pus; in six a few cells or a very small quantity was found (four, womb cause obvious); in one it was noted as considerable (old gonorrhœa and syphilis, four examinations); in one a small quantity (male, cause assignable); in one it was small in amount, twice only in some fifty examinations; in one it was abundant and due to genito-urinary infection as well as pyelitis. Pyuria is not present unless an accidental infection has taken place from the lower tract.

ALBUMIN. In twenty-one patients albumin was found. It was in large excess in three, due to coexisting Bright's disease. As a trace it is of frequent occurrence and does not imply a coexisting nephritis.

CASTS. Casts are present in the urine in nearly all cases of renal calculus. Sedimentation must be used. They are hyaline—not abundant—long and narrow. Their persistence without other kinds, with or without albumin, is diagnostic of renal irritation, and with other signs points quite unfailingly to calculus.

THE SPECIFIC GRAVITY of the urine is an aid in the diagnosis. Its persistence above the normal is both a comfort and a sign. It enables one to exclude renal cirrhosis and aids to eliminate hysteria or a renal neurosis.

FREQUENT MICTURITION is not in my experience an indication of stone in the kidney, save when attempts are made for its passage, although spoken of as a symptom of value by most authorities.

PAROXYSMAL RENAL FEVER, allied to hepatic fever in its expression, rarely occurs, but when present may be due to calculus. It may also be due to absorption of retained products, if the kidney is floating and becomes twisted. It may be due to pyelitis.

DURATION OF SYMPTOMS AND FAMILY HISTORY are also valuable data.

DIAGNOSIS. Middle life is a predisposing factor, and persistent hæmaturia is symptomatic, but pyuria rarely so, while albuminuria and hyaline casts in urine of high specific gravity are prominent elements of the symptom-complex upon which a diagnosis is made.

The diagnosis can be established by the symptom-complex of *pain*, local *tenderness*, persistent *hæmaturia*, *albuminuria*, and *casts* (the cardiac, vascular, and nephritic origin of which is excluded), by the phenomena of *renal colic* and by *passage* of *fragments* of stone.

If the hemorrhage persists after prolonged rest, it is more likely of cancerous or tuberculous origin.

The differential *diagnosis* must be made from appendicitis, movable and twisted kidney, biliary colic, and other affections simulating these. Catheterization and exploration by the ureter are required in many

cases. Hollander believes we can in a large number of cases make a diagnosis without the aid of catheterization, and fears the danger of infection from below.

Kelly, very skilfully after ureteral catheterization, aspirates the ureters and thereby brings down fragments of calculi. He also explores the ureters with hard-rubber bougies tipped with wax. He can determine the presence of calculi by the markings on the tips of the bougie.

Frequency of Micturition. There are four causes of frequent micturition: (1) Disease of the kidneys, the ureters, or the bladder; (2) an increase in the amount of urine, as in diabetes; (3) concentration of the urine, as in fevers, gout, or acute nephritis; (4) a reflex or pure neurosis.

Increased frequency of micturition occurs in almost all organic affections of the genito-urinary system. It is seen in all forms of congestion and inflammation of the kidneys. In some forms of nephritis the increased frequency may be due to increase in the amount of urine as well as to increased sensitiveness of the organs. In chronic nephritis it may not be noticed, save that the patient is called upon to pass urine at night, arousing him from sleep for this purpose. Disease of the ureter and disease of the bladder are also associated with this troublesome symptom. It occurs in its most aggravated and characteristic form in renal calculus, or when any foreign substance is located in the ureter or bladder. The frequency amounts to six, eight, or even a dozen times in an hour. It is often associated with tenesmus, the patient having a constant desire to urinate, but passing small amounts. This form of tenesmus is more frequent when the bladder or urethra is the seat of disease, and in renal calculus.

The Data Obtained by Observation The Objective Symptoms.

The data obtained by observation are secured: 1. By physical examination of the kidney. 2. By an examination of the urine. 3. By catheterization of the ureters. 4. By a skiagraphic examination. The examination of any person who is sick is not complete without an examination of the kidney and of the urine. The third and fourth methods of examination are not necessary unless the subjective symptoms indicate their necessity, or general symptoms are not otherwise explained.

Topography of the Kidneys. (Plate XIII., Fig. 2.) The kidneys are situated in the right and left lumbar regions respectively, the left being a little higher than the right. They extend from the eleventh rib, or twelfth dorsal vertebra, to the third dorsal vertebra. The left kidney is in contact above with the spleen, and the right with the liver.

Palpation and Percussion. The kidneys are enveloped in more or less abundant fat; their distance from the anterior surface of the abdomen renders them inaccessible to percussion from that direction, and the thick dorsal and lumbar tissues, coupled with the relation of the kidneys with the organs, spleen, and liver, which give a dull note on percussion, make it difficult to outline the kidneys from behind.

Palpation of the normal kidney is difficult. It can only be bimanual.

Place the fingers of one hand below the last rib outside of the lumbar muscles—erector spinæ—and apply the other below the ribs in front. Firm, persistent pressure with the abdominal muscles relaxed, especially in thin subjects, will often enable the normal kidney to be felt.

Palpation of the kidney becomes easy when it is either enlarged or displaced. In the case of an enlarged kidney the patient should lie upon his back or be slightly turned to the opposite side; one hand is placed beneath the kidney and pressed upward, while the other is pressed firmly and steadily from above, or laterally toward the kidney. In this manner the kidney can be grasped between the two hands, its size estimated, and its physical characteristics as regards hardness, softness, fluctuation, and mobility determined. Enlargements are also detected by palpation of the abdomen. (See Palpation of the Abdomen.) The fact that the tumor moves a little with respiration aids in its detection; and if it is unusually movable the edge of the hand can be slipped above its upper end, by turning edgewise that border of the hand which is adjacent to the ribs. A renal tumor is usually two or three inches to either side of the median line, a little above the transverse umbilical line.

A very favorable position for palpating movable kidneys is that assumed by standing and leaning forward over a chair, with the trunk supported by the hands resting on the seat of the chair. In this position the abdominal muscles are relaxed and the kidneys fall forward.

In the diagnosis of renal tumors, in general, it should be borne in mind that they are slightly *movable* with *respiration* unless adherent, as in malignant disease, abscess, and cysts. Unless too large they preserve their *reniform shape*, and press in front of them the ascending or descending *colon*, whereas ovarian tumors lie in front of it. The position of the colon should, therefore, always be ascertained, and to this end it may be necessary to inflate it.

Percussion. The best results are obtained by having the patient lie face downward, and placing a cushion under the belly, so as to make the lumbar regions a little more prominent. Strong percussion is required, and an artificial plessor and pleximeter are to be preferred. Percussion should be conducted with a view to marking the angle which the liver-dulness and splenic dulness make with that of the kidney on the right and left sides respectively. The kidneys extend below the lower lines of liver and splenic dulness, and laterally for a width not greater than four inches. The difficulties in the way of outlining the kidneys by percussion are greatly increased in persons with much flesh, or when the abdominal walls are waterlogged, as they become in ascites, and it is practically impossible, under such circumstances, to be sure of the boundaries of the kidneys. The colon must be emptied to yield trustworthy results.

Movable Kidney.

Movable kidney is usually seen in women after the age of forty years, who have done physical work or have had many children. Adult males and single women do not escape. Its occurrence is frequently

preceded by a history of unusual lifting or strain, followed by tearing or dragging sensations in the abdomen. Pain may continue for several weeks after the injury, and then subside and the occurrence be forgotten, or subjective sensations may continue. In other instances the movable kidney is a part of a general visceral displacement. Gastroptosis and gastro-enteroptosis can usually be made out in such cases.

The symptoms that arise are due to the local dragging or pulling of the kidney on its bloodvessels and nerves, or to reflex symptoms, or to pressure upon adjacent organs.

The pain that attends movable kidney is usually referred to the right or left of the median line; sometimes to the hypogastrium. It may be constant, dull, and aching in character. Paroxysms may arise in the course of the constant pain, or a paroxysm alone may take place. The paroxysms continue for three or four days, during which time other subjective symptoms are more pronounced. The attacks are known as *Dietl's crises.* Nausea may attend the paroxysms, or be more or less constant. Sometimes vomiting takes place. The great pain is associated with swelling and tenderness of the kidney. The pain, vomiting, and local tenderness may simulate peritonitis.

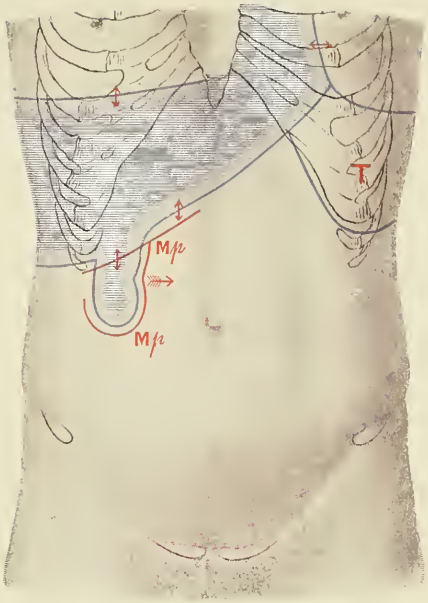
In addition to pain a dragging sensation is experienced; the patient may be aware of the presence of a tumor or lump in the abdomen, and also of its movability. The reflex symptoms are chiefly referable to the nervous system. Emotional disturbance is observed when the organ is displaced. Hysteria may be present. Palpitation of the heart is a common reflex symptom. There are often depression of spirits and hypochondriasis. Jaundice may occur from pressure, and the intestine may be occluded.

The urinary symptoms are of interest. When the local pain and other symptoms are more pronounced the urine may be scanty. In one case it was reduced to sixteen ounces in twenty-four hours. At the same time that the urine is scanty hydronephrosis will develop. It will be referred to again. As the kidney slips back into its bed the twisting of the ureter is relieved, and copious discharges of urine take place.

Objective Symptoms. (Plate XLV., Fig. 1.) The abdominal walls are usually relaxed, and may or may not contain a large amount of fat. *Movable kidney* is best detected by palpation. The patient should stand with the body bent forward and the hands resting on a chair, as described above. The organ is recognized by its rounded borders, its bean shape, its movability, the detection of the hilus and perhaps of the pulsation of vessels in it, and by the fact that it can be replaced. Palpation causes a sickening feeling, analogous to that experienced when a testicle is compressed, but less in degree. Percussion will, however, demonstrate that a body, supposed from palpation to be the kidney, is a solid organ. The tumor can be found to the right or left of the median line, freely movable and changing its position with that of the patient. If the tumor is situated on the right side, it may be in close proximity to the liver, or be felt opposite the umbilicus, or often in the iliac region. When near the liver, by careful palpation the fingers can be introduced

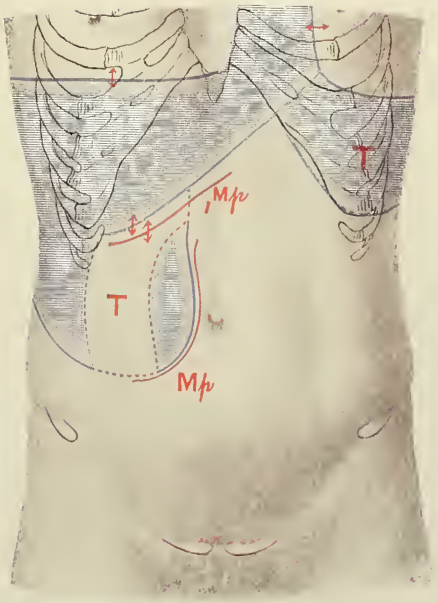
PLATE XLV.

FIG. 1.



Movable Kidney.

FIG. 2.



Sarcoma of the Right Kidney.

between the border of the liver and the mass. Usually it does not move with respiration, but sometimes it is found to do so. On the left side it may be as high up as the margin of the ribs. It is generally felt in the mid-clavicular line, a little above the level of the umbilicus.

In a case recently under the writer's care the woman, aged fifty-five years, would experience pain in the abdomen about once a month, to the right of and above the umbilicus. At times nausea and vomiting accompanied the attacks, at other times marked depression or hysteria. Anuria always occurred and continued for a variable time, not longer than five days. With one of the paroxysms a tumor was found in the region of the gall-bladder, movable with respiration, but distinctly defined from the liver by placing the fingers between the lobe and kidney. It moved with each change of position of the patient, and at first the hilus could be distinctly felt. As the pain continued the anuria persisted, and a marked change in the tumor was observable. It gradually increased in size, and a portion of it fluctuated; it was round and partook of the character of a cyst. The fluctuation was detected by placing the hand on the tumor in front and pressing firmly toward the other hand placed in the loin above the pelvis. After several days a copious discharge of urine took place and the swelling subsided.

Movable kidney may be confounded with tumor of the gall-bladder, tumor of the pylorus, and with tumors in the pelvis. It is not likely to be confounded with an omental tumor, carcinoma, or tuberculosis, because the phenomena of these processes are not present and ascites does not occur, nor is there rise of temperature, as in many cases of tuberculosis. As pointed out by Henry Morris, tumor of the gall-bladder and movable kidney are frequently of conjoint occurrence. Movable kidney is distinguished by the absence of previous history or of symptoms or signs indicating disease of the gall-ducts. If jaundice is present, it is not so intense as in tumors of the gall-bladder. While the gall-bladder is movable, it is not so distinctly so as movable kidney. The gall-bladder moves in an arc of a circle, the centre of which is at the edge of the right lobe of the liver. It can be pushed further to the left than to the right, but never downward as a movable kidney. Moreover, the gall-bladder is always palpable, the movable kidney cannot always be felt. The gall-bladder, if it contain calculi, is very hard compared to the kidney. Anuria does not occur.

The kidney tends to spring back to its place in the loin; the gall-bladder to the anterior part of the abdomen. Even if the gall-bladder is enlarged, the kidney can be felt by bimanual palpation; while the opposite does not obtain. In cancer of the pylorus the emaciation and anæmia are more pronounced than in movable kidney. The vomiting, usually characteristic in that affection, and the physical signs of dilated stomach, can be made out. Tumors of the pelvic organs are determined by examination according to the usual methods.

Horseshoe Kidney. There are usually no symptoms. The kidney can sometimes be felt through the abdomen if its walls are relaxed, or by bimanual examination.

Enlargement. Renal Tumor.

Enlargements of the kidney may be detected by percussion; the width of the kidney is increased, and the percussion-dulness therefore extends further to the right or left, according as the right or left kidney is affected. As the causes which produce enlargements of the kidney sufficiently great to be detected by percussion do not, with rare exceptions, involve both kidneys at the same time, comparison of the two sides is of great value in the diagnosis.

Renal tumors rarely bulge in the lumbar region, although there is a sensation of increased resistance in this area. The mass is never notched, is usually smooth, and often takes the shape of the kidney if that organ is involved in its entirety. Otherwise the outline is not reniform. The bowel is usually in front of the mass, although in tumors of the right kidney the cæcum and colon may be pushed to the inner side, and of the left kidney the colon may be pressed outward.

The diseases of the kidney attended by enlargement are: *malignant tumors, cystic kidney, hydronephrosis* and *pyonephrosis, abscess*, and *perinephritic abscess.*

Sarcoma and Carcinoma of the Kidney.

Either disease may be primary or secondary. Sarcoma may be congenital. The tumor may occur at any age, but is relatively common in young children. Twenty-five out of sixty-seven cases collected by Dr. William Roberts occurred in children under ten years of age. In older persons it is often preceded by calculus. *Symptoms:* In some instances there are no symptoms during life. In others the disease may advance considerably before it presents any signs. If symptoms are complained of they are usually limited to pain, the occurrence of hæmaturia, or the development of a tumor. The pain is dull and seated in the lumbar region. It may be neuralgic in character; and, indeed, there may be a true *sciatica* with paresis of the leg from pressure of the tumor. The *tumor* (Plate XLV., Fig. 2) is firm; its surface is smooth or nodulated. It may be felt in the loins, and in front, above the umbilicus, a few inches to the right or left of the median line; the descending colon lies in front of the tumor. The latter may grow with great rapidity and attain enormous size, filling the abdominal cavity and giving rise to pressure-symptoms in surrounding organs. The growth occurs more often anteriorly and downward toward the pubis, because there is less resistance in these directions. As rapidly growing cancers are soft, the tumor frequently exhibits a certain degree of elasticity, which may be mistaken for fluctuation. It is immovable either by the hands or with respiration.

On percussion the resistance is increased and the note is dull, except in front, where the colon, which has been pushed forward, gives a tympanitic note. If the colon should be flattened out between the tumor and the abdominal wall, it may be felt as a band stretching across the tumor, with dulness on percussion. In such a case inflation of the colon will be of great assistance in the diagnosis. Rare physical signs

are pulsation and a blowing murmur. The *hæmaturia* may be constant or intermittent. The clots of blood may cause renal colic.

The general symptoms are those of carcinoma. A marked rapidity of the pulse has been noted in several cases. In girls a premature development of hair on the pubes and in the axillæ and pigmentation of the skin have been observed.

Hemorrhage is an early symptom, and in the absence of nephritis or cystitis should always suggest tumor. It may occur early and may be intermittent or persistent. In some instances it occurs but once, usually it is frequent. When excessive, the growth is never innocent. Pain is not of much value, and may be absent until perinephritis occurs. Symptomatic varicocele may occur. The examination of the urine, save that it discloses the presence of blood, is negative. In this sense it is of value. Pus occurs if there is secondary infection or if calculi precede the growth. Rarely fragments of carcinoma are said to be detected. In order to determine the kidney affected separate urine should be obtained from each organ.

The tumor must be distinguished from tumors of the lymphatic glands, of the liver, of the spleen, and of the ovary. It must not be confounded with psoas abscesses and perinephritic abscesses, which cause a tumor in the lumbar region.

Cystic Kidneys.

1. *Congenital.* The kidney consists of a small mass of cysts filled with clear fluid. It may interfere with the birth of the child on account of its large size.

2. *Acquired.* The cause is trauma and obstruction of the ureter, the presence of which is determined by catheterization. The symptoms are those of a fluctuating renal tumor. The urine may be normal or hæmaturia may be present.

Hydronephrosis.

Causes. It may be congenital. Obstruction of ureter by stone; pressure of tumor; twist, as in movable kidney; exudates.

Symptoms. In addition to the symptoms of the causal condition we have, upon the development of hydronephrosis, the presence of a *tumor*, arising in the region of the kidney and extending toward the middle line. Sometimes fluctuation can be detected; often it cannot. Variations in size of the tumor may occur with changes in amount of urine passed. Puncture, and the finding of a fluid with elements of urine in it, are valuable means of diagnosis; but if the hydronephrosis is old, this fails, as the fluid loses its urinary character, and cannot, for instance, be distinguished from that of an ovarian cyst. When on one side the urine may be normal; when on both sides it is diminished; anuria and uræmia may occur. If pyelitis is present, pyuria is observed.

Intermittent hydronephrosis is associated with movable kidney, hence it is more frequent in women. It is characterized by the development of a renal tumor with variable frequency, and with pain, nausea, and vomiting. At the same time the urine is scanty. In a few hours or

days there is an increase in the amount of urine with subsidence of the tumor.

Pain may or may not be present. Gastric symptoms are very common. Either constipation or diarrhœa is seen. Hypertrophy of the left ventricle may occur, as in *chronic* nephritis.

Hydronephrosis consists in a dilatation of the kidney pelvis with urine, which is prevented from escaping by obstruction of the ureter, either by the pressure of a tumor, or by disease of the bladder or ureter itself. In time the kidney atrophies from the pressure and a large cyst forms. The tumor has the physical characteristics of pyonephrosis, but the history is different, and if there is any discharge, it is free from pus. As in pyonephrosis, the tumor may become small, following a copious discharge—in this case of urine—or may even wholly disappear, if the obstruction is removed. This sign is pathognomonic.

If obstruction continue to be absolute, the diagnosis must be made by the detection of a fluctuating renal tumor, the absence of fever and signs of suppuration, and by the result of exploratory puncture. The urine is usually free from pathological changes.

It may be confounded with ascites, if very large, but hydronephrosis is rarely bilateral, and the fluid in it does not change its level upon change of position of the patient, as is the case with ascites. The history of the two conditions will be different.

An ovarian cyst can usually be traced into the pelvis; it does not carry the colon in front of it, and hence is dull, even on superficial percussion, and it leaves the loins resonant.

Pyelitis. Pyonephrosis.

Pyelitis is rarely primary; usually secondary. Severe infections diseases (typhus, variola, diphtheria, pyæmia); toxic substances ingested (cantharides, etc.); chronic nephritis; inflammation of the bladder or ureter; strictures of the ureter or urethra; hypertrophy of the prostate; spinal palsies of the bladder; calculus; parasites; blood-clots, are the antecedent causal factors; infection the active cause.

Symptoms. The Urine. Pus in the urine with pelvic epithelium—although it is not safe to base a diagnosis on the presence of the latter; casts of the canals opening into the pelvis are more characteristic; epithelial casts, and casts containing micro-organisms. The urine is often increased, acid, and contains pus and albumin, rarely blood. Pyuria may be the only renal sign. In all forms of pyuria above the bladder Kelly withdraws the pus by catheterization and suction. He allows the catheters to remain from ten minutes to four or five hours, in order to estimate the functional power of each kidney. Of course, the pus is studied microscopically and bacteriologically. *Pain* in the region of the kidney, often severe, is complained of, although it may be absent. When present, it is often of a tearing character. *Tumor.* A tumor is often present. It is most prominent in the loin or in the abdomen. In the latter the mass can be felt two inches to either side of the umbilicus, usually above the transverse line.

Pyelitis differs from abscess of the kidney. The latter may be the

result of a local infection from the pelvis of the kidney or may be pyæmic.

In *abscess* of the kidney there is some fulness in the loin of the affected side. The kidney is felt to be enlarged, and is tender and painful. A tumor may be detected anteriorly. The diagnosis is based on a study of the cause (acute nephritis, pyæmia, impacted calculus in the ureter, erysipelas), or the detection of blood and pus in the urine, which is scanty, and on the constitutional symptoms. The progress of the case is usually acute. If the abscess is *tubercular*, tubercle bacilli can be detected in the purulent sediment of the urine, and there will be other foci of tuberculosis with a corresponding clinical history.

When the pus is confined by an occluded ureter, the pelvis is overdistended. In *pyonephrosis* the tumor is tense, smooth, and globular. Fluctuation may be detected. Tenderness is usually absent; the course is slow and does not affect the general health so much as abscess. The pus may be discharged copiously from time to time, and the tumor be therefore diminished in size. The urine may be occasionally almost clear. Pyonephrosis arises secondarily to pyelitis, and often after the latter has lasted some time.

Fever is irregular, remitting, or septic. The fever and pyuria may be the only symptoms. If the *bladder* is healthy, its symptoms fail to aid in diagnosis.

Perinephritic Abscess.

It occurs as a primary disease in apparently healthy individuals, or after infectious diseases.

Perinephritis arises usually from extension of inflammation and suppuration from the kidney, but may be the result of strain, exposure to cold, or injury. Perinephritis may also be pyæmic, and occur after infectious fevers, and in actinomycosis.

Symptoms. The *secondary forms* have symptoms of the primary disease, and, later, swelling and pain in the renal region.

Primary form. Chills and fever, pain, difficulty in defecation. The general condition suffers. Finally, in all cases, there is the formation of a swelling in the lumbar region, at first hard; then œdema of the skin follows, and fluctuation is detected. The abscess may descend and point above Poupart's ligament. It may press upward and cause dyspnœa. Great tenderness and pain in the region of the swelling may arise, and the pain may radiate to the leg. Irregular septic fever and chills appear. The *urine* is not generally changed unless some communication with the pelvis or ureter has formed. The patient lies on his back, turned toward the affected side. The knee and hip of this side are flexed and the thigh rotated outward. The affection may simulate *coxitis* and *appendicitis*.

The swelling of a *perinephritic abscess* appears in the lumbar region of the side affected. It is rounded in form and doughy (Da Costa). Like other kidney tumors, it is not affected by respiration. The usual signs of confined suppuration exist, and pulmonary or pleural complications may occur. As the abscess progresses, the local signs of suppu-

ration become more marked, the skin reddens, and pus may be discharged externally.

The most marked subjective symptom is *pain*, which may amount to agony, and is paroxysmal; soreness from restricted motion of the psoas muscle is apt to be complained of.

A tumor was present in the loins in sixty-five out of seventy-one cases analyzed by Fenwick, but did not generally manifest itself until the inflammation had made considerable progress. There is dulness on percussion even in the early stage, and, later, fluctuation. The general symptoms are vomiting, constipation, fever, and sometimes rigors. It is more common in males than in females (sixty-one males to thirty-nine females in Fenwick's cases).

Hydatid Cyst.

A *hydatid cyst* of the kidney presents the usual physical signs of such cysts. A fremitus may be detected, or small cysts may be found in the urine.

It is comparatively rare. Usually there are no symptoms until a tumor is felt. Then pain gradually develops. The cyst may open into the pelvis of the kidney, and cysts or scolices be discharged, with *colic*.

Pyelitis and cystitis may also develop.

Echinococcus cyst may inflame and lead to general pyæmia. Puncture of the discovered tumor is otherwise the only means of diagnosis. It must be differentiated from hydronephrosis and ovarian tumors. Puncture is necessary.

Examination of the Urine.

1. Inspection. The urine in health is a clear yellow or amber-colored fluid, having a specific gravity of about 1020, and generally acid in reaction. It contains normally about forty-five parts in the thousand of solid matter, the principal part of which is urea—twenty-one and a half parts. The other solids are uric acid and its salts; certain extractives—creatin, creatinin, ammonia, hippuric acid, xanthin, hypoxanthin, sarcin, pigment, etc.; and chlorides, phosphates, sulphates, with their bases, soda, potash, lime, and magnesia.

The *volume* of urine passed in twenty-four hours is usually from forty to fifty ounces, but it may fall to thirty ounces or rise to seventy without the existence of disease. Women are believed to pass from five to ten ounces less than men. The volume is diminished when the skin is acting freely, as in warm weather, and when the bowels are loose; and, on the other hand, cold, constipation, and nervous excitement, especially if it induce anxiety and fear, all tend to increase the quantity secreted.

Color. The *color* of the urine is due largely but not wholly to urobilin, which is formed from the hæmatin of the blood. The color deepens when the urine is concentrated, which occurs after a hearty meal, or exercise, especially in warm weather; and it becomes paler when a large quantity is passed. The color is frequently changed in

disease. In fevers the urine, soon after being passed, is apt to become *turbid* from the precipitation of urates, and the color varies from white, especially in children, to yellow, brown, or pink. When the precipitate settles the supernatant urine may be high-colored and clear, or slightly opaque from some suspended matter.

The admixture of pus and chyle gives the urine a *milky* color. The urine may also be *yellowish-white* and turbid from phosphates, semen, sarcinæ, and bacteria.

The urine is *red*, reddish-brown, or "smoky" in acute nephritis, the color being due to blood. It is bloody in hæmaturia, cancer of the kidneys and bladder, and in injuries of the genito-urinary apparatus. The urine is very red and clear when concentrated and containing a large amount of urates. The red color of the urine may be due to hæmoglobin, constituting *hæmoglobinuria*, or to excess of urobilin, as in scurvy and pernicious anæmia. Hæmoglobinuria occurs as the result of the action of certain poisons, such as chlorate of potash; in infectious diseases, such as scarlet fever; and in malarial fevers; also in a peculiar disease known as paroxysmal hæmoglobinuria.

Again, a *golden-red* discoloration of the urine is common in jaundice; frequently the upper layers have a greenish tinge by reflected light.

Finally, a *red color* is produced by the internal administration of logwood and fuchsin.

A *yellow* color, when opaque, may be due to suspended phosphates and urates. Urine is sometimes golden yellow or of a saffron color in jaundice, and from the effects of santonin, picric acid, and rhubarb taken internally. A yellow or yellowish-white turbidity may be due also to a mixture of pus and phosphates, and sometimes to semen, sarcinæ, and bacteria. The urine usually becomes more or less opaque and yellow when it has undergone alkaline fermentation. Such a change occurs normally within a longer or shorter time after the urine has been passed. It is promoted by heat and exposure to air, and retarded by cold and exclusion from air. If possible, the urine should be examined before this fermentation has occurred. Pathologically, in cases of cystitis, the urine when passed is already in alkaline fermentation.

The urine is sometimes *chocolate-brown* when it contains blood and the blood has been acted upon by the urine, producing methæmoglobin.

Brown, *greenish-brown*, or *black* urine may result from contained bile-salts; from indican; from carbolic acid, creosote, and tar used internally and externally; from the internal use of senna, and in cases where there are melanotic tumors. Senator injected melanin into human beings and obtained in four cases only a large indicanuria.

Urine is *pale* usually in proportion as it is copious in quantity. It is paler in those who are using milk or vegetable diet than in those who eat meats. Under the influence of nervous excitement, especially anxiety and the dread of an approaching ordeal, such as an examination, an abnormal quantity of very pale urine is secreted.

Pathologically, *pale* urine is characteristic of diabetes, chronic Bright's disease, and polyuria. Such urine is also secreted in hysterical attacks, at the crises of febrile diseases, and in anæmic conditions.

The Quantity. The volume may be increased, diminished, or unchanged in disease. It is *increased* principally in three diseases—diabetes mellitus, diabetes insipidus, and in the middle period of chronic Bright's disease, especially the interstitial form. In diabetes mellitus it sometimes exceeds thirty-two pints. It may be increased also in hypertrophy of the left ventricle, which induces greater pressure in the renal arteries as well as in the whole arterial system; and also in cystic degeneration, and in double hydronephrosis.

Diabetes Insipidus. This form of diabetes differs from the saccharine in that the urine is normal, but of low specific gravity. The disease may come on suddenly after mental emotion, or develop gradually. The amount of urine may range from ten to forty pints. The urine is of low specific gravity—from 1001 to 1005. It is pale and watery. The solid constituents are not reduced. Urea is sometimes increased, but abnormal constituents are very rare. The passage of large amounts of urine induces thirst, but otherwise the symptoms do not tally with the symptoms of diabetes mellitus. The patients are usually well nourished.

The disease is usually secondary to some organic disease of the brain, or of the abdomen, as tubercular peritonitis, abdominal tumors, or aneurisms. It usually occurs in males, and is often hereditary. It is most common in young people. Traumatism, meningitis, affections of the brain involving the sixth nerve, tumors of the brain or of the medulla, are causal factors. It may follow fright, a protracted debauch, or perturbation of the nervous system from other causes.

The *diagnosis* is not difficult. It must be distinguished from the polyuria that is seen in chronic interstitial nephritis, and in amyloid disease. In hysteria, polyuria is common, although transitory. The presence of the stigmata and other hysterical manifestations lead to the diagnosis in hysteria.

The urine is *diminished* in acute nephritis and in the final stages of chronic nephritis; sometimes, also, it is diminished in the middle period of chronic nephritis, but usually it is here increased. All diseases which directly or indirectly impair the force of the circulation lessen the secretion of the urine. Hence, the quantity is diminished in diseases of the heart-muscle and in valvular diseases not fully compensated; in emphysema and in chronic bronchitis. It is lessened also in cirrhosis of the liver. In febrile diseases the urine is scanty and high-colored, and sometimes it is almost suppressed (anuria).

The urine is sometimes *suppressed* in acute nephritis, such as follows scarlet fever, and in the final stages of all the organic affections of the kidneys—chronic nephritis, hydronephrosis and pyonephrosis, etc. It may result (1) from the destruction of the secreting tissue of the kidney or interference with its nervous or vascular supply, or (2) from mechanical obstruction to the outflow of urine. To the first class belong the cases of suppression occurring in acute and chronic nephritis, and the suppression from shock and collapse, whether occurring in the stage of collapse of yellow fever, cholera, and other grave febrile diseases, or from serious internal injuries.

Such suppression sometimes follows slight operations on the urethra

(urethral fever); or results from the internal administration of drugs the excretion of which occasions violent irritation of the kidney—cantharides, turpentine, and even the inhalation of ether. Clinically, suppression not due to obstruction is distinguished from the obstructive form by the character of the urine, which is usually not entirely suppressed, and by the more rapid course of the disease. The urine, according to Roberts, is either concentrated or it contains albumin, blood, and casts. Death or recovery results within a day or two. In the obstructive form, on the other hand, the urine which escapes past the obstacle is pale, watery, and devoid of albumin and casts.

Obstructive suppression is the result of the plugging of the ureter by a calculus, when the opposite kidney is either absent or incapable of secreting. It also results from the occlusion of the ureters by morbid growths, especially at the vesical orifices, from lateral pressure upon the ureters, or from some interference with or malformation of the ureters or renal arteries.

Acute transient obstructive suppression occurs sometimes in persons with enlarged prostates, or old strictures, who have drunk too freely of alcoholic beverages, and, perhaps, have wound up a debauch by sexual intercourse.

The Density of the Urine. The average density of normal urine is about 1020. It may fall to 1015 or rise to 1025, depending upon the quantity of fluid and food taken, the condition of the atmosphere, especially as regards temperature, and upon mental influences usually of an emotional character. The specific gravity of the urine is tested by a urinometer graduated for degrees of density between 1000 and 1040. Only a reliable instrument should be used. As the density of the urine passed at different times during the day varies greatly, the urine for the whole twenty-four hours should be saved and a specimen of this tested.

The method of taking the specific gravity is very simple. A test-tube or graduate, having a diameter of about one and a quarter inch and a length of six or seven inches, is filled with urine to such a point that the lowest part of the urinometer when inserted floats clear of the bottom of the tube. The instrument must also float free of the sides of the tube. The specific gravity should then be read off from below—that is to say, by holding the tube up so that the level of the fluid is a little above that of the eye. Most urinometers are graduated for 60°, but in ordinary examinations it is not necessary to have the urine exactly at this temperature; it should, however, be allowed to cool after it has been passed, otherwise the specific gravity will appear to be too low.

In *disease* the specific gravity varies more widely than in health; it may fall to 1000 or 1005 in diabetes insipidus and chronic Bright's disease, and rise to 1060 or even higher in diabetes mellitus. As a rule, to which the urine in diabetes mellitus is the principal exception, the color is an index of the density, pale urine being of low density and high-colored urine of high density.

The density is increased when the urine is scanty in amount, whether as the result of fever, acute nephritis, large consumption of solid food, exercise, or free sweating. In all such cases the specific gravity rarely

rises above 1035, and usually not above 1028 or 1030. When the specific gravity rises above 1035, and the urine is pale in color, the presence of sugar is to be suspected; and when it rises above 1040 sugar is almost certainly present.

The specific gravity is lowered by drinking copiously, by the effect of external cold, by a diet of vegetables and milk, and, in general, by the same causes that make the urine copious. Usually, but not always, a urine containing a large amount of albumin is of low density.

Pathologically, a low specific gravity is encountered in diabetes insipidus, in which it may fall nearly or quite to 1000; generally in the middle or quiescent period of chronic Bright's disease; in the crisis of fevers; in obstructive suppression; in hysterical attacks, and in hydronephrosis.

Specific Gravity as an Index of the Amount of Solids. If the last two figures of the specific gravity be doubled, the sum will represent the amount of solid matter in 1000 grains of urine. This is Trapp's method; the estimate is only rough, but it is useful. Of course, the urine for twenty-four hours must be used.

The Reaction. The reaction of *healthy urine* is usually *acid*, but it may be neutral or slightly alkaline about two hours after a meal of mixed food. The acidity is tested with litmus-paper; the blue paper is turned purple or red by an acid, and the red paper is turned blue by an alkali. Violet paper is to be preferred, as it is suitable for showing both reactions, an alkali turning it blue and an acid red.

The acidity of the urine is *increased* in gout, lithiasis, acute rheumatism, diabetes, chronic Bright's disease, and as the result of the administration of vegetable or mineral acids.

The urine is *alkaline* as the result of alkaline fermentation in the bladder in cystitis; from the presence of much blood or pus; from prolonged immersion of the body in a cold bath; in debilitating diseases and in some cases of nervous dyspepsia, and as the result of the internal administration of alkalies.

Urinary Sediments. A white, flocculent sediment, composed of epithelium and mucus, occurs normally in most urines after they have stood for some hours.

A dense sediment, varying in color from that of brown sugar to pink or red, consists of amorphous urates. It dissolves upon the application of heat. A sediment usually resembling red pepper, but sometimes of a brown color, consists of uric acid. It can be proved to be uric acid by the murexid test. The suspected material is placed in a crucible or evaporating dish with a few drops of nitric acid. As heat is applied the uric acid or amorphous urate dissolves with effervescence. Heat is now kept up until the material is evaporated to dryness; it is then allowed to cool. If it is now touched with a glass rod, dipped in strong ammonia, a characteristic blue or violet color is produced. Uric acid is not usually so abundant as the sediment of amorphous urates; it sinks more rapidly, and is deposited from acid, high-colored urines.

A yellowish or whitish sediment may consist of urate of sodium.

A white sediment usually consists of phosphates, associated with which we sometimes find a white sediment consisting of urate of ammo-

nium, with or without pus. Such urines are alkaline. A white sediment may be due to uric acid, especially in children.

A yellowish-white sediment may consist of pus, with or without mucus. If the urine is acid, the sediment is loose and free to move; but when the urine is alkaline the sediment consists of a viscid, coherent mass, which can be drawn out into tough, stringy filaments.

A chocolate-brown sediment, occurring in a reddish, smoky urine, consists of blood from the kidneys. Clots of blood come from the ureters, bladder, or urethra.

Odor. The odor of normal urine is sometimes spoken of as aromatic, but generally it is sufficiently characteristic to be best described as urinous. When the urine is concentrated the odor is intensified, and may become unpleasantly strong, like the urine of the horse.

Certain articles of food, such as garlic and asparagus, give the urine characteristic odors. Turpentine, both when taken internally and inhaled, gives to it the odor of violets. The odors of copaiba and of cubebs can easily be detected in the urine of patients who are taking these drugs.

In marked cystitis the natural urinous odor becomes more pungent, and is blended with a strong ammoniacal odor. When much pus is present, and the urine has stood awhile, a putrid odor is developed.

In diabetes mellitus the urine has a sweetish, hay-like odor. In diabetic coma the odor is sometimes that of chloroform, due to the presence of acetone and diacetic acid in the urine. This odor, however, is more likely to be detected in the breath.

2. Chemical Examination of the Urine. Examination of the urine by the unaided senses, which has been dwelt upon thus far, is simply preliminary to an examination by chemical methods and by instruments of precision, particularly the microscope.

Urea. Urea is freely soluble in water, and hence never appears as a sediment. It is the most important final product of nitrogenous disintegration in the body, and is an index of the eliminative power of the kidneys. Usually the density of the urine increases in proportion to the amount of urea contained in it. The average daily amount of urea excreted by an adult man between the ages of twenty and forty years is about 500 grains. The urea, like the total volume of the urine, is subject to variations within the limits of health. It is increased after a meal, especially if the latter be rich in nitrogenous food; after copious ingestion of liquids, and by a close atmosphere. On the other hand, fasting, free perspiration, a loose condition of the bowels, and a vegetable or milk diet diminish the quantity of urea. Again, the quantity varies with the age of the person. According to Ralfe, at five years the amount daily is 180 grains; at 12, 320; at 21, 535; and at 40 years, 555 grains.

A large man will excrete absolutely more than a small man, and a large, muscular man will excrete relatively more than a fat man of the same height.

The excretion of urea is increased in fever and inflammatory diseases; in diabetes mellitus and insipidus; in malaria, pernicious anæmia, and after a crisis in pneumonia. It is increased also by certain bever-

ages, as coffee, and by many drugs, especially those which act as hepatic stimulants.

It is diminished in all forms of nephritis, especially when uræmia results; in acute gout and chronic rheumatism; in disease accompanied by emaciation and cachexia; and in leprosy, pemphigus, melancholia, imbecility, catalepsy, hysteria, and cholera (Saundby).

Estimation of Urea. For the methods employed in the exact quantitative estimation of urea the student is referred to special works on the urine.

For ordinary clinical purposes the apparatus devised by Professor Charles Doremus, and known as his ureometer, gives sufficiently accurate results. The principle upon which it is based is that urea when brought in contact with sodium hypobromite is decomposed, and free nitrogen is eliminated. The nitrogen evolved is the measure of the urea contained in the urine. The instruments are graduated so that each division of the scale represents one grain of urea per fluidounce of urine.

The hypobromite solution is prepared by dissolving 100 grammes of sodium hydroxide in 250 c.c. of water, cooling the solution, and then adding 25 c.c. of bromine.

It is better, however, to freshly prepare the hypobromite solution for each examination. This can readily be done by having a solution of sodium hydroxide containing six ounces to a pint of water. It should be kept tightly corked with a rubber or paraffined stopper. The sodium hydroxide solution is poured into the long tube of the ureometer to the mark =, then one-tenth of its volume of bromine is introduced by means of a pipette, and sufficient water added to fill the long arm and the bend of the tube. The hypobromite solution should fill the tube completely, and any bubbles rising to the top of the tube should be removed before the introduction of the urine. The pipette is then filled with the urine up to the 1 c.c. mark, any urine adhering to its surface being carefully wiped off. The pipette is introduced carefully, so as not to compress the bulb until the point extends as high up as possible beyond the bend. The bulb is now compressed slowly until 1 c.c. of urine has been introduced. Decomposition of the urea occurs and bubbles of nitrogen rise to the surface of the long arm of the tube; when bubbles of gas cease to be evolved the volume of nitrogen gas is read off, and according to the graduations on the tube considered as so many grains of urea per fluidounce of urine, or as so many milligrammes of urea in 1 c.c. of urine, according to whether it is graduated in the English or the metric system.

The Chlorides. The presence or absence of chlorides is sometimes of diagnostic value. They are increased when absorption of exudations or transudations is going on, and in malarial fevers, diabetes insipidus, and Bright's disease. They are diminished or absent in pneumonia during its progressive stage, and in fevers. The chlorine of the chlorides can be detected and roughly estimated by an 8 or 10 per cent. solution of argentic nitrate. A few drops of nitric acid are first added to the urine, to prevent the silver from precipitating phosphoric acid. A single drop of the silver solution mentioned will precipitate the chlorine of the chlorides in a thick white lump, which falls to the bot-

tom of the test-tube, provided the amount present is normal. If, on the other hand, the quantity is diminished to one-tenth per cent. or less, it will not be precipitated in a lump or lumps, but a white cloudiness is produced which renders the whole solution opaque. If no precipitation or cloudiness occurs, the chlorides are absent.

Serum-albumin. Albumin is of common occurrence, but cannot ever be looked upon as a normal constituent of the urine, though its presence by no means indicates disease of the kidneys. The ordinary form is serum-albumin, but other proteids, as globulin, mucin, peptone, albumose, fibrin, and also hæmoglobin methæmoglobin, are found at times. The most trustworthy tests for ordinary albumin (serum-albumin) are : boiling, with the addition of nitric or acetic acid ; overlaying cold nitric acid with urine (Heller's test) ; the picric acid, the potassium ferrocyanide, and the potassium-mercuric-iodide (Tanret's) tests. The author believes that many of the recent tests, such as sodium tungstate, acidulated brine, magnesium nitrate, phenic-acetic acid, and trichlor-acetic acid, are too sensitive and precipitate other substances in the urine, and, therefore, are not reliable for clinical work.

Serum globulin responds to all the following tests for serum-albumin. Its differentiation is not difficult, but usually unnecessary. (See note on page 937.)

Boiling and Nitric Acid Test. A narrow, long test-tube is filled two-thirds full of urine and the upper third boiled thoroughly, and then a few drops of nitric acid are added. Any albumin present will be coagulated and appear as a white cloud, contrasting strongly with the clear unboiled urine beneath it. When the albumin is moderate or even small in amount it can be detected without difficulty by simply holding the test-tube up to the light. When there is only a faint trace present it will be overlooked unless the tube be examined against a dark surface in such a way that the light falls upon it from above, in front, and preferably a little to one side. A cloud may escape detection when looked for by artificial light, but may be distinct by daylight. Serum-globulin is also precipitated by this test. But serum-globulin is not often present by itself, and its significance is not yet understood. It may be detected in any urine, as Roberts points out, by diluting the urine with pure water, the urine then becoming more or less milky. It may be removed from urine by saturating the latter with magnesium sulphate and filtering off the precipitated globulin. The presence of serum-globulin in no way interferes with the test for serum-albumin.

If the urine is opaque from amorphous urates, it is unnecessary to filter them out ; heat much below boiling will dissolve them, the precipitation of albumin occurring later at a higher temperature.

If the urine is alkaline or faintly acid, phosphates will produce a cloud upon heating the urine ; but they are instantly dissolved upon the addition of a few drops of nitric or acetic acid.

Mucin produces an opalescence upon heating with an organic acid, but Saundby declares that it coagulates not in flocculi, as is the case with albumin, but in the form of tiny filaments.

Boiling and Acetic Acid Test. This is preferred by many to the preceding test. It is performed in a similar manner. Acetic acid is,

however, not reliable for acidulation: it precipitates the mucin which is often found in healthy urine, forming a white cloud which is apt to be mistaken for albumin; this is especially true in urines of high specific gravity containing uric acid, urates, or oxalates.

The Nitric Acid (Heller's) Test. This test, while not so delicate as the acetic acid test, is very simple and accurate in its results. Cold nitric acid is poured into a test-tube to the depth of about an inch. The tube is then inclined to an angle of about 45 degrees, and urine allowed to flow gently down upon the acid by trickling along the side of the tube from a pipette or glass tube. At the point of contact of the acid and urine a zone of white, coagulated albumin forms. The test can also be made as follows: Into a short, broad test-tube several cubic centimetres of urine are poured; nitric acid is introduced with a pipette provided with a rubber bulb by passing the pipette through the urine to the bottom of the tube and gently pressing the rubber bulb; care must be taken to withdraw the pipette as the last portion of acid is expelled, so that no air-bubbles will break up the point of contact of the urine and acid. The thickness of the white zone is generally an index of the amount of albumin present. If there is barely a trace of albumin, half an hour may be required to develop any opalescence.

A cloud of urates is sometimes produced and obscures the test. This cloud does not, however, begin at the point of contact and extend upward, but at the upper level of the urine and extends downward, and is dissipated by heat.

Patients who are taking copaiba or cubebs pass a urine which gives a white zone at the point of contact with cold nitric acid, but heat diminishes the opacity, and the precipitate is soluble in alcohol; the odor of the drugs in the urine assists in the detection of their presence.

The Picric Acid Test. This is an extremely delicate test for albumin. A saturated solution of picric acid is allowed to flow down upon and slightly mix with the upper layers of the urine, which half fills a good-sized test-tube. At the point of contact an opaque white zone of coagulated albumin is formed. If no white zone appears, albumin is almost certainly absent. Hence, the picric acid test is a valuable negative test. But, unfortunately, a white zone is formed by peptone, mucin, and various alkaloids, particularly quinine. The white zone produced by the presence of the substances just named disappears upon the application of heat, whereas an opalescence due to albumin becomes diffused throughout the whole urine.

The Potassium Ferrocyanide Test. This test is highly recommended as simple, rapid, and accurate by Purdy, who performs it as follows: Into a test-tube are poured fifteen to thirty drops of acetic acid, and then two or three times that amount of potassium ferrocyanide solution (1 to 20) is added, and the two thoroughly mixed by shaking the tube. The urine is now added to the depth of two-thirds of the test-tube. If any albumin is present, it will be precipitated throughout the whole volume of urine in the form of a milk-like flocculent cloud, more or less according to the amount of albumin present. By this method all modifications of albumin, acid or alkaline, are precipitated and the

precipitation of mucin is avoided. It gives no reaction with phosphates, urates, peptones, vegetable alkaloids, or the acids found in the urine after the ingestion of copaiba, etc. This test may also be performed as follows: An ordinary test-tube is half filled with urine and a drachm or two of the potassium ferrocyanide solution (1 to 20) are added. After thoroughly mingling the reagent and the urine a few drops of acetic acid are added. If albumin is present, it will plainly come into view. This test, therefore, depends upon the production of a cloudiness or milkiness throughout the entire mixture in the tube. To some eyes the albumin is not so readily perceived as in those tests which depend upon the formation of a distinct line at the point of contact.

The Potassium-mercuric Iodide Test (*Tanret's*). The solution is made as follows: Potassium iodide, 3.32 grammes; bichloride of mercury, 1.35 grammes; acetic acid, 20 c.c.; distilled water about 30 c.c. (the potassium iodide and the bichloride of mercury should be dissolved separately in the water and the solution mixed, to which the acetic acid is added and the whole made up to 60 c.c. with distilled water). As thus prepared the test is applied by the contact-method by overlaying the reagent with urine. This test responds to all modifications of albumin, also to peptones and proteoses, as well as to the vegetable alkaloids and acids found in the urine after the ingestion of copaiba, etc. All reactions except those occurring with albumin, mucin, and the acids found in the urine after the ingestion of copaiba, etc., disappear with heat. It is a very good and delicate control-test for albumin. The solution, however, is of a yellowish hue, quite similar to the color of urines of low specific gravity. This sometimes renders the line of contact difficult to perceive.

It is well to follow a routine method in testing for albumin: first, by boiling and the addition of nitric acid, and then the contact (Heller's) test; if there is doubt, either the potassium ferrocyanide or picric acid test; finally, Tanret's solution will reveal minute quantities of albumin, and may be used as a confirmatory test.

In all the tests for albumin mentioned a clear urine is necessary, especially when the amount of albumin is very small. This can be obtained by filtration when the opacity is due to pus, blood, mucus, and uric acid; and, more effectively, by the addition of a small quantity of sodium hydroxide, warming slightly, and filtering. If the filtrate is not clear, a few drops of magnesium fluid (sulphate of magnesium, pure ammonium chloride, and pure liquor ammoniæ, of each 2 drachms; distilled water, 2 ounces), as recommended by Hoffmann and Ultzmann, may be added, and the urine again warmed and filtered.

Résumé of Tests for Albumin.

I. The heat test.

A. Method: Albumin is precipitated on boiling.

B. Exception: 1. In alkaline urines albumin may be overlooked from the formation of soluble potassium and magnesium compounds. When patients are taking alkaline salts the test may be fallacious.

2. An excess of acid may also interfere with the test.

3. Feebly alkaline or neutral urines produce a precipitate of earthy phosphates, but it is instantly soluble in a small quantity of acid.

4. Patients on a vegetable diet pass urine containing carbonates which precipitate with heat. The addition of an acid causes great evolution of gas.

II. The heat and acetic acid test.

Method: Determine the reaction of the urine. If alkaline, make faintly acid with acetic acid; then boil and add a little more acetic acid. If there is no precipitate, boil again. The acetic acid precipitates nucleo-proteids, which are excluded by the methods above described.

III. The heat and nitric acid test.

A. Method: Bring the urine to the boiling-point and add nitric acid, drop by drop, shaking the mixture between each addition. A small precipitate is thrown down even if a very small amount of albumin is present. The nitric acid should not exceed more than one-tenth of the volume of urine examined. The urine must not be heated after the addition of the acid.

B. Exceptions: 1. In concentrated urines, uric acid or its salts may precipitate. Distinguish from albumin by filtering off the precipitate and testing it by the biuret reaction, or dilute the urine with an equal volume of water when uric acid will not precipitate.

2. Resin acids in turpentine, benzoin, cubebs, and other balsams, if present in the urine, are precipitated by nitric acid. Distinguish from albumin by adding one or two volumes of alcohol when the solution is cool. The precipitate of resin acids is dissolved.

3. In urines containing biliverdin a precipitate is formed. Distinguish from albumin by adding alcohol, which dissolves biliverdin.

IV. Cold nitric acid test.

A. Method: Pour the urine gently on the nitric acid. The albumin coagulates in the presence of an excess of strong nitric acid. A ring appears at the surface of contact if albumin is present. A second ring may be seen $\frac{1}{10}$ to 1 cm. above the junction, due to nucleo-proteids. Distinguish from albumin by repeating the test with urine diluted with two or three volumes of water. The albumin ring diminishes and the nucleo-proteid ring is unchanged or increased. A haze due to nucleo-proteid may form, and also continue after dilution.

B. Exceptions: 1. In concentrated urines a secondary ring due to uric acid may form above the junction. It is soluble on gently heating, and does not form when the urine has been diluted.

2. In highly concentrated urine a precipitate of nitrate of urea may fall. Distinguish by its crystalline nature.

3. Resin acids cause a precipitate of uniform cloudiness. Distinguish by solubility in alcohol.

4. In highly colored urines the urinary pigments form a colored ring at the plane of contact, and in bilious urines the play of colors, as in Gmelin-Malin-Heintz's test for bile, is seen.

5. The urine of patients taking alkaline iodides gives a dense brown ring of iodine. Distinguish by adding a few c.c. of chloroform and mixing them. A violet tinge is imparted to the liquid.

6. Albumoses are precipitated, as well as all forms of albumin. Distinguish by the previously mentioned tests. Peptone and vegetable alkaloids are not precipitated.

V. The potassium ferrocyanide and acetic acid test.

A. Method: It is best performed as a ring test. The urine should be carefully run into a mixture of twenty or thirty drops of acetic acid and sixty or ninety drops of saturated solution of potassium ferrocyanide. A white ring forms at the junction if albumin is present. With small amounts of albumin the ring takes some minutes to form.

B. Exceptions: 1. Albumoses are precipitated. They are soluble in excess of acetic acid. They disappear on heating and reappear on cooling.

2. Resin acids give a precipitate which is soluble in alcohol.

3. Phosphates, urates, alkaloids, and peptones are not precipitated.

VI. Roberts' brine test.

Saturated sodium hydrate solution with 5 per cent. hydrochloric acid. It does not darken the urine nor precipitate uric acid.

A. Method: Use the ring test, which shows albumin and albumoses.

B. Exceptions: Resin acids precipitate. Distinguish by dissolving in alcohol.

VII. The salt and acetic acid test.

The acetic acid is substituted for HCl, and a large excess of salt solution used.

A. Method: The salt solution is first added to the urine and thoroughly mixed. Acetic acid is then poured in. Nucleo-proteids are not precipitated. (All other forms of albumin are precipitated.) Salt and vinegar may be used, and the mixture heated in a metal spoon.

B. Exceptions: 1. Albumoses form and disappear on heating, to reappear on cooling.

2. If albumoses and albumin appear together, boil for a short time and filter the hot fluid through a warm filter. The clear filtrate becomes turbid from albumoses as it cools.

3. Resin acids and uric acid are precipitated, the latter only in concentrated urines, and after standing. Distinguish by the usual tests.

V. and VI. do not generally precipitate nucleo-proteids. With VII., if equal parts of urine and salt solution are used with a few drops of acetic acid, nucleo-proteids are not precipitated. The solution must be boiled when test VII. is employed.

VIII. Salicylsulphonic acid.

All forms of albumin are precipitated. The precipitate becomes flocculent on heating. If the urine is alkaline more of the reagent is needed than if acid. Phosphates, urates, bile, alkaloids, and drugs do not give a reaction.

A. Method: After adding the solution to the urine heat and allow to stand.

B. Exceptions: Albumoses are precipitated, but disappear on heating and reappear on cooling.

IX. Trichloracetic acid.

Exceptions: 1. Precipitates uric acid when in excess. Distinguish by heating, which dissolves the acid, or dilute the urine before applying the test.

2. Nucleo-proteids give an opalescence. Albumoses are not precipitated.

X. Picric acid.

A. Method: A saturated solution of picric acid must be used alone, in combination with HCl, or with acetic acid. Value doubtful.

B. Exceptions: Uric acid, creatinin, nucleo-proteids, alkaloids, potassium salts, and albumoses are precipitated.

XI. Millard's reagent.

Value doubtful. Precipitates albumoses, nucleo-proteids, alkaloids, and resin acids. Distinguish by usual tests.

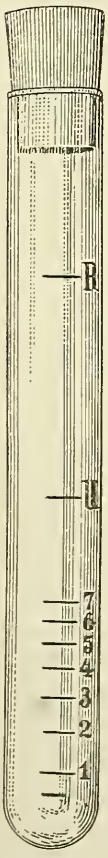
XII. Tanret's reagent.

Very delicate. Precipitates all forms of albumin, albumoses, nucleo-proteids, peptones, alkaloids, and resin acids. Distinguish by usual tests.

XIII. Spiegler's reagent.

Delicate. Precipitates albumin, albumoses, and nucleo-proteids, but not peptones.

FIG. 205.



Esbach's albuminimeter.

XIV. Acetic acid.

Method: Filter the urine and add acetic acid to a portion, pouring the two in the tube held against a black background. Albumin and nucleo-proteids are precipitated. Distinguish by diluting the filtered urine with two or three volumes of distilled water, then add acetic acid, and compare the precipitate with that in an undiluted specimen. A nucleo-proteid precipitate will increase in intensity. An albumin precipitate will diminish or remain unchanged.

Salicylsulphonic acid is the most delicate test. An objection to it is the fact that it precipitates nucleo-proteids. Control the test by Heller's cold nitric acid test, from which the nucleo-proteids are removed, as above described.

The *quantitative* estimation of albumin is of some importance. The most direct method is by coagulating the albumin by boiling, collecting it upon a weighed filter, washing with water and finally with alcohol, drying and weighing it. Such a process, however, consumes too much time for clinical purposes, and it is not faultless. An approximate estimation may be made by boiling the urine in a test-tube, adding several drops of nitric acid, allowing the albumin to settle, and then comparing the depth of albumin with the height of the column of urine. In this way we may speak of urine furnishing one-tenth or one-quarter of its bulk of coagulated albumin.

Esbach has invented an albuminimeter (Fig. 205) which gives good results. The solution used to precipitate the albumin consists of 10 grammes of picric acid and 20 grammes of citric acid, chemically pure and dry, dissolved in 900 c.c. of hot water; and after cooling, diluting the solution to 1000 c.c. The urine is diluted with a definite amount of water if it contains too much albumin. The albuminimeter is filled to the mark U with urine, and from that mark to R with the

reagent. The tube is then corked with a rubber stopper, turned upside down ten times, so as to mix the urine intimately with the reagent, and then allowed to stand undisturbed for twenty-four hours. At the end of this time the depth of the sediment of coagulated albumin is ascertained by observing where the top of the sediment comes in contact with a mark on the scale on the tube. Each mark corresponds to one-tenth per cent. of albumin.

This estimation, as already stated, is not absolutely accurate. Nevertheless, if used systematically, and always in the same way, relative values will be obtained, and these are the most important in watching the progress of a case, as they give positive information regarding an increase or diminution of the amount of albumin in the urine. It scarcely need be said that the urine tested must be a portion of the whole twenty-four hours' urine.

The estimation of the amount of albumin is also readily made with the centrifugal machine: to 10 c.c. of the albuminous urine are added 3.5 c.c. of potassium ferrocyanide solution (1 to 10) and 1.5 c.c. of acetic acid; the mixture is then revolved in the machine about three minutes, and the amount of precipitate read off.

Albuminuria. Albuminuria is not indicative of disease of any one organ, nor does it point to any general pathological condition. It occurs as follows:

1. In diseases of the kidney: acute and chronic Bright's disease, amyloid disease, tuberculosis, cancer, abscess, and calculus.

2. In disturbances of the circulation: diseases of the heart and chronic pulmonary diseases, as emphysema; obstruction of the renal arteries or veins, cirrhosis of the liver, peritonitis, pregnancy, abdominal tumors; in passive congestions due to great weakness; in anæmia and Graves' disease.

3. In febrile and inflammatory diseases: in the eruptive and infectious fevers, and in rheumatism, diphtheria, pneumonia, and gout.

4. In blood diseases: purpura, leucocythæmia, and scurvy.

5. From the poisonous action of drugs: lead, turpentine, and others.

6. In nervous disorders: concussion of the brain and cerebral hemorrhage, epilepsy, tetanus, and delirium tremens; as Pye-Smith remarks, it is doubtful whether albuminuria is caused by the nervous diseases.

7. Local extra-renal affections: pyelitis, cystitis, gonorrhœa, and leucorrhœa.

8. Functional. In young persons, particularly of the male sex, there occurs occasionally slight albuminuria after exercise, a special diet, or a cold bath. Albumin may be found after rising in the morning, or early after dinner, or toward evening. On account of its occurring only at certain times it has been called "cyclical" or "intermittent," and because there is no evident disease present, it is occasionally spoken of as "physiological" albuminuria.

Goodhart examined the urine of 1500 individuals and noted albumin in 272, or in 20 per cent. In 39 cases the albuminuria could not positively be said to be due to disease of the kidney. Of these 39, 26 were males and 13 females. In 32 of the 39 cases it was temporary, and in most of them it had disappeared within forty-eight hours, or sooner.

In 2 cases there were oxalates in the urine; in 1 hæmoglobinuria; in 8 leucorrhœal discharges and discharges from other parts of the genital passages (see division 7); and in 17 a markedly neurotic temperament. These last he thinks the most typical cases of intermittent albuminuria; on the whole, he regards the condition as less common than has been supposed.

One variety of functional albuminuria is apparently due to the irritation of the kidney produced by the excretion of oxalates and uric acid. The urine is of increased density, 1028, 1030 or higher, and contains uric acid or oxalate of lime, or both, and cylindroids. Tube-casts are very uncommon. The albuminuria usually disappears under proper diet. This condition is sometimes called "morbus Da Costæ."

It is conceded that there may be albuminuria of renal origin without renal disease, but the diagnosis must be by exclusion, and can be reached safely only after extended observation. The most important elements in the diagnosis are: the age of the patient, unimpaired general health, a specific gravity of the urine normal or above normal, the fact that the albuminuria is influenced by diet and exercise, and that it tends to disappear under suitable regimen. The prognosis is favorable.

Mucin. Nucleo-albumin, or nucleo-proteid, is nucleic acid and chondro-sulphuric acid combined with a proteid. Sometimes, pathologically, tauro-cholic acid enters into the combination. This is not true mucus, but urinary mucus. It is present in the urine in health, being especially abundant in women from the admixture of the vaginal secretion, and in excess in inflammatory conditions of the urinary tract. It is distinguished from albumin by the fact that it gives a precipitate upon the addition of vegetable acids, as acetic or citric. The precipitate is increased by removing the salts of the urine by dialysis, or by dilution of the urine, with two or three volumes of distilled water, diminishing thereby the relative proportion of salts to mucus. It is precipitated by dilute mineral acids, but is soluble in concentrated mineral acids or dilute alkalies.

According to Roberts, the best method for the detection of mucin is by means of a saturated solution of citric acid, employed in the same manner as the contact-method of applying the nitric acid test for albumin. A small quantity of the urine is first put in a test-tube, and citric acid allowed to trickle down the sides of the tube until it forms a distinct layer below the column of urine. If mucin is present there will gradually appear an opalescent zone immediately above the layer of acid. Acetic acid, mixed with one-third of its volume of glycerin, answers admirably as a test for mucin. Sometimes, when mucin is very abundant, the addition of an excess of acetic acid produces a marked milkiness in the urine, which is not discharged by boiling the liquid.

Blood. Urine containing blood is usually red in color or reddish-brown and opaque, but it may be chocolate-brown if the blood is present in large quantity and has been acted upon by the urine. Such urine necessarily contains albumin.

Blood occurs in the urine from (1) *diseases of the kidney and urinary passages*, among which are Bright's disease, acute congestion of the

kidney, renal calculus, cancer, tuberculosis; from ureteritis, cystitis, and urethritis, and from injuries; (2) from *general diseases*, such as the eruptive and intermittent fevers, scurvy, purpura, peliosis rheumatica, leucocythæmia, cholera; (3) from *adjacent organs*, as in menstruation and hemorrhage from the uterus; (4) from the *toxic action of drugs*—cantharides, turpentine, and other violent irritants of the kidney; (5) *vicariously*—occasionally menstruation fails to occur and hæmaturia replaces it. The same is true of bleeding from piles. Latour has reported a case of asthma which subsided suddenly upon the appearance of hæmaturia.

The chemical tests for blood are the same as those for its coloring-matter, and will be referred to under Hæmoglobin.

Hæmoglobin. Hæmoglobin is, of course, present whenever blood is, but sometimes it occurs independently of hæmaturia. Thus, it is found in grave infectious diseases, as the result of toxic action of drugs, such as carbolic acid, and in an independent disease known as paroxysmal hæmoglobinuria. A suitable test consists in adding one or two drops of freshly prepared tincture of guaiac to about one drachm of urine, then shaking the mixture and adding several drops of a solution of hydrogen peroxide. If blood-coloring matter be present, a beautiful blue coloration will be produced.

The same test answers for methæmoglobin and hæmatin.

Paroxysmal Hæmoglobinuria. The urine contains blood, or only the coloring-matter of the blood is present. Hæmoglobinuria is more frequent in adult males; it may be excited by a cold bath, or exposure to cold, or by exertion. It is sometimes associated with Raynaud's disease. The attacks come on suddenly, often preceded by chills. Sometimes fever accompanies the disease. Vomiting and diarrhœa occur with hæmoglobinuria. Pain in the loins is sometimes complained of. The paroxysm may last a day or two, or two or three paroxysms may occur in the course of twenty-four hours.

Albumose (Proteoses, propeptone or Meissner's peptone). Formerly the reactions which we know now determine the presence of the albumoses were thought to indicate the presence of peptone. The latter substance is extremely rare. Recent chemical investigations show that that which was called peptonuria is truly albumosuria. Albumose has been found in the urine in osteomalacia and diseases of the medulla of bone and in myxœdema. When persistent it is in all probability due to multiple tumors of the bones or to myxœdema. The albumosuria may be considered as primary. Transitory albumosuria is found in pneumonia, deep-seated suppuration, meningitis, and in dermatitis, intestinal ulcer, measles, scarlatina, and mental diseases. Its frequent occurrence renders its presence of not much diagnostic value. According to von Jaksch, its presence may indicate that a suppurative process exists. In the diagnosis of epidemic cerebro-spinal from tubercular meningitis transitory albumosuria speaks for the former if no ulcerative tuberculous process exists elsewhere. Urine containing it does not respond, at first, to the heat and nitric-acid test, but on cooling a precipitate forms which responds to the *biuret test.* (In this test the urine is first treated with about one-half its volume of sodium hydrox-

ide solution, and then a 1 per cent. solution of cupric sulphate is added, drop by drop. If albumose is present, the resulting cupric hydroxide is dissolved, and the fluid becomes of a violet-red color.) The probability of the presence of albumose is strengthened if a turbidity occurs with the acetic acid and potassium ferrocyanide test (acetic acid, specific gravity 1064, to which a few drops of a 10 per cent. solution of potassium ferrocyanide have been added), and also with the biuret test, applied directly to the urine itself. Albumin also responds to this test.

The best test for albumoses is that of Hofmeister, modified by Salkowski. Twenty to fifty c.c. of urine are acidified with acetic acid and then added to an equal quantity of a saturated solution of common salt, boiled and filtered. In this manner the urine is freed from albumin; the albumin remaining as a filtrate while the albumose is re-dissolved. The filtered fluid containing the albumose is placed in a beaker and a few drops of HCl added. A solution of phosphotungstic acid is added and the precipitate consolidated by heat into a coherent mass. Then pour off the supernatant fluid; wash the precipitate with water and dissolve in a solution of soda (sp. gr. 1.16), which is added, drop by drop, until dissolved. If the solution is blue it is to be gently heated, to decolorize. A few drops of a 1 per cent. solution of sulphate of copper is added to the soda solution. If a red or violet color, the *biuret reaction* results, albumose is present.

The late Dr. N. A. Randolph suggested the following test, which is given by Tyson: To 5 c.c. of urine, which must be cold and faintly acid, add two drops of a saturated solution of potassium iodide and then three or four drops of Millon's reagent. If albumoses or bile-acids are present, a yellow precipitate falls. If the yellow precipitate does not respond to the test for bile-acids, it is due to albumose.

Sugar (Glucose). Next to albumin, sugar is the most important abnormal constituent of the urine. It is not present in normal urines in quantities that can be detected by ordinary clinical methods. The best tests for its detection are Fehling's test and the fermentation test.

Fehling's Test. Fehling's solution is prepared by dissolving 34.652 grammes of pure crystallized cupric sulphate in about 200 c.c. of water. About 173 grammes of sodic potassium tartrate (Rochelle salt) are dissolved in about 480 c.c. of sodium hydroxide solution of 1.14 specific gravity. The cupric sulphate solution is added slowly to the sodic potassium tartrate solution, stirring constantly until all of the cupric sulphate solution has been added. The bluish-white precipitate of cupric hydroxide which first forms will, on stirring the liquid, be completely dissolved. The blue liquid is then diluted with water to exactly 1000 c.c. One c.c. of this solution will be reduced by 0.005 of a gramme of glucose. Fehling's solution is prone to decomposition, and as much as possible, to avoid the occurrence of decomposition, it is best to keep the cupric sulphate and sodic potassium tartrate solutions in separate bottles closed with rubber stoppers. To accomplish this, the 34.652 grammes of cupric sulphate are dissolved in water and diluted to 500 c.c., and the sodic potassium tartrate is dissolved in water and diluted to 500 c.c., and the two solutions preserved in separate bottles closed with rubber stoppers. The solution, prepared in

this manner, is made ready for use by mixing one volume of the cupric sulphate solution with an equal volume of the sodic potassium tartrate solution. The resulting liquid will be Fehling's solution, and 1 c.c. of it will be equal to 0.005 of a gramme of glucose.

Certain precautions are necessary in the application of this test. 1. Any albumin present must be removed by boiling and filtration. 2. The Fehling solution, diluted with 4 to 5 volumes of water, must be boiled first and the urine added to it; the urine must not be boiled first and the Fehling solution added to it. Boiling the reagent first is a test of its stability: if a precipitate occurs, the solution is unfit for use. As Wormley correctly says, a precipitate is more likely to occur when the Fehling solution has been diluted with four or five times its volume of water than on boiling the undiluted solution. 3. Prolonged boiling is to be avoided. The solution is to be heated to the boiling-point and the urine then added; if no precipitate indicating sugar occurs until urine is added almost equal in volume to that of the reagent, the mixture should be again heated to the boiling-point and then set aside. 4. When the earthy phosphates are abundant, it is well to get rid of them by adding a small quantity of sodium hydroxide and filtering before applying the sugar test. 5. Changes in color may occur from the presence of urea, uric acid, and extractives. These changes can be obviated, when necessary, by the method proposed by Seegen, who recommends repeated filtering through animal charcoal until the urine is rendered colorless. Fehling's test is then applied to the filtered urine.

The method of applying Fehling's test is as follows: Fehling's solution is poured to the depth of about one-quarter of an inch into a test-tube, and diluted with four or five times its volume of water, and heated until it begins to boil; then one or two drops of the suspected urine are added. If it be ordinary diabetic urine, the mixture, after an interval of a few seconds, will suddenly turn to an intense opaque yellow or reddish-brown color, and in a short time an abundant yellow or reddish-brown precipitate falls to the bottom. If, however, the quantity of sugar present be small, the suspected urine is added more freely, but not beyond a volume equal to that of the diluted Fehling's solution employed. In this latter case it is necessary to raise the mixture once more to the boiling-point. It is then allowed to cool slowly. If no cuprous oxide has been thrown down when the liquid has become cold, then the urine may be pronounced sugar-free.

Sir William Roberts has recently pointed out the value of repeated filtration through animal charcoal of urine which reacts doubtfully to the test for sugar; by this filtration the urates, uric acid, and other normal constituents of the urine, which have more or less power of reducing Fehling's solution, are removed, while the sugar passes through and is found in undiminished quantity in the filtrate.

The test is made as follows: A test-tube is charged with Fehling's solution to the depth of about one-quarter of an inch, diluted with four or five times its volume of water, and brought to the boiling-point; the urine, filtered through charcoal, is added to the depth of about two inches, and the two fluids mixed. The flame of a lamp is then applied to the upper half of the column of liquid, and this is boiled for a couple

of seconds. If sugar is present, the upper half loses its blue color and assumes a yellowish tinge, and the earthy phosphates which are thrown down in light flakes by the alkali of the test are tinted more or less of a gold color by the precipitation on them of the cuprous oxide.

The Fermentation Test. This is based upon the fact that sugar by fermentation with yeast breaks up into alcohol and carbon dioxide. It is a reliable but not a very delicate test for sugar.

A piece of yeast-cake the size of a pea is added to a test-tube full of urine. The open end of the tube is inverted under water in a saucer or beaker. If sugar is present in amounts larger than two and a half grains to the ounce, bubbles of carbon dioxide collect at the upper part of the tube after standing twelve hours in a temperature of about 90° F.

The Phenyl-hydrazin Test. Von Jaksch believes this test to be a very accurate one. About two grains of phenyl-hydrazin hydrochloride and about three grains of sodium acetate are put into a test-tube half-full of water. The contents of the tube are heated and the tube filled with the suspected urine. The tube is kept for fifteen or twenty minutes in boiling water, and then put in a vessel of cold water. When a large amount of sugar is present a deposit of yellow, needle-like crystals is visible to the naked eye; but when only a small amount is present, the sediment must be examined under the microscope. The crystals appear singly, or in sheaves and fine radii. Yellow plates and brown balls do not indicate sugar. (Plate XLVI.)

Quantitative estimation of sugar can be made with Fehling's solution by using a burette and measured quantities of urine and reagent. Wormley recommends a method which answers very well for office-use: One cubic centimetre of Fehling's solution is diluted in a large test-tube with four cubic centimetres of distilled water, and boiled. One-tenth of a cubic centimetre of the suspected urine is then added from a graduated pipette. Heat is then applied, the precipitate watched, and then another one-tenth cubic centimetre added, and heat again applied. The addition of one-tenth of a cubic centimetre, followed by heat, is continued, until it is found, after proper subsidence, that all the color is removed from the diluted Fehling's solution. If in doing this one cubic centimetre of urine has been added, it will have contained just 0.5 per cent. of sugar. If more than one cubic centimetre, it will have contained less than 0.5 per cent. If exactly two cubic centimetres are used, it will have contained exactly 0.25 per cent. If one-tenth of a cubic centimetre has been used, the urine will have contained 5 per cent. of sugar. If the quantity of sugar in the urine is large, the urine should first be diluted with a measured volume of water, allowance being made for this in the estimation.

When the quantity of sugar is relatively large fermentation is the simplest and most trustworthy method. Roberts has shown that saccharine urine loses by fermentation one degree in density for every grain of sugar contained in an ounce of urine. For example, if the urine before fermentation had a specific gravity of 1040, and after fermentation a specific gravity of 1010, then the urine contained 30 grains of sugar to the ounce. In the application of this method, about four ounces of diabetic urine are put in a twelve-ounce bottle, and a piece

PLATE XLVI.

Crystals of Phenyl-glucosazone.

(Oc. 4, Obj. D.) Drawn by J. D. Z. Chase.

of Vienna yeast, about the size of a pea, is broken up and then added to it. This bottle is closed with a perforated cork to allow the CO_2 to escape, and stood aside in a warm place to ferment. Beside it is placed a tightly corked four-ounce bottle filled with the same urine, but without any yeast. In about twenty-four hours the fermentation will have ceased. The specific gravity of the fermented urine is then taken and also that of the unchanged urine. Every degree of loss in density represents one grain of sugar per ounce of urine.

Diabetes Mellitus. The occurrence of any of the following conditions should lead to an examination of the urine for *sugar*, and an estimation of the quantity of urine passed in twenty-four hours, apart from the routine examination, which should be made in every case of chronic disease or of obscure acute disease: 1. Muscular weakness without cause. The weakness is progressive and rapidly advances to an extreme degree. 2. Emaciation. In young subjects this is rapid in cases of diabetes. In older patients it is not so striking, particularly if the gouty diathesis is present. 3. Thirst. This is a symptom which is of common occurrence in diabetes, and is most distressing. If the amount of fluids taken be compared with the amount of urine excreted, it will be found that the two bear a definite ratio. The thirst is greater immediately after meals, although the patient does not necessarily have indigestion. 4. Hunger. Excess of appetite, boulimia or polyphagia, also occurs in diabetes. The amount of food that is taken is sometimes enormous, and the ravenous way it is devoured is revolting. 5. Loss of sexual power.

The five symptoms just mentioned, with increased frequency in micturition, are the common symptoms of diabetes mellitus. They may develop gradually. In rare instances the onset is sudden. The occurrence of these symptoms should lead at once to an examination of the renal secretion.

Three special characteristics of the urine are observed. *A.* The *amount* is increased, so that from six to ten pints, or even as much as thirty to forty pints, are passed in twenty-four hours. *B.* The *specific gravity* ranges from 1025 to 1045, and may even be higher. *C.* The presence of *sugar*. The sugar is detected by the ordinary tests. (See Examination of Urine.) In addition the urine is usually of pale color, of a sweetish odor and acid reaction.

In addition to thirst and increased appetite, some gastro-intestinal symptoms may be of diagnostic importance. Of these, first, the appearance of the *tongue* is characteristic. It is dry, red, and glazed. The dryness is increased because of the scanty flow of *saliva*. The *gums* are swollen and spongy, and marginal gingivitis and *stomatitis* are often present. There are no marked dyspeptic symptoms. *Constipation* is of common occurrence.

In diabetes other secretions diminish. *Perspirations* do not occur, except in inflammatory complications. The *skin* is harsh and dry. As the disease progresses the *heart's* action becomes weak and the *pulse* frequent, with lowered tension. The *temperature* of the body is usually below normal.

Diabetes may occur at any age, but is most frequent in adult life.

In young adults the symptoms are more pronounced, and the duration shorter. In patients past middle life the disease may continue for a number of years without marked interference with the health and nutrition.

While the symptoms just mentioned should lead to an examination of the urine, diabetes mellitus may not be suspected by any of the usual objective or subjective symptoms. It may happen that none of these symptoms is sufficiently marked, and that only by routine examination of the urine, or by the occurrence of affections known to be associated with sugar in the urine, is the disease discovered.

Of the complications which should lead to the suspicion of sugar in the urine the following are the most important :

1. *Cutaneous Complications.* Boils and carbuncles should always lead to an examination of the urine. Pruritus and chronic eczema may have diabetes in the background. Gangrene of the extremities, chiefly of the feet and legs, and gangrene in other situations, is of common occurrence in the course of diabetes.

2. *Lung-complications.* Tuberculosis, both of the chronic and the acute pneumonic type, is frequently associated with diabetes. Lobar pneumonia is apt to occur. In all cases of pneumonia the urine should be examined for sugar. Its presence would modify the prognosis of an otherwise moderate case. Gangrene is likely to ensue in the acute and chronic lung affections. Gangrene of the lung in the course of diabetes may be latent, and recognized only by the odor and the character of the expectoration, or it may run an acute febrile course.

3. *Nervous Symptoms. Diabetic coma* may develop in the course of the disease. In young subjects, particularly, the occurrence of coma should lead to a suspicion of diabetes. Such coma may occur before the disease has been recognized. The coma may follow an attack of fainting and prostration, with stupor, which deepens into complete unconsciousness. It may be preceded by nausea and vomiting or by the lung-complications previously mentioned. This form of coma is usually preceded by extreme dyspnœa, by agitation, pain in the head, and some delirium. The pulse becomes rapid and feeble, and coma develops gradually. For this form of coma the term *acetonæmia* is used. The breath is of a peculiar sweetish odor, due to acetone, and this compound is detected in the urine. Coma may occur without any premonitory symptoms whatsoever, the patient reeling for a short time, and complaining of pain in the head as if intoxicated.

Peripheral neuritis should always lead to an examination of the urine. It may be limited to one group of nerves, or may be more or less general, with symptoms like those of locomotor ataxia, as the lightning-pains, abolition of reflexes and loss of power in the extensor muscles. Diabetic patients are also subject to neuralgia, and to peripheral hyperæsthesia and paræsthesia, probably due to neuritis. The neuritis may be so extreme as to lead to paraplegia.

4. *Eye-symptoms.* A curious symptom of diabetes is the occurrence of *cataract.* This may develop at any age, and is often rapid in its course. Cataract or alterations of vision should always demand an examination of the urine. Diabetic *retinitis* is sometimes present. Atrophy of the optic nerves, or muscular insufficiencies, may take

place, the latter causing the pronounced symptoms of eye-strain. Ringing in the ears, deafness, the occurrence of acute otitis, are phenomena which arise in the course of diabetes.

Diagnosis. Sugar in the urine occurs temporarily when there is an excess of saccharine diet, or when there is functional disorder of the liver. The sugar is small in amount, and the glycosuria is transient. The diagnosis of true diabetes is not difficult, although the disease may be overlooked unless the habit, previously insisted upon, of constant urinary examinations is fully developed.

Indican. An excess of indican in the urine is known as indicanuria. The substance is detected by several methods. Jaffe's test: Equal volumes of hydrochloric acid and urine are mixed. By means of a glass pipette a solution of sodium hypochlorite is dropped into the fluid. An indigo-blue color is produced if indican be present. The hypochlorite must not be added in excess. A quantitative determination is made by the colorimetric process of Salkowski. A rough analysis is first made, to determine the quantity of calcium hypochlorite, which causes the greatest amount of indigo to unite with it. If the urine contains much indican, a small portion, as 2.5 to 5 c.c., is diluted with water to 10 c.c. If there is but little indican, 10 c.c. of the urine are used without dilution. An equal quantity of hydrochloric acid is added. To this the amount of hypochlorite solution with which, in the first test, indigo combined in the greatest amount is added. Then the liquid is neutralized with sodium hydroxide, then enough sodium carbonate is added to make it alkaline. The indigo-blue is thus precipitated and collected on a filter. The precipitate is repeatedly washed with water until the alkaline reaction disappears. The filtrate is dried and extracted by heating with chloroform, until the latter no longer assumes a blue color. The chloroform extract is increased to a round number of c.c. by the addition of chloroform, and placed in a vessel with parallel sides. The intensity of its color is compared with a freshly prepared chloroform solution of indigo blue of known strength. To one or other of these chloroform is added until the tint of both is the same. The quantity of indigo-blue derived from the urine is determined, and its percentage calculated from the intensity of color and strength of the solution of indigo of known strength. Five to twenty milligrammes of indigo-blue are passed in twenty-four hours in health. Indican is increased by animal diet—an increase which, under other circumstances, is pathological. Its presence is a sign of intestinal putrefaction. It may accompany a decomposition of albumin in cavities. It is present in empyema and in puerperal peritonitis. By detection of its presence in these diseases cavities due to pus may be distinguished from those due to other causes. Indican is increased in acute diarrhœa and in intestinal tuberculosis. Von Jaksch states that large quantities of indican in the urine imply that abundant albuminous putrefaction or putrid suppuration is in progress in the system. It must not be forgotten that indicanuria will often arise in simple constipation.

Bile-pigments and Bile-acids. Bile-pigment or bilirubin occurs in the urine in cases of hepatogenic and hæmatogenic jaundice and in portal thrombosis.

Gmelin's test and its modifications are the ones usually employed. A small quantity of nitric acid, to which some nitrous acid has been added, is put into a test-tube and then gently overlaid with urine. If bile-pigment is present, a series of colors appear at the junction of the two fluids—green, blue, violet, and yellow. A green color (biliverdin) must be present to prove the existence of bile-pigment.

The same test may be applied by placing a few drops of the acid upon one side of a plate and the urine on the other, and then allowing the two to run together. The play of colors takes place, as before, at the line of junction of the acids and urine.

Rosenbach's modification is an improvement. About 200 c.c. of urine are allowed to flow through pure white filter-paper, and then a drop of nitric acid is placed upon the paper saturated with the urine. The colors appear as before described.

A very simple test consists in allowing a few drops of the acid to fall into a test-tube full of urine. If bile-pigment is present, a green color appears at the line of junction of the two fluids. This test may fail, however, if only small quantities of bile-pigment are present.

The tests for bile-acids are either too elaborate or too unsatisfactory for clinical use.

Pus. Pus is found in the urine whenever there is suppuration or a catarrhal condition of the genito-urinary tract. Hence, it occurs in abscess of the kidney, pyonephrosis, pyelitis, tuberculosis, cystitis, gonorrhœa, leucorrhœa, etc. It is relatively common in women, from a catarrhal condition of the vulva and vaginal mucous membrane, and is, therefore, of less significance than in men. Urine containing much pus is slightly albuminous; but frequently pus-cells are found in urine which gives no reaction for albumin.

The chemical test for pus is its conversion into a tenacious (gelatinous), glairy mass by boiling with caustic potash.

Acetonuria. An excess of acetone occurs in the following diseases: (1) In diabetes; (2) in cancer independent of starvation; (3) in starvation; (4) in certain psychoses; (5) in auto-intoxications; (6) in derangement of digestion; (7) in fevers. In diabetes acetone indicates an advanced stage of the disease. Lieben's test for acetone is as follows: To several c.c. of urine a few drops of iodo-potassium iodide solution and sodium hydroxide are added. If acetone is in excess, the precipitation of iodoform takes place, which may be recognized by its odor.

Diaceturia. Diacetic acid is found in the urine in diabetes, in fevers, and in auto-intoxications. It is common with children in fever. It is of grave significance when in the urine of adults. Coma usually follows its occurrence in the urine in fevers and in diabetes. Test: A concentrated solution of ferric chloride is cautiously added to the urine. If a precipitate be formed, it should be removed by filtration and more ferric chloride added to the filtrate. If diacetic acid be present, the liquid will become claret-red in color.

Hæmatoporphyrinuria. This is a rare constituent of the urine derived from the blood. It is said to be a form of hæmatin freed from iron. Nakarai thinks that the occurrence of hæmatoporphyrinuria is

constant in lead-poisoning, and occurs with some degree of frequency in intestinal hemorrhage.

Alkaptonuria. The substance in the urine which has been identified as alkapton is also known as pyrocatechin (Ebstein and Müller, Virchow's *Archiv*, Bd. lxv. s. 394), protocatechinic acid (Smith, *Dublin Journ. Med. Sc.*, 1882, vol. i. p. 465), urrhodinic acid (Kirk, *British Medical Journal*, London, 1886, vol. ii. p. 1017), glycosuric acid (Marshall, *Medical News*, Philadelphia, 1887, p. 35), uroleucinic and uroxanthinic acids (Kirk, *British Medical Journal*, London, 1888, vol. ii. p. 232), and homogentisinic acid (Baumann and Wolkow, *Ztschr. f. physiol. Chem.*, Strassburg, Bd. xv. s. 228). It reduces copper, as does glucose, and its occurrence is of interest, because the presence of the substance has led to the diagnosis of glycosuria in many instances, in consequence of which persons have been refused life insurance. The urine containing this substance deepens in color on exposure to air. It is of a peculiar aromatic odor, and reduces cupric salts rapidly. There is, however, no reaction to the fermentation test, to Böttger's bismuth test, or to phenylhydrazin, and no deviation of the rays of polarized light. The urine does not contain bile-pigment. It is of normal specific gravity, and becomes very dark on the addition of an alkali or of a temporarily bluish-green color with perchloride of iron. Ammonia nitrite of silver is instantaneously reduced when added to the urine with a deposit of metallic silver.

Alkaptonuria is usually congenital. Several members of the same family will have it. No symptoms attend the condition.

Note.—*Serum-globulin* is converted into a coagulated proteid when heat is applied or concentrated nitric acid added to a solution. Globulin is soluble in dilute salt solutions. If urine, rich in globulin, is added, drop by drop, to a large volume of distilled water, the globulin is precipitated as the percentage of salt is reduced by dilution. Globulin is also precipitated by dialysis. If a portion of urine containing globulin is saturated with magnesium sulphate or half saturated with ammonium sulphate, globulin is precipitated.

Hills[1] describes the method as follows: "25–50 cubic centimetres of the urine are made neutral or slightly alkaline with ammonium hydroxide, and the precipitated phosphates removed by filtration. An equal volume of a saturated solution of ammonium sulphate is then added, the mixture shaken, and allowed to stand for some time, and finally filtered. The precipitate is washed with a half-saturated solution of ammonium sulphate for the removal of the last traces of albumin and the filtrate and precipitate tested for albumin and globulin respectively, as previously described. The formation of a precipitate upon the addition of either magnesium or ammonium sulphate is not in itself evidence of the presence of globulin."

Microscopical Examination of the Urine. Microscopical examination of the urine is chiefly concerned with the sediments, and these are conveniently divided into the organized and unorganized.

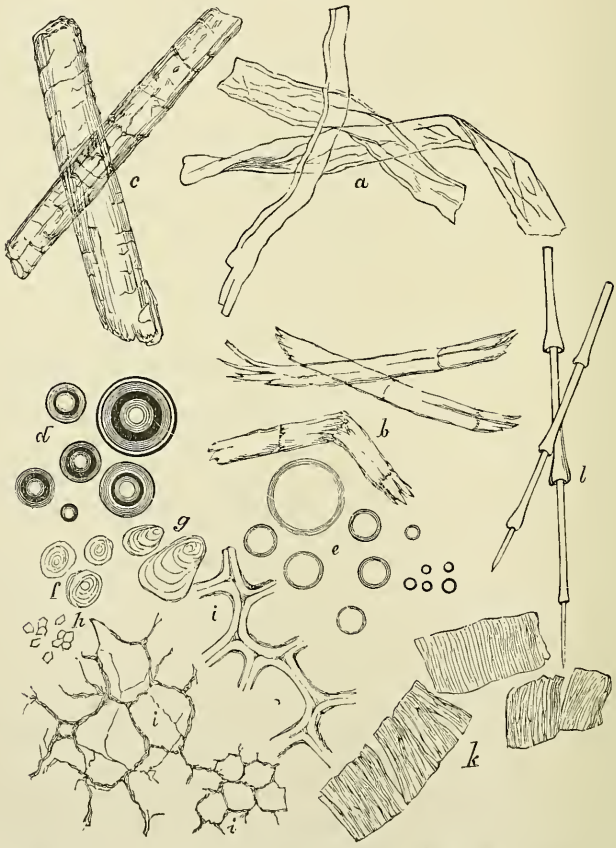
[1] Boston Medical and Surgical Journal, 1899, vol. cxli, No. 6.

The *organized deposits* in the urine are blood, pus, mucus, epithelium, casts, spermatozoa, micro-organisms, cancerous and tuberculous matter, entozoa.

The *unorganized deposits* are uric acid and its compounds, oxalate and carbonate of lime, phosphates, leucin and tyrosin, cystin and cholesterin.

Normal urine forms a slight sediment, consisting of epithelium from different parts of the genito-urinary tract, principally from the bladder in males, and from the vagina and bladder in females. There are also some crystals of the different urinary salts, sometimes mucus and a few white blood-cells, and, if the urine has stood a while, especially if alkaline, more or fewer bacteria. It may accidentally contain extraneous matter, derived from the vessel which contains it or from the air. (Fig. 206.)

FIG. 206.



Extraneous matters found in urine: *a*, cotton-fibres; *b*, flax-fibres; *c*, hairs; *d*, air-bubbles; *e*, oil-globules; *f*, wheat-starch; *g*, potato-starch; *h*, rice-starch granules; *i*, *i*, *i*, vegetable tissue; *k*, muscular tissue; *l*, feathers.

The centrifugal machine has now become an important adjunct to the rapid and accurate microscopical examination of the urine. There

are now numerous varieties to be secured at the instrument-stores, some of which are devised solely for urinary examination, while others have additional apparatus for examination of the blood and sputum. The majority of them are revolved by hand. Electricity can be readily applied to any of them and labor be saved by such a device. The advantages of centrifugal force over the older gravity method employed in microscopical examination are marked. Some few of them can be briefly outlined :

1. Centrifugalization secures complete, rapid, and concentrated sedimentation. It is, therefore, best suited to microscopical diagnosis.

2. Casts or other organic material, if present, can be studied carefully before they are macerated or partially destroyed by bacteria or changed by the deposition of amorphous or crystalline material. This is a most important aid to correct diagnosis.

3. Crystals, if present at the time of urination, can be discovered and differentiated from those that normally crystallize out after some hours.

4. Certain bodies, hyaline casts, for instance, because of their rather light specific gravity, do not settle on the simple standing of the urine, and thus escape detection. These with all other substances are thrown down with the centrifugal machine.

5. Bacteria are discovered with greater ease, especially the tubercle bacillus.

The method commonly used for the examination of the urinary sediment is as follows : The urine for examination (the chemical analysis having previously been made) is decanted until there remains but a small amount in the bottle, which amount contains any sediment already formed, and heavier organic materials. This is then poured into one of the tubes of the centrifugal machine to within one-half inch of the top ; if but one specimen of urine is to be examined, fill both tubes with the same urine. If there is not sufficient urine to do this, fill the remaining tube or tubes with water. It is well to mark the external metal shields of the tubes with a figure, say 1 and 2, or *a* and *b*, so that the urines, if different specimens, may not become confused.

The tubes are then rapidly revolved for three minutes, then removed from the machine and a few drops of the sediment withdrawn with a pipette and placed upon the slide for examination under the microscope. It is necessary to remember that care must be exercised in removing this sediment from the tube. The straight glass pipette without a pointed end seems to give the best results in securing the sediment. The finger is placed upon one end, the pipette inserted to the bottom of the tube and the finger is then elevated just enough to secure a few drops of the sediment that has been cast down by centrifugalization. If the urine contains but the normal mucous cloud, a very small whitish sediment or cloud is found at the bottom of the tube. If oxalate of lime is present, a small filmy whitish sediment is seen. The sediment of amorphous urates is pinkish, fawn, or salmon color. Uric acid appears as a "brick-dust" sediment. Pus produces a heavy yellowish sediment ; phosphates a heavy white sediment, which is sometimes yellowish-white from admixture with leucocytes. Blood in small

quantities produces a rather characteristic brownish deposit. Large amounts of blood appear as reddish coagulæ at the bottom of the tube.

With some of the centrifugal machines the various urinary salts and the amount of albumin present can readily be estimated. Such instruments are provided with graduated tubes, in which the urine and the necessary reagents are put and the resulting precipitate rapidly cast down.

Fig. 207.

Cellular elements from the urine. 1, squamous epithelium; 2, red blood-corpuscles; 3, polynuclear leucocytes; 4, transitional cells; 5, epithelium from the kidneys; 6, epithelium from the bladder; 7, micrococcus aureæ; 8, yeast-fungi.

In this manner Purdy estimates the chlorides, sulphates, and phosphates, and also the amount of albumin most satisfactorily. It is questionable, however, whether the estimation of the salts is accurate.

Organized Sediments. Blood. If the blood comes from the kidney, it is usually intimately mixed with the urine, which remains of a red or reddish-brown color, and contains possibly tube-casts and renal epithelium. The blood-cells appear singly, have frequently lost their hæmoglobin, and hence look like pale-yellow disks. (See Fig. 207.)

Sometimes blood coagulates in the ureters, and long, cylindrical plugs are passed, causing symptoms resembling those of renal colic. When blood comes from the bladder or neck of the bladder (fissure) there are symptoms of frequent micturition, of acute pain and tenesmus, and the blood is not intimately mixed with the urine. When from the neck of the bladder, it often occurs in a few drops at the end of micturition, accompanied with great pain and a sense of faintness. Intermittent hæmaturia, according to Von Jaksch, points directly to calculus or tumor of the bladder.

Blood-cells, when unaltered, are unmistakable, on account of their well-known biconcave appearance. When they have lost their coloring-matter they appear as circular, very pale disks, with extremely faint outline and feeble refractive power. Absence of a nucleus serves

to distinguish them from yeast-spores, and the latter, moreover, are often oval in shape. They are less likely to be confounded with the ovoid and circular shapes of oxalate of lime crystals, because the latter are not common, and can be seen usually in their more common forms as octahedra and dumb-bells in the same urine.

Pus. The sources of pus in the urine have been referred to already. The pus-corpuscle is an opaque, spherical, granular cell, usually somewhat larger than are blood-cells. In dilute urine, or urine to which water has been added, it swells sometimes to twice its original size. At the same time, it becomes less granular, and two, three, or four nuclei may appear. In concentrated urines the pus-cell is small. The addition of acetic acid also causes it to swell, and brings out the nuclei more distinctly and rapidly. Sometimes the pus-cells are discrete, sometimes in dense clumps, and sometimes nothing but a dense mass of pus-cells appear in the field of the microscope.

It cannot be decided from microscopic examination whether a cell is a pus-corpuscle, a mucus-corpuscle, a white blood-cell, or an inflammatory leucocyte. It must be a matter of inference from the general characters of the urine. If red blood-cells are also present, the probability of finding white blood-cells is increased, but pus-cells are not necessarily excluded. So, too, if much mucus be present in the urine, the doubtful cell may be a mucus-corpuscle. Some clue to the source of the pus can be obtained from the urine itself. Urine containing pus from the kidney is usually acid, whereas in cystitis it is alkaline, and almost always contains phosphates, mucus, and abundant bacteria. Again, pus from the kidney, or kidney pelvis, is apt to vary greatly in amounts, or be discharged intermittently; and the urine, when filtered free from pus-cells, is usually still albuminous. Renal epithelium and casts may also be found.

Casts. Casts are the most important of the urinary deposits. They vary greatly in number and size. Sometimes in acute nephritis they form a considerable part of the sediment, but usually they have to be sought for carefully and patiently. A few words as to the method of examining for them may not be superfluous.

Sedimentation by the centrifugal machine is now much in vogue. If the centrifugal machine cannot be employed, proceed as follows:

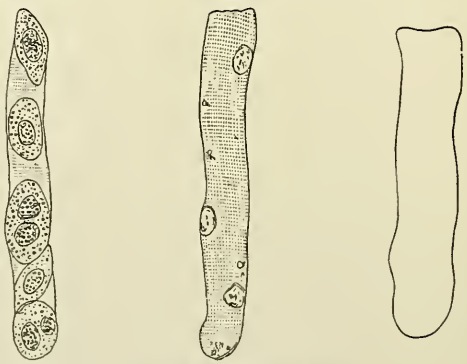
Six or eight ounces of the urine to be examined should be allowed to settle in a bottle as soon after being passed as possible. The bottle should be tightly corked, because urine exposed to the air decomposes very quickly; it should be sent to the person who is to examine it as soon after being passed as possible, in order that an examination may be made before fermentative changes spoil it for trustworthy analysis. After standing twelve, or preferably twenty-four hours, nearly all of the solid matter will have collected at the bottom of the bottle. The supernatant clear fluid can now be poured off, and the lower portion of the urine and the sediment poured into a conical subsiding-glass. If the urine is febrile, there may be by this time a large deposit of amorphous urates, which will obscure the search for casts; they may be dissolved by gentle heating without destroying the casts, and the clear urine again allowed to settle for a few hours. So, too, if phosphates are

abundant, they should be gotten rid of by gentle heating and acidulation with two or three drops of dilute acetic acid.

After the urine in the conical subsiding-glass, which will not now amount to more than an ounce or two, has stood for a few hours, any casts that may be present will have fallen into the bottom. If the urine is very concentrated (1030 or more), epithelium, blood, and casts will be suspended longer; hence, it may be well to dilute the urine before allowing it to settle.

A glass tube, with an internal diameter of about one-eighth of an inch, and with one end drawn out fine, is the most convenient thing for collecting the sediment. The ordinary glass pipette, with a rubber suction-bulb at one end, commonly known as a "medicine-dropper," sometimes answers admirably. If the common glass tube is used, the forefinger of the right hand should be placed over the open upper end, and the fine lower end passed down to the bottom of the glass. The finger is then removed sufficiently to permit a few drops to be sucked in. The same thing is attained if the finger is entirely removed as soon as the point on the tube reaches the bottom of the conical glass; but in that case more than the lowest layers of the sediment or urine are sucked up, and hence all but a few drops should be allowed to flow out when the tube is removed from the urine. In this way the drops preserved for microscopical examination will contain the sediment from the very bottom of the glass. In this sediment, in pale urines free from much urates, phosphates, and pus, the casts will be found, if any are present in the urine. It is most important to examine the bottom layers of the sediment when the latter is scanty, or when phosphates or urates have begun to precipitate after the urine has been standing some time. If the urine is already cloudy with phosphates, urates, or pus, when it is put aside to settle, any casts that may be present will be carried down with the heavier sediment, and will be found intimately mixed with it, or even on top of the other sediment.

FIG. 208.

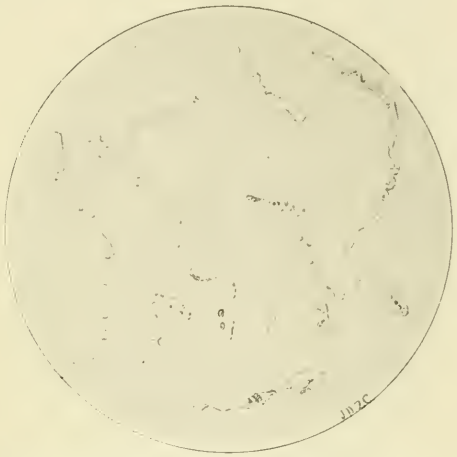


Epithelial and hyaline casts.

The few drops preserved for microscopical examination are now deposited on several slides, without a cover-glass, and examined carefully for casts under a power of 50 to 60 diameters. Casts may be numerous,

so that nearly every field contains one dozen or more, or they may be very few, not more than one or two being found on a slide. The best routine method for microscopical examination is as follows: place a few drops of the urinary sediment upon the slide; spread the drops in

FIG. 209.



Hyaline casts and cylindroids in hypostatic congestion of kidney. Low power.

a thin layer; use no cover-glass; *examine with the low power*—a diameter of 50—with a small amount of light; the whole slide can be carefully searched in three minutes, and casts discovered can be minutely

FIG. 210.

Hyaline casts from a case of acute nephritis. 1, plain hyaline cast; 2, granular deposit on hyaline cast; 3, cellular deposit (blood and epithelium).

studied with the higher power. When but few casts are present, several slides can be rapidly examined with the low power, and an accurate estimation of the number made.

All the pipettes used in examining urine must be kept clean. They should be allowed to stand in water which is frequently changed, and carefully rinsed in running water before being used.

Tube-casts usually indicate acute or chronic nephritis; but they are *sometimes* found in cases of renal calculi; in icterus, usually without albuminuria; in diabetes, and sometimes in secondary congestion of the kidney.

FIG. 211.

Granular casts.

Several varieties of casts are found. 1. *Hyaline casts*, as their name implies, are clear, translucent bodies, which refract light so slightly that they are easily overlooked. They have well-defined margins, the ends being frequently rounded; they are rarely very long, and are straight, or but slightly bent. They are rarely equally translucent throughout; at some part more or less granulation will generally be found. They vary in diameter from that of a white blood-cell to six or eight times as large. They can be stained, and so rendered more distinct, by allowing a drop of gentian-violet solution to flow in under the edge of the cover-glass. (Figs. 209 and 210.) 2. *Granular casts* are hyaline casts which appear granular either from some deposit on their surface or from a granular change of the cast itself. When the granulation does not interfere with the translucency the casts are described as "pale" or "slightly" granular; and when they become very dark, so as to resemble closely a blood-cast, they are called "dark" or "opaque" granular casts. (Plate XLVII., Fig. 1, 1; and Figs. 210, 211.) 3. *Waxy casts* appear to the eye to be more solid in structure than the hyaline casts; they also appear more cylindrical in form, are more or less yellow in color, and are apt to be larger than hyaline casts. (Plate XLVII., Fig. 1, 2.) 4. *Fatty casts* are hyaline or faintly granular casts on which are deposited, in spots, minute oil-drops. They are sometimes called "oil-casts" if the oil-drops are very abundant. (Fig. 212.) 5. *Blood-casts* are either made up of a mass of blood-cells pressed together into a cylindrical shape, or, more frequently, a hyaline cast is

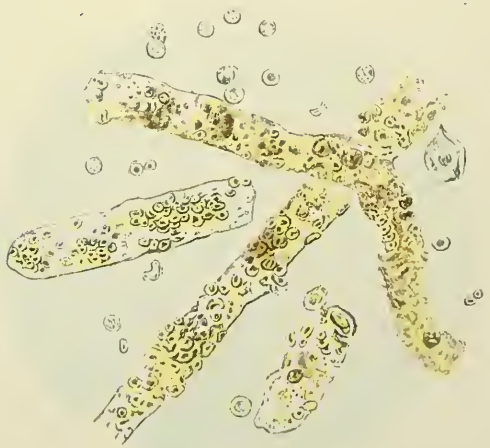
PLATE XLVII.

FIG. 1.

1. Hyaline Casts with Granular Matter and Epithelial Cells deposited upon them. 2. Amyloid (waxy) Cast.

(Oc. 4. ob. D.) Drawn by J. D. Z. Chase.

FIG. 2.

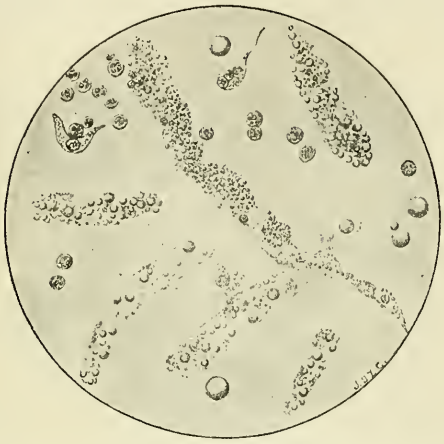


Blood-casts from Case of Acute Nephritis.

(Oc. 4. ob. D.) Drawn by J. D. Z. Chase.

studded with blood-cells. (Plate XLVII., Fig. 2.) 6. *Epithelial casts* sometimes seem to be composed entirely of epithelial cells closely packed together. Such casts are relatively rare, and very beautiful. Ordinarily, just as in the case of blood-casts, an epithelial cast consists of

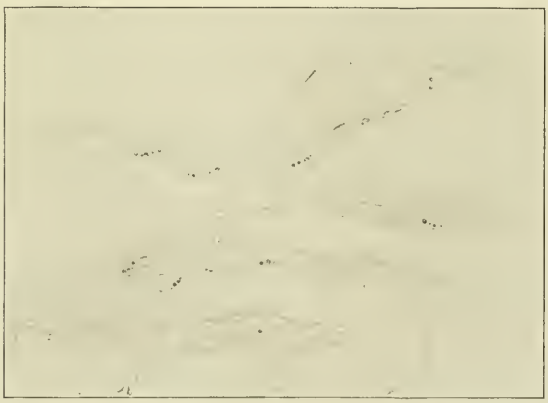
FIG. 212.



Fatty casts from a case of chronic parenchymatous nephritis.

a hyaline cast more or less covered with renal epithelium. (Plate XLVII., Fig. 1, 1; and Fig. 208.) 7. Dr. George Johnson has described casts composed of *pus*-corpuscles. In two cases in which they were found in the urine the patients were found at the autopsy to

FIG. 213.



Cylindroids.

have multiple abscesses of the kidney. 8. *Cylindroids* are very common. In general appearance they resemble hyaline casts; but they are apt to be much longer, bent, twisted or split, and to have, on close examina-

tion, a striated or finely ribbed appearance. Moreover, the diameter of the cast frequently varies greatly at different points; sometimes it appears constricted in several places, and in other cases one end tapers off into a thread. Often cylindroids consist of fine, narrow, ribbon-like threads. (Figs. 209 and 213.)

Spermatozoa. Spermatozoa are easily recognized by their tadpole shape and by the vibratile motion of their long, delicate tails. They

FIG. 214.

Human semen. *a*, spermatozoa; *b*, cylindrical epithelium; *c*, bodies enclosing lecithin granules; *d*, squamous epithelium from the urethra; *d'*, testicle-cells; *e*, amyloid corpuscles; *f*, spermatic crystals; *g*, hyaline globules. (VON JAKSCH.)

are found in the urine of both sexes after sexual intercourse. (Figs. 214 and 215.)

Many continent men have occasionally nocturnal emissions, accompanied by erections and erotic sensations. These cannot be looked upon as abnormal, and they are compatible with robust health. There are other persons, neurotic, anæmic, and generally constipated in habit, who have emissions at night two or three times a week, of which they are unconscious until they wake and find themselves wet. Semen may also be lost during micturition and defecation, especially when much straining is required. Such a condition (spermatorrhœa) is abnormal. It is due to general nervous and muscular relaxation, associated with nervous dyspepsia and anæmia, and aggravated by sedentary life, constipation, and the reading of salacious literature or the cultivation of erotic thoughts. In young men, it sometimes follows habits of masturbation, which have been broken up but have left behind a hyperæsthetic condition of the prostatic portion of the urethra, with or without dilatation of the orifices of the ejaculatory ducts; or a stricture of gonorrhœal origin may be its cause. Students and overworked and overstrained business and professional men are the ones most frequently affected.

However caused, the condition is apt to beget a most distressing state of despondency, in which the patient imagines all possible ills, and is liable to drift into a hysterical, melancholic, even suicidal frame of mind, and so falls a victim to quacks.

Epithelium. Epithelium from the kidney, bladder, and genito-urinary passages occurs in the urine. Epithelial deposits in male urine are very scanty, unless there is some disease of the kidney or bladder,

or a catarrhal condition of the prostatic urethra, such as is left from an old gonorrhœa. On the other hand, considerable epithelium may be normally present in the urine of women, being derived principally from the vagina and bladder.

Vaginal epithelium consists of large, flat pavement-cells, and is readily distinguished.

Fig. 215.

Spermatozoa from urine.

The type of epithelium of the kidney, kidney pelvis, ureter, and bladder is the same, and it is not possible to distinguish with certainty the cells which come from each. If the cells are scanty, Von Jaksch thinks they come from the ureter. He has found them in moderate quantities and superimposed upon one another.

Renal cells closely resemble the oval polygonal cells from the deeper layers of the bladder, but they have a relatively larger nucleus. (See Fig. 207.)

Lipuria. Oil is found in the urine in fatty degeneration of the kidney and its epithelium, and occasionally in the urine of those who are taking cod-liver oil, and in calculous disease of the pancreas. Tyson suggests that it may come from cystic cheesy degeneration of the kidney.

It is also found in chronic nephritis, in phosphorus-poisoning, and in diabetes mellitus, as well as in chyluria. The urine is turbid, but clears when agitated with ether. The fat may be separated by a sedimentator, and can be recognized by its refractive properties.

Staining for Fat. Reeder recommends Soudan Three for staining human secretions and excretions, to determine the presence of fat. Large fat-droplets take a bright red, and small droplets a yellow or orange color. Fat can thus be demonstrated in the blood in lipæmia, lipuria, and chyluria. By this method fat can be demonstrated in the stomach-contents and in the feces of adults with jaundice. A saturated solution of Soudan Three in 96 per cent. alcohol is employed. Equal

parts of this solution and 96 per cent. alcohol are added to the urine. In urinary sediments the fat-droplets in casts stain a scarlet red.

Chyluria. This is a more or less milky condition of the urine, due to the presence of fat, which probably gains entrance to some part of the urinary tract by rupture of the lymphatic vessels. A case has been reported by Saundby, in which a young unmarried girl, being pregnant, compressed her abdomen so much, in order to conceal her condition, that œdema of the legs, thigh, vulva, and lower parts of the abdomen resulted. After her confinement the urine became milky, and remained so for many days. It contained fatty matters and cholesterin, but no albumin or sugar.

Fat and albumin appear at the same time in some diseases. They recur at long intervals. Red and white blood-corpuscles are also found in small amounts. The urine coagulates on standing, or gelatinizes.

Parasitic chyluria is due to the filaria sanguinis hominis, whose embryos obstruct the lymphatics. The latter may be found in the urine.

Entozoa. The most common is the *echinococcus* or *hydatid*. When this infects the kidney or urinary vessels, hooklets and even cysts have been passed in the urine. The disease is, of course, extremely rare in this country.

The *filaria sanguinis hominis*, which causes parasitic chyluria, is occasionally found in the urine. (See Filaria.)

The *Bilharzia hæmatobia* sometimes lodges in the urinary tract and causes hæmaturia. It is peculiar to Egypt.

Distoma Hæmatobium. Common in Egypt and Abyssinia. Eggs collect in great masses in the urinary passages, and lead to inflammation, ulcers, stenosis, etc. Eggs found in the urine alone make the diagnosis possible.

Strongylus Gigas. Very rare. Symptoms of pyelitis. (The parasite is of the size of an earth-worm.)

Intestinal worms may creep into the bladder through fistulous or other openings, and be discharged through the urethra.

Micro-organisms. Normal urine contains no micro-organisms at the time it is voided. As the result of exposure to air, however, they may develop in great abundance. The non-pathogenic organisms found are classed as mould-fungi (hyphomycetes), yeast-fungi (blastomycetes), and fission-fungi (schizomycetes).

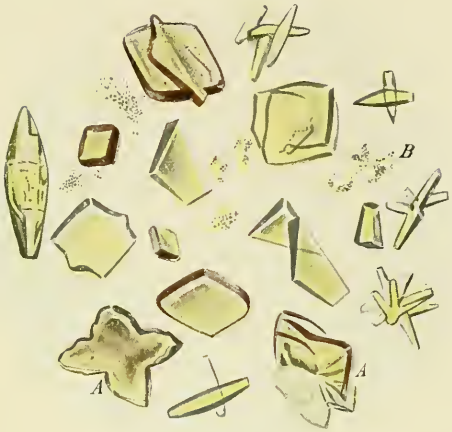
Mould-fungi, according to Von Jaksch, are rarely found in foul normal urine. Yeast-fungi are also rare in normal urine. Fission-fungi are found in urine undergoing ammoniacal decomposition.

Sarcinæ, usually smaller than those of the stomach, are occasionally met with—especially, according to Roberts, where there is some disorder of the urinary organs, renal pains, painful micturition, cystitis, etc.

Under the name *bacteriuria*, Roberts and others have described cases in which the urine contained bacteria at the time of being voided. He makes four groups: (1) Cases in which the presence of bacteria is associated with incipient putrefactive changes in the urine; (2) cases associated with ammoniacal fermentation of the urine; (3) cases in which common forms of bacteria are present without decomposition of the

PLATE XLVIII.

FIG. 1.

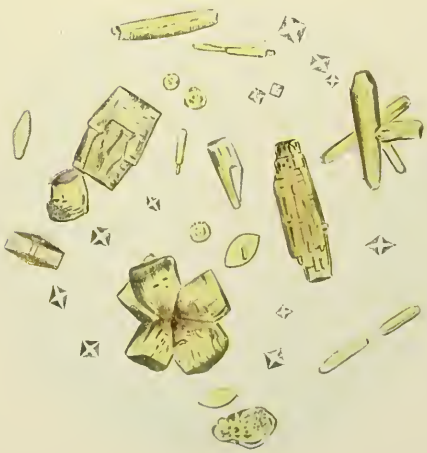


Uric Acid.

A. Common forms. *B*. Amorphous urates.

(Ob. D. and A., Oc. 4.) Drawn by J. D. Z. Chase.

FIG. 2.



Combination of Uric Acid and Calcium Oxalate.

(Oc. 4, Ob. D.) Drawn by J. D. Z. Chase.

urine; and (4) cases in which micrococcus-chains are voided in the urine.

The pathogenic organisms which are more or less closely associated with infectious diseases, septic processes, and tuberculosis are found at times in the urine, and can be demonstrated by the proper staining-methods.

FIG. 216.

Vibriones in urine. (ROBERTS.)

Dock has given an admirable account of the occurrence of the trichomonas in the genito-urinary passages. This parasite belongs to the flagellate infusoria. The prominent symptoms caused in Dock's case were painful, difficult, and frequent urination, followed by hæmaturia. The urine contained pus, epithelium of all kinds, and a number of bodies slightly larger than pus-corpuscles of a peculiar amyloid appearance—the trichomonades.

Morbid Growths. The urine very rarely contains the elements of morbid growths. Von Jaksch says he never has found them in any way reliable in the case of tumors of the kidney. The detection of cancer-cells or pigmented cells, such as occur in melanotic cancers, may confirm the diagnosis if the clinical symptoms point to cancer. Tumor-elements are most likely to be found in ulcerating tumor of the bladder.

Unorganized Sediments. Uric Acid. Uric acid is present in small quantities (eight to ten grains a day) in normal urine. It is *increased* in febrile and wasting diseases, such as phthisis; in diseases of the liver and spleen (leukæmia), and in malarial fever, diabetes, scurvy, rhachitis, and following an attack of gout. Excessive use of milk is said to increase it. Its excretion is also increased by certain drugs—colchicum, corrosive sublimate, salicylic acid, and euonymin.

It is *diminished* in anæmia, chlorosis, and during a paroxysm of gout; in chronic nephritis; by certain drugs—large doses of quinine (Ranke), caffein, sodium chloride and sodium carbonate, lithia, and iodide of potash. (Plate XLVIII., Figs. 1 and 2.)

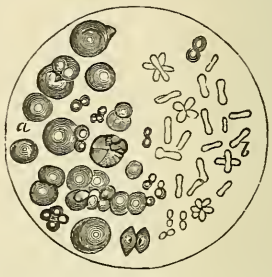
According to Roberts, a deposit of uric acid occurring some twelve to twenty-four hours after the urine has been passed has no pathological significance. If the deposit occurs within three or four hours after the urine has been passed, it is certainly not natural. It is frequently observed in convalescence from febrile complaints, especially articular rheumatism; also in the middle periods of chronic Bright's disease, in chorea, in certain types of diabetes, and in enlargement of the spleen. If, however, the uric acid is precipitated before the urine cools, or immediately afterward, it is probable that the same precipitation may occur within some part of the urinary passages, and so form a calculus.

Urates. Amorphous urates appear under the microscope as opaque granular particles, which dissolve upon heating, and respond to the murexid test. The deposit is more or less dense, and is sometimes arranged so as to resemble granular casts.

FIG. 217.

Sodium urate.

a a. From a gouty concretion. *b b.* Artificially prepared by adding liq. sodæ to the amorphous urate deposit. (ROBERTS.)



Ammonium urate spontaneously deposited.

a. Spheres and globular masses. *b.* Dumb-bells, crosses, rosettes. (ROBERTS.)

SODIUM URATE appears as spherules or globules, from which project short spines, either straight or curved. It occurs most frequently in concentrated acid urines, such as are passed by children with acute febrile diseases. (Fig. 217.)

FIG. 218.

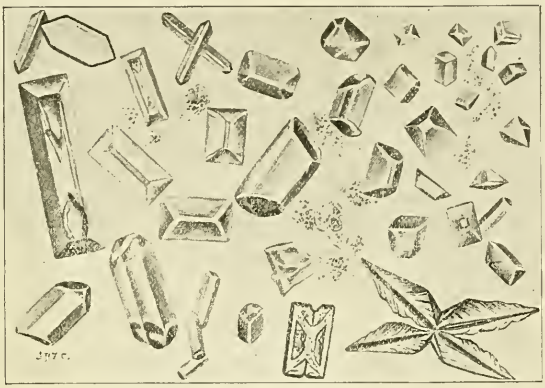
Ammonium urate.

AMMONIUM URATE resembles sodium urate. It is frequently associated with phosphatic deposits, and is precipitated from alkaline urines. Sometimes it appears in the shape of dumb-bells. (Figs. 217 and 218.)

Phosphates. Phosphates appear in the urine as ammonio-magnesium phosphate and as the crystalline and amorphous phosphate of lime.

They are precipitated in alkaline or faintly acid urines, which produce a cloud upon being heated; the cloud is distinguished from albumin, as already pointed out, by the fact that it disappears when the urine is

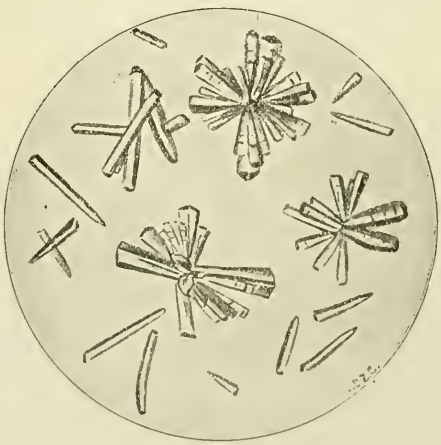
Fig. 219.



Triple phosphates.

acidulated with acetic or nitric acid. Ammonio-magnesium phosphate is easily recognized by its rhombic prisms—"coffin-lid" shape. Other shapes are produced by modification of the primary one, chiefly by bevelling of the edges and hollowing out of the sides. These crystals

Fig. 220.



Calcium phosphate crystals.

are usually large, and are frequently found, together with amorphous phosphates, bladder epithelium, and pus, in cases of cystitis.

Amorphous phosphate of lime consists of fine granular particles much resembling amorphous urates, but distinguished from them by not dis-

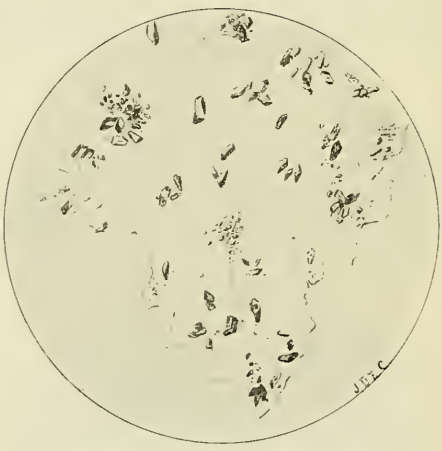
appearing upon the application of heat, but instantly dissolving when the urine is acidulated.

Crystalline phosphate of lime is a not infrequent deposit. It is found as narrow-wedged crystals, occasionally grouped together in the form of stars, sheaves, or bundles, with their apices at a common centre.

According to Roberts, this deposit, in quantity, is an accompaniment of some grave disorder. He has found the stellar phosphates in cancer of the pylorus, once in phthisis, and more than once in patients exhausted by obstinate rheumatism. It may, however, occur in health, when the urine is rich in lime and its acidity greatly reduced.

In one or two cases of renal colic the writer has observed numerous shining particles, which, upon microscopical examination, have been shown to be an opalescent film, covered with small, sharp phosphatic (probably calcium) crystals. (Fig. 221.)

Fig. 221.



Opalescent film in a case of renal colic.

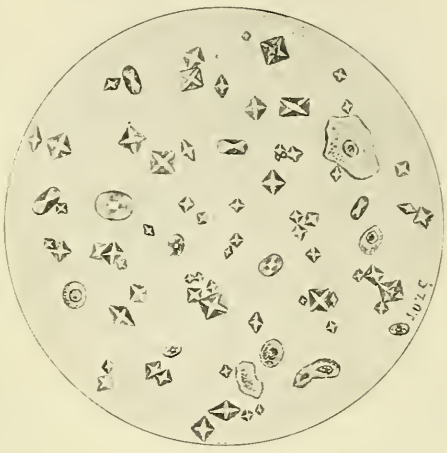
Oxalate of Lime. Oxalate of lime occurs in the form of small octahedral crystals, or, more rarely, as dumb-bells, and in the form of ovals or disks. It is precipitated almost always from acid urines. (Plate VI., Fig. 2; and Fig. 222.)

Oxaluria. According to Beneke, oxaluria has its proximate cause in an impeded metamorphosis, an insufficient activity of that stage which changes oxalic acid into carbonic acid.

When oxalates are constantly found in the urine a condition of profound hypochondriasis is found to exist, but it has no necessary relation to the oxaluria. An increase of oxalates in the urine is found in diabetes, especially when there is diminution in the amount of sugar. It is in excess in certain forms of indigestion. Its constant passage may be attended by pains in the back and loins. Flatulent and nervous dyspepsia usually accompany the increase, and neurasthenia also may be present.

Cystin. Cystin occurs in the form of hexagonal prisms, either as irregular masses or superimposed one upon another, so as to form truncated pyramids. It is a very rare sediment, but appears to be most common in children and young male adults. Several members of the same family have been known to pass it. Its chief clinical significance arises from the fact that rarely it is the basis of calculi.

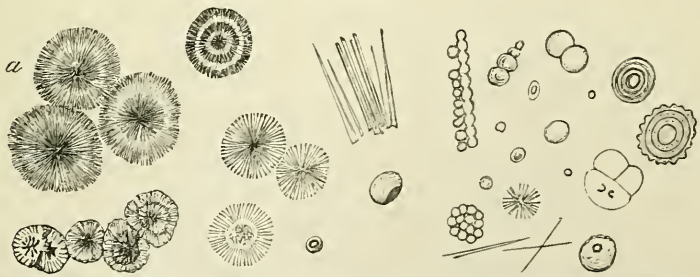
FIG. 222.



Calcium oxalate.

Leucin and Tyrosin. Leucin and tyrosin are generally described together, though the former is not spontaneously deposited from urine. It appears in the form of spheres, which refract light strongly and have a radiating arrangement. (Fig. 223.)

FIG. 223.



Crystals of leucin (different forms). (Crystals of creatinin chloride of zinc resemble the leucin crystals depicted at *a*.) The crystals figured toward the right consist of comparatively impure leucin. (From CHARLES: *Chemistry*.)

Tyrosin has been found as a sediment, of a light greenish-yellow color, in typhoid fever and acute yellow atrophy of the liver. It appears in the form of tolerably long, needle-like crystals, or as bundles and sheaves. Frerichs attaches great importance to leucin and tyrosin in the diagnosis of acute yellow atrophy of the liver. (Fig. 224.)

Cholesterin. This occurs at times in fatty degeneration of the kidneys, jaundice, chyluria, diabetes, and, according to Pohl, in the urine of epileptics treated with bromide of potash. (Fig. 225.)

Fig. 224.

Tyrosin crystals.

Melanuria. Melanin is held in solution or suspended in small granules. The urine is dark in color, and blackens intensely when sulphuric acid or tincture of chloride of iron is added to it. A concentrated solution of perchloride of iron serves to detect the presence of the substance.

Fig. 225.

Crystals of cholesterin.

A few drops added to the urine turn it gray. If a few drops more are added, the phosphates are precipitated along with the coloring-matter. Both are dissolved by an excess of the iron solution. Melanin is usually found in cases of melanotic carcinoma.

Catheterization and Exploration of the Ureters.

Examination of the bladder, the ureters, and the pelvis of the kidney has been wonderfully advanced by the genius of Howard Kelly. The following instruments are required for the examination of the bladder: Female catheter; urethral calibrator; a series of urethral dilators; a series of specula with obturators; common head-mirror and a lamp, Argand burner or electric light; long, delicate mouse-toothed forceps; suction-apparatus for completely emptying the bladder; ureteral searcher; ureteral catheter with a handle; small bran-bags for elevating the pelvis.

The procedure is as follows: Empty the bladder; measure the meatus urinarius externus; dilate the urethra to twelve or fifteen millimetres; insert speculum of diameter of last dilator and remove obturator; elevate the hips of the patient about a foot above the level of the table; inspect with light; remove residual urine by suction or with cotton and mouse-toothed forceps.

For anæsthesia, a pledget of cotton saturated with a 5 per cent. solution of cocaine may be introduced seven minutes before dilatation. On removal of the obturator the bladder becomes distended with air. The bladder is viewed by turning the speculum, and each ureteral orifice is brought into view by turning the speculum thirty degrees to one side or the other. Kelly says: "The orifice appears as a dimple or a little pit, or, in inflammatory cases, as a round hole in a cushioned eminence; at other times as a $\wedge$ with the point directed outward; again, it may be scarcely visible even to a trained eye, appearing as a fine crack in the mucosa, and occasionally is so obscure as to be recognized only by the jet of urine as it escapes, or by a slight difference in the color of the mucous membrane at that point. In rare cases it has the form of a truncated cone with gently sloping sides; this appearance is most apt to be developed in the knee-breast position. The bladder mucosa is usually of a slightly deeper rose color around the ureter, and in the presence of an inflammatory process it even appears deeply injected."

Catheterization of the Ureters. The catheters are sterilized; they are stiffened with a wire stylet. The orifice is exposed, and then the outer end of the catheter being held over the shoulder by an assistant, the conical end is introduced and pushed up the ureter, while at the same time the stylet is being removed. The speculum is removed and again introduced beside the first catheter. The remaining ureter is then catheterized; both are properly designated and allowed to drain into test-tubes plugged with sterilized cotton and fixed in a block of wood. By catheterization, aspiration, and exploration of the ureters with a bougie, the source of pyæmia anywhere from the urethral orifice to the renal pelvis can be found; renal calculi diagnosticated; strictures of the ureter located; hydronephrosis distinguished from soft malignant growths; and the functional value of each kidney determined.

Kelly suggests the following guide to the ureteral orifice: "A point is marked on the cystoscope $5\frac{1}{2}$ cm. from the vesical end, and from this point two diverging lines are drawn toward the handle with an angle of sixty degrees between them. The speculum is introduced up to the

point of the V, and turned to the right or left until one side of the V is in line with the axis of the body; then by elevating the endoscope until it touches the floor of the bladder the ureteral orifice will usually be found within the area covered by the orifice of the speculum."

By means of a searcher, or sound, the suspected orifice is further examined.

Objective Symptoms due to Impairment of the Function of the Kidney.

Uræmia. Under symptoms due to impairment of the functions of the kidney belong the various manifestations of uræmia. Diseased kidneys do not eliminate the products of tissue-waste, which are poisonous materials. The toxic matter is retained within the blood, and produces toxæmia, which may be acute or chronic. In *acute uræmia* the manifestations develop suddenly and continue but a short period of time, with alarmingly active symptoms until death or recovery. In *chronic uræmia* the onset is gradual. The manifestations may be limited to one or two conditions, as headache or morning nausea, or they may include the more pronounced symptoms of uræmia.

Nervous Symptoms. 1. *Headache.* The pain is situated in the occipital region, and may extend down the neck. It is severe and of a bursting character. It may be associated with giddiness. In both acute and chronic nephritis it is often the first manifestation. It may be associated with eye-symptoms. It may be present on waking, and continue only through the morning hours. In acute uræmia it persists throughout the attack. Numbness and tingling of the fingers are often complained of at the same time.

2. *Delirium.* The delirium may be mild. This is usually the case in the typhoid state or if a subnormal temperature prevails. It is sometimes attended by delusions. There is often subsultus, and picking at the bedclothing. The delirium may amount to true mania, and the patient may exhibit other maniacal symptoms. On the other hand, the patient may be noisy, restless, and sleepless. Melancholia and delusional insanity may develop after the violent nervous symptoms of uræmia pass off.

3. *Convulsions.* A convulsion may be the first indication of disease of the kidneys, or it may succeed a few days of persistent headache, or follow an attack of uræmic vomiting. The convulsion resembles epilepsy, and hence is known as an epileptiform convulsion. If the spasms recur in rapid succession, the interval is occupied by delirium or coma. If they are infrequent, the patient's mind may be clear in the intervals. Sometimes a focal or Jacksonian epilepsy occurs instead of the true epileptiform convulsion. The *temperature* is usually elevated. In worn-out subjects, or those who have had exhaustive diarrhœa, or are debilitated from other causes, the temperature may be subnormal. A temporary blindness often follows the convulsion (*uræmic amaurosis*). Uræmic deafness may occur.

4. *Coma.* After the convulsion the mind may be restored, or the patient may lapse into stupor, followed by complete coma. Coma may

develop without convulsions, or immediately succeed a general convulsion. Headache or eye-symptoms may precede the coma. In some instances the patient lapses into a typhoid state, in which the tongue is heavily furred and the breath very offensive. Unless the coma is profound there is usually some twitching of the muscles of the hands and face.

5. *Local palsies.* Dercum was among the first to call attention to the occurrence of uræmic monoplegia or hemiplegia. The cases resemble central cerebral disease. The nature of the palsy is inferred from the results of the examination of the urine and the condition of the heart and arteries. Palsy develops suddenly, or may occur after a convulsion.

6. *Cramps* in the muscles of the calves, particularly at night, are of common occurrence, and should always lead to an examination of the urine.

7. *Pruritus,* local or general, is another nervous symptom which may be of uræmic origin.

8. *Pain* in the upper abdomen, particularly in the median line, is a frequent precursor of more severe uræmic symptoms. It is of uræmic origin itself. It may be seated in either of the upper quadrants, and thence extend to the lower portion of the abdomen.

Uræmic Dyspnœa. Modifications of the breathing often accompany symptoms of uræmia. The dyspnœa may be constant. It may occur in paroxysms, or both types may alternate. A common type in the uræmia of chronic nephritis is the Cheyne-Stokes breathing. Paroxysmal dyspnœa usually occurs at night, and resembles asthma in every respect. Cheyne-Stokes breathing continues, even through the period of coma, although not necessarily associated with it. (See page 456.)

In addition to uræmic dyspnœa, the occurrence of inflammatory pulmonary complications may be the first indication that the condition of the urine should be inquired into. Bronchitis, pneumonia, and pleurisy are common complications.

Gastro-intestinal Symptoms of Uræmia. Several forms are seen. 1. *Loss of appetite* is common. It is attended with absolute distaste for food after a small portion is taken. 2. *Nausea,* which may be continuous, or more frequently limited to the early morning. 3. *Vomiting* may be paroxysmal, occurring chiefly in the early morning, or it may be sudden in onset, uncontrollable, and continue until nervous symptoms of uræmia develop. Urea is found in the vomit. The matter ejected is profuse, of a low specific gravity, and at first acid in reaction. If chronic, it may become alkaline. The odor is often sufficient to cause its recognition. 4. *Constipation* is generally the rule in the course of chronic Bright's disease. 5. *Diarrhœa.* One of the manifestations of uræmia is the occurrence of sudden, profuse serous purging. This may be so extreme as to cause collapse, or may usher in coma and convulsions. 6. *Hiccough,* although a muscular affection, is usually associated with gastric disturbances.

Latent uræmia was first recognized by Sir William Roberts. It is seen in its most characteristic form in calculus suppression. The

patient for several days will have subnormal temperature, myosis, occasional vomiting, and toward the end twitching of the voluntary muscles and slight drowsiness. After the end of five or ten days coma, convulsions, or dyspnœa ensue.

Cardio-vascular Symptoms of Nephritis. The symptoms are the effects of the retention of morbid products. First, the *heart and bloodvessels*. The poison which is not excreted circulates throughout the system. One of its effects is irritation of the vasomotor nerves of the bloodvessels. Excitation of these nerves causes peripheral contraction of the smaller vessels. At once the flow of blood is obstructed, so that, on account of the contraction, hypertrophy of the heart rapidly ensues. The first prominent symptom, therefore, is due to changes in the heart-muscle.

Hypertrophy of the Heart. The most pronounced change is *hypertrophy*. The persistent spasm of the peripheral vessels causes increased arterial tension. The blood-pressure is raised and causes increased accentuation of the aortic second sound. *High tension* in the artery is recognized by the peculiar character of the pulse and by means of the sphygmograph.

Dilatation of the Heart. Unfortunately, hypertrophy of the heart cannot always be kept up. If it fails, we then have a second condition of the heart which is frequently found in renal inflammations; it is dilatation. The state of the coronary arteries predisposes to this condition of the heart-muscle. The previously mentioned arterial tension favors the development of chronic endarteritis with general atheroma. The coronary arteries take part in this process. The endarteritis hinders cardiac nutrition, dilatation of the heart-muscle follows, and later comes the development of two other conditions, atrophy and myocarditis.

Here may be mentioned other relations of the heart and kidneys: *a*. We have renal disease following forms of cardiac disease. In dilatation of the heart passive congestion of the particular organ takes place. The kidney very quickly becomes the seat of such congestion. In the course of simple dilatation, or of valvular heart-disease, the secondary dilatation, passive congestion, and chronic inflammation develop slowly. Embolism may also occur. *b*. Renal disease and cardiac disease may develop at the same time from a common cause, as alcoholism, gout, or endarteritis.

In addition to high arterial tension and accentuation of the aortic second sound, the objective symptoms of atheroma of the aorta and arteries are present with the chronic inflammations of the kidney. These vascular changes need not be again rehearsed. (See Endarteritis.)

It is important, however, to bear in mind that they frequently occur together, and also that in all instances of arterial disease the condition of the urine must be inquired into. It need not be said that symptoms due to rupture of the bloodvessels, particularly in the brain, or to aneurism, necessarily may be present in the course of renal inflammation.

Gastro-intestinal Symptoms. Fermentative dyspepsia, gastralgia, chronic gastritis, enteritis, and ulcerative colitis are of common occurrence.

Hemorrhages. The arteries are very liable to rupture, causing epistaxis, retinal hemorrhage, hemorrhages from the bowels and lungs, and hemorrhages underneath the skin. Frequent hemorrhages in large amounts from any portion of the body should call attention to the condition of the urine.

Ophthalmoscopic Changes. The eye-ground should always be examined; indeed, the patient himself by his complaints often directs attention only to the eye, the examination of which discloses the presence of an albuminuric retinitis. The changes may occur in the acute or chronic forms of nephritis, although they are more common in the latter. 1. A diffuse, slight opacity and swelling of the retina, due to œdema. 2. White spots or patches of various sizes, for the most part the result of degenerative processes. 3. Hemorrhages. 4. Inflammation of the intraocular end of the optic nerve. 5. Atrophy of the retina and nerve may sometimes result from and succeed the inflammatory changes. These changes may affect one eye only (Gowers). It must not be forgotten that temporary blindness may occur independently of retinitis.

Dropsy. Dropsy may occur in all forms of nephritis. It is most common in acute varieties, but it is also present in chronic diffuse nephritis with exudation. Renal dropsy usually begins in the face. It may develop suddenly in acute forms. In the marked forms, œdema of the eyelids may continue for a long time. All varieties may be found, from local œdema to extreme anasarca. The serous cavities are also filled. The œdema is usually associated with a diminished amount of urine. Its improvement is attended by increased diuresis. Dropsy, in chronic disease, is usually due to dilatation of the heart. (See page 100).

The Cutaneous Symptoms, and Appearance of the Face. In inflammatory affections of the kidney, the appearance of the skin and expression of the face are often characteristic, and point at once to an examination of the urine. The face is pallid, and of an ivory whiteness. In the chronic form the pallor gives way to an ashen-gray or sallow complexion. In chronic nephritis the skin becomes dry and harsh, and, rarely, is covered with a powdery substance, giving it the appearance of frost on the skin. The powdery substance is due to urea.

Petechiæ. In the later stages of chronic inflammatory affections *hemorrhages* under the skin and in the mucous membrane are seen.

Anæmia. Anæmia is a frequent symptom in all forms of nephritis; it is usually marked. It is associated with the peculiar pallor just described, and attended by all the other usual symptoms.

General Symptoms. The cause of renal disease, as far as symptoms pointing to the kidneys are concerned, is often latent. Instead of renal symptoms, a generally depraved state of the system may be seen, with *emaciation* and *weakness*. Lassitude without cause demands an examination of the urine.

Diabetic Coma. Acetonæmia is a toxæmia which develops in the terminal stages of diabetes. It is due to an accumulation of acetone in the blood. It is also called *diabetic coma*. It develops acutely. A sudden onset is attended by sharp pain in the stomach with nausea, and frequently vomiting. At the same time there is severe dyspnœa. The breathing is irregular and of a panting character, with inspiratory and

expiratory dyspnœa. There may or may not be cyanosis. The patient is obliged to sit up in bed on account of the air-hunger. Restlessness begins at once. Delirium develops within the first hour. In a few hours coma sets in. The temperature is subnormal; the pulse is irregular, and soon becomes weak and thready. The odor of acetone is detected on the breath.

Congestion of the Kidney.

Congestions of the kidney are acute and chronic, and depend upon changes in the circulation, whereby blood accumulates in the kidney.

Acute congestion of the kidney is caused by the action of irritant poisons; it follows surgical operations, particularly if prolonged, and may follow extirpation of one kidney. Diseased kidneys are apt to become the seat of active congestion.

Symptoms. The urine is diminished in amount, or may be suppressed entirely. Only a small amount is passed at frequent intervals, or it can be secured by the catheter alone. Albumin is present in considerable amount, and blood and epithelial casts are numerous. Death may take place, with symptoms of uræmia.

Chronic Congestion of the Kidney. It is also called passive congestion. This form of congestion is usually a part of general venous stasis, due to disease of the heart or lungs, as valvular disease of the heart, with secondary dilatation or pulmonary emphysema. It is quite common.

Symptoms. The *urine* is diminished in amount; dark in color; of high specific gravity, ranging from 1020 to 1030. Uric acid and urates are increased. Urea to the amount of from 10 to 12 grains to the ounce is passed in twenty-four hours. At first there is no further change, but, subsequently, albumin appears in small amounts in an intermittent manner. Later, it is constant and increased in amount. Hyaline casts are found in the urine, and a few red blood-cells.

The condition is recognized by its association with congestion in other organs; by the diminution in the amount of urine, its high specific gravity, and excess of uric acid and urates. This form of congestion is serious, because it leads to chronic nephritis. The latter is recognized by the usual changes in the urine.

Inflammations of the Kidney.

The inflammations of the kidney are divided in accordance with the activity of the process and the degree of exudation or cell-proliferation that attends the inflammation. We, therefore, have the following varieties:

Acute exudative nephritis (acute Bright's disease).

Acute productive or diffuse nephritis (acute Bright's disease).

Chronic productive or diffuse nephritis with exudation (chronic tubular nephritis).

Chronic productive or diffuse nephritis without exudation (chronic interstitial nephritis).

Suppurative nephritis.

Tubercular nephritis.

Acute Exudative Nephritis or Glomerulo-nephritis. In this form of nephritis there are congestion, exudation of plasma, transudation of red and white blood-cells, and changes in the epithelium.

Causes. It may occur without definite cause, save exposure to cold, and at times even without such history. It occurs in most of the infectious diseases. It is of common occurrence after scarlet fever, and in the course of pregnancy and in septicæmia. It occurs in diphtheria, erysipelas, and pneumonia frequently. It is the expression of a peculiar type of typhoid fever. It may complicate dysentery and acute tuberculosis. It forms one of the modes of termination of diabetes.

Symptoms. The course of the disease may be mild, presenting only changes in the urine, or there may be, in addition to decided changes in the character of the urine, local and general symptoms.

In mild cases the *urine* is diminished in amount; micturition is frequent; the color of the urine is increased, and the specific gravity is usually high. A small amount of albumin is found, and a few epithelial and blood-casts, and sometimes blood. At the termination of the disease the casts are hyaline.

In severe cases the disease is ushered in by *chill*, attended and followed by *pain* in the loins, with *fever*, *headache*, and much restlessness. The *urine* may be passed more frequently than usual, but in small amounts; or micturition may diminish in frequency or cease entirely. Examination of the urine reveals the characteristic changes. The quantity of the urine is lessened; the specific gravity is normal or increased. There is a large amount of albumin, and an abundance of hyaline, granular, epithelial, and blood-casts. Free white and red blood-cells, and epithelium from the pelvis and tubules are found.

The fever continues; the pain in the loins is sometimes very severe, and may be taken for lumbago, unless an examination of the urine is made. Within the first forty-eight hours the characteristic symptoms that follow the chill and that attend the urinary changes are *headache*, *sleeplessness*, more or less *stupor*, muscular *twitchings*, or general *convulsions*. *Eye-symptoms* may be present. Instead of cerebral symptoms, *dyspnœa* may be marked. With both, *nausea* and *vomiting* are of common occurrence. The *heart's* action is increased in force and frequency. The left ventricle rapidly becomes hypertrophied. The aortic second sound is accentuated. The *pulse* is hard and exhibits the characteristic features of high tension. From the onset of the first symptom, or within the first week, two other striking phenomena arise. They are, first, the occurrence of *dropsy*; second, the occurrence of *anæmia*.

Dropsy or œdema is one of the most constant symptoms. It appears first in the face, especially the eyelids. It may be limited to this region. It is worse in the morning. From the face, in bad cases, it extends to the lower extremities and to the scrotum, and thence all over the body. Anasarca is the name applied to the general dropsy; the connective tissue is infiltrated with serum. It is recognized by the pallor of the swollen surface; the pitting on pressure; the absence of heat and of pain. (See page 148.)

Effusion may take place into the serous cavities, either the pleura,

pericardium, or peritoneum, causing the symptoms due to effusion. In some instances there is œdema of the mucous membranes, as the conjunctiva, the soft palate, and the glottis.

Dyspnœa may be a pronounced symptom, due either to uræmia (uræmic asthma) or œdema of the glottis, effusions into the pleura, or to bronchitis. If dilatation of the heart occurs, dyspnœa may arise, due to that or to the secondary œdema of the lungs.

With or without the occurrence of nausea or vomiting there is always loss of *appetite*, and usually *constipation*.

The *fever* is usually moderate and irregular in type. *Prostration* is common; often there is emaciation. Symptoms of *uræmia* may occur at any time.

Exudative nephritis with excessive *pus* formation is of sudden onset, characterized by high fever and extreme prostration. There is rapid *emaciation* and the early development of the *typhoid state*. This is preceded by delirium, headache, and stupor, with great restlessness. There is but little, if any, dropsy. Large numbers of red and white blood-cells and the usual casts are found in the urine. There is not so much diminution in the urine as is usually seen. The disease may arise without apparent cause, or complicate scarlet fever or diphtheria.

This form is very fatal, and resembles *acute meningitis*, from which it is diagnosticated by the change in the urine.

Acute Productive or Diffuse Nephritis. In this form there is an overgrowth of connective tissue, and excessive growth of the capsule-cells in the glomeruli, in addition to the lesions of the first form. The whole kidney is not necessarily affected, but only portions at a time. *Symptoms:* The onset is sudden. The subjective symptoms previously described are present in a marked degree. *Nervous* symptoms (uræmia) are most pronounced. *Dropsy* develops rapidly and to an extreme degree. There is rapid development of *anæmia* and *loss of flesh*. The remaining symptoms tally with those of the first affection.

The *urine* is scanty, bloody, and of high specific gravity. The microscopical appearances are like those of acute exudative nephritis. If convalescence is established, the urine becomes more abundant, with a corresponding fall in the specific gravity. The albumin and casts may appear for a time, but eventually disappear.

Diagnosis. The diagnosis of acute nephritis of either form is based upon the examination of the urine. Etiological associations are of value. The more pronounced cases follow scarlet fever and pregnancy.

In the latter condition it usually advances slowly. There may be no symptoms until the occurrence of uræmia. In some instances the disease resembles typhoid fever. In cases in which the onset is sudden, with early uræmic symptoms, it must not be mistaken for epilepsy, delirium, or mania.

Chronic Productive or Diffuse Nephritis with Exudation. In chronic inflammations the formation of new tissue always takes place. They are divided, therefore, into exudative and non-exudative inflammations. The exudation is from the vessels. *Causes:* This form usually follows acute productive nephritis and chronic congestions or degenerations of the kidney. It develops in the course of syphilis,

tuberculosis, endocarditis, disease of the bones, and prolonged suppuration. Frequent exposure to cold and wet, a residence in damp dwellings, and the alcoholic habit are causal conditions. It usually occurs in middle life, more frequently in men. When it occurs as a primary disease it is usually found in young adults. *Symptoms:* The disease develops slowly. General symptoms may first be observed. *Dropsy* may develop at first and continue throughout the disease, or recur at long intervals. The appearance of the patient is striking. The skin is of a peculiar *pallor* and is pasty in appearance. The sclerotics are very white. The *anæmia* which gives rise to the pallor is profound, and often closely resembles that of pernicious anæmia. The anæmia is due to diminution in the hæmoglobin and reduction in the number of red blood-cells.

Headache and *sleeplessness* are common symptoms. Pronounced acute uræmia does not often occur. Chronic *uræmia* may prove fatal by the patient lapsing into a typhoid state, in which delirium alternates with stupor.

The *urine* is variable in quantity and character. It must not be forgotten that the course of the disease and the urinary symptoms are often quite variable in chronic nephritis. The urine may be normal in amount, but during the exacerbations it is scanty or suppressed. The specific gravity and the amount of urea lessen. In the most rapid cases it varies between 1012 and 1020. In chronic cases it falls as low as 1005 and even 1001. In the later stages the amount of the urine and the specific gravity may both be increased. Albumin is present in large amounts. When the disease is most active, and the dropsy at its height, the quantity of albumin is very large. In the quiescent period of the disease the amount is lessened. Casts are abundant, both epithelial, fatty, and granular; red blood-cells are often found.

Retinitis albuminurica is frequently developed in the course of the disease.

Dyspnœa is a common symptom. The dyspnœa may be due to any one of the many causes previously described which produce this symptom in the course of nephritis. It is frequently limited to sudden attacks which develop in the night or early morning. There is often some bronchial catarrh.

Nausea and *vomiting* are common symptoms. The appetite is lost.

Hypertrophy of the *left ventricle* takes place in all cases, except in those who had been previously weakened by other disease. The right ventricle is often hypertrophied also. The second aortic sound is accentuated, and the pulse is of high tension. Symptoms, such as *headache* and *vertigo*, arise on account of the profound *anæmia*.

The disease is characterized in its course by remissions and exacerbations. During the exacerbations any one of the prominent symptoms that occur in renal inflammations may be present. Œdema is the one symptom which occurs most frequently, and is likely to continue the longest. The disease lasts from three months to three years, and may pass into the second variety of chronic inflammation.

Course of the Disease. Delafield has well outlined the course. The constant symptoms are anæmia, dropsy, and albuminuria. 1. The

symptoms may be continuous and progressive in severity, death taking place at the end of one or two years, on account of dropsy or uræmia. 2. The symptoms may continue for several months, and the patient finally improve. Recurrent attacks take place, the symptoms being more severe with each attack. In the intervals of the attacks there is a small amount of albumin in the urine. 3. The patient may apparently recover, but the urine continues to be of low specific gravity, and contains some albumin. A fatal attack of uræmia, or an apoplexy, or the onset of an acute disease may cause an exacerbation of the renal symptoms. 4. The symptoms may persist in a mild degree for years, the patient at the same time feeling comparatively well. 5. Spasmodic dyspnœa may be the first and only symptom for a long time.

Chronic Productive or Diffuse Nephritis without Exudation. This is the form of nephritis which is also called *interstitial nephritis*, *granular kidney*, or *cirrhosis of the kidney*.

The kidneys are diminished in size, the capsules are adherent, and the surface roughened. There is an overgrowth of connective tissue with atrophy of the epithelium and of the tubules, and dilatation of some of the tubes, forming cysts.

Causes. This form of nephritis follows chronic congestion of the kidney, and is also caused by alcohol, lead, gout, syphilis, malaria, and by chronic endarteritis. The latter condition, as well as cirrhosis of the liver and pulmonary emphysema, frequently develops hand-in-hand with the nephritis. This form of nephritis is notably prevalent in several generations of different families, so that an hereditary history is often readily obtained.

Symptoms. The onset of the disease usually occurs late in life, although well-defined cases may occur as early as the twenty-fifth year. The progress at first is very insidious, and the disease may have advanced to an extreme stage without the occurrence of a single symptom. Death, indeed, may be due to other causes; or a person in apparently perfect health may suddenly manifest symptoms of uræmia, or may develop apoplexy or some other usual accompaniment of interstitial nephritis.

The *urine* is increased in amount, clear in color, and of low specific gravity. The albumin is small in amount, or may be absent. Repeated examinations extending over a considerable period of time may disclose its presence. Hyaline casts are present in small numbers. In some cases it may be necessary to examine a dozen or fifteen slides before they are found. Sometimes there are a few red blood-cells. Rarely the urine is bloody at irregular periods in the course of the disease, or actual hæmaturia may take place. With the exception of the state of the urine, the only symptom present may be the loss of flesh and strength. At the same time the skin becomes dry and harsh. Œdema, however, is not usually present unless there is dilatation of the heart. Special symptoms are due to uræmia, to changes in the heart and arteries, and to neuroretinitis.

The Heart. The left ventricle hypertrophies. The aortic second sound is accentuated. The pulse is of high tension. The arteries become more prominent, and present all the signs of endarteritis. In

the later stages, as nutrition fails, dilatation of the heart takes place, with regurgitation at the mitral valve, and the development of a train of symptoms due to these changes. Among others we find general malaise, palpitation of the heart, dyspnœa, œdema, and visceral congestions.

Uræmic Symptoms. These symptoms may occur at any time in the course of the disease. Headache is most common and constant. It may occur early in the morning only, or continue throughout the day. It may be continuous and cause sleeplessness. General neuralgic pains may be present instead of severe headache. Muscular twitchings or general convulsions may be other pronounced symptoms, or, instead, delirium, mild or violent, stupor, and coma may come on. These symptoms occur suddenly or develop very gradually. In acute uræmia with the above-mentioned cerebral symptoms there is peripheral spasm of the arteries, causing high arterial tension, and there is elevation of the temperature. The fever may rise to 103° or 104°, but is usually about 102°, and is irregularly continuous. After the patient lapses into deep coma, if the attack is fatal, the tension of the pulse is lost, and it is increased in frequency and diminished in strength. In chronic uræmia the cerebral symptoms develop gradually. The temperature is likely to be subnormal, particularly if diarrhœa or other debilitating influence is coincident. The pulse is rapid and feeble.

Pulmonary symptoms due to uræmia are quite common. They may be the first expression of uræmia. This is seen in all forms of nephritis. The most marked symptom is dyspnœa, which is spasmodic and of short duration. The attacks may occur frequently, and are usually increased by exertion and aggravated by a recumbent posture. The shortness of breath may occur in the early morning hours, or may continue throughout the day.

Pulmonary symptoms, other than those of uræmia, may be due to an intercurrent bronchitis, pneumonia, or pleurisy. Chronic bronchitis or œdema of the lungs may be present, on account of dilatation of the right heart. The chief pulmonary symptoms that point to these conditions are dyspnœa and cough.

Spasmodic dyspnœa is the first and sometimes the only symptom for a long time. Later the renal symptoms become pronounced, pointing to the true nature of the disease.

Gastro-intestinal Symptoms. Catarrhal gastritis almost always complicates nephritis. In addition, gastric symptoms due to uræmia, and hence to deficient action of the kidney, ensue. The most common is the occurrence of morning nausea or of morning vomiting; the occurrence of spasmodic vomiting at irregular periods, or the occurrence of violent, acute vomiting, which is followed in two or three days by other symptoms of uræmia. The patients are usually constipated. When the disease is complicated with cirrhosis of the liver, intestinal catarrh is common, and intestinal ulceration with consequent diarrhœa is frequently found. The onset of uræmia may be characterized by violent and profuse serous purging, which of itself may cause collapse and death.

Neuroretinitis is a frequent complication of nephritis, and may advance more rapidly than other complications, so that dimness of

vision, blindness, or other eye-symptoms may cause the patient to consult an oculist before attention is called to the condition of the kidneys. The occurrence of this complication points at once to the necessity of an examination of the urine.

It is common, in the course of an interstitial nephritis, to have accidents due to the condition of the arteries that accompanies this disease. On account of the atheroma, aided by the hypertrophied heart, rupture of the vessels frequently takes place. Apoplexy is, therefore, of common occurrence, and hemorrhage into other organs sometimes occurs.

The renal disease is often not suspected until after the patient has had an attack of apoplexy. The course of this form of nephritis is varied very much by the occurrence of complications, notably emphysema, endocarditis, or cirrhosis of the liver.

Catarrhs. There is always a tendency to chronic inflammations of the mucous membranes, and to acute inflammations of serous membranes in the course of chronic diffuse nephritis. It is necessary, therefore, when local inflammations of this character are present, to make thorough and repeated examinations of the urine, especially in a patient over forty years of age, with a history of one of the causal factors previously mentioned.

Course of the Disease. Several clinical forms of interstitial nephritis are observed. In the latent form the disease may have advanced to an extreme degree without any symptoms of renal disease during life, death taking place from an intercurrent disease or accident. On the other hand, palpitation of the heart may be the only symptom complained of, and the observer finds a hard pulse, general atheroma, and hypertrophy of the left ventricle with accentuation of the second sound. Apart from this the patient may enjoy very good health. The danger lies in the occurrence of pneumonia or inflammation of a serous membrane. Often the local inflammatory symptoms are slight or masked by the symptoms of renal disease, which develop rapidly.

In another group of cases some special symptom only may be complained of. In some instances it may be gastric catarrh, in some eye-symptoms alone may be present, while in others hemicrania or other forms of headache are observed. With the headache there is usually vomiting. Again, we may have constant neuralgia or persistent muscular rheumatism as the only symptom. Nose-bleed is a symptom which may be the only indication of chronic nephritis, particularly if the epistaxis occurs frequently.

In other cases the course is not latent, but characterized by a series of attacks at varying intervals.

During the attacks the symptoms resemble the acute form of nephritis, with acute uræmia, the occurrence of dyspnœa and loss of appetite, nausea and vomiting. The tension of the arteries is higher at the time of the attacks. The urine contains albumin, and is of low specific gravity during the time of the attack; during the interval the albumin is found at irregular times.

Suppurative Nephritis (Abscess of Kidney). Infectious matter is conveyed to the kidney either through the *blood*, as in pyæmia and ulcerative endocarditis (rarely dysentery and actinomycosis), or by the

ureters, as when it follows pyelitis or cystitis. A wound may infect the kidney directly.

Symptoms. The symptoms are those of primary disease, and the affection is usually only recognized post-mortem. Or the symptoms are merely those of suppuration. Pus is seen in the urine only on rupture of the abscess into the pelvis of the kidney.

Tubercular Nephritis. Fever, emaciation, anæmia, and prostration characterize the course of the disease. Tuberculosis is usually found elsewhere. There may be no other symptoms. Sometimes hydronephrosis is present. A tumor is often present. It may be in the loins, or may be in front, above, and a few inches to the right or left of the umbilicus. The *urine* is normal or contains pus and detritus or even bacilli. The finding of the latter is necessary often to establish a diagnosis. In all instances of pyuria renal tuberculosis should be suspected. Catheterization of the ureters may disclose the organ affected. The urine should then be centrifugalized and the sediment examined for bacilli, and, as Reynolds points out, a portion inoculated in guinea-pigs. The tuberculin test may be employed. The testicles and bladder should be carefully examined for primary tuberculosis.

Tuberculosis of the kidney presents symptoms like those of pyelitis, renal calculus, or a new growth. It is almost impossible to distinguish any one of the four until an interval has elapsed. In all cases the patient suffers from dull pain, sometimes with a bearing-down sensation. Hæmaturia occurs, and the patient is liable to attacks of renal colic. These symptoms may continue until a tumor can be made out. Even before this pain will be elicited on palpation, which may extend all along the urinary tract. With the occurrence of the tumor the general symptoms of tuberculosis arise. Further diagnosis is based upon the results of the urinary examination.

The Degenerations.

Degeneration may be either acute or chronic. The process is always secondary, due to the action of inorganic poisons, as arsenic or phosphorus, or the poison of infectious disease, or is produced as the effect of chronic disease of the organs, or by disturbance of the circulation.

In *acute degeneration* of the kidneys the urine is unchanged, or its quantity is diminished. It contains a little albumin, or the albumin is present in large amount, with casts and blood-corpuscles.

There may be no symptoms except changes in the urine, or symptoms of uræmia may develop at once. Dropsy and hypertrophy of the heart do not occur.

Chronic degenerations in the kidneys follow chronic congestion, or are produced by alcoholism or syphilis. They occur in the course of pulmonary phthisis, and of chronic suppuration; they may develop in the course of gout or malarial cachexia. *Symptoms:* In the simpler forms there may be no clinical symptoms whatsoever. In others there is loss of flesh and strength, the development of anæmia, and, in rare instances, the development of the typhoid state.

The changes in the urine vary. It may be abundant, scanty, or suppressed. The specific gravity is not changed, but albumin and casts are found.

Amyloid degeneration of the kidney is associated with similar degeneration in other organs. It occurs in the course of phthisis, of chronic suppurations, of syphilis, of chronic dysentery, and is thought to occur in the malarial cachexia, or with gout. *Symptoms:* The degeneration may be present without clinical symptoms. If symptoms arise, they are due to the anæmia and cachexia that attend the primary disease, and to the involvement of the other organs in the same process, as the liver, spleen, and intestines. Purdy says dyspepsia is prominent and diarrhœal attacks are common. The liver and spleen become enlarged during the course of the disease in the majority of cases. Œdema may be present, although it is more frequently absent. Uræmia is of rare occurrence. In the uncomplicated degenerations there is no hypertrophy of the left ventricle, and albuminuric retinitis is a rare complication.

The Urine. It may be diminished, normal, or increased, usually the latter; it varies from time to time in the same case, depending upon complicating symptoms, as diarrhœa, which causes diminished amount of urine. It is usually very pale. The specific gravity is not constant. It ranges from 1008 to 1014. Albumin is constantly present, and usually in considerable amount. Hyaline casts and white blood-cells are always found. When other casts are present nephritis probably complicates the condition. The chief distinctive feature of the casts is their large size and hyaline character.

The *diagnosis* of amyloid disease is based upon the presence of the cause; changes in the urine; and signs of similar disease in other organs.

CHAPTER VIII.

DISEASES OF THE NERVOUS SYSTEM.

The Data Obtained by Inquiry.

The Social History. This includes a knowledge of the patient's occupation, whether he or she is married or not, the conditions under which he may live, as, for example, in cases of great wealth, there is perhaps more tendency or at least more opportunity to dissipation; in conditions of poverty the patient may have been insufficiently nourished, or have suffered from continual anxiety. The most important factor is probably the occupation. Occupations, from a clinical stand-point, may be divided into those that require mental exertion, those that require physical exertion, and those that expose the workmen to the possibility of various forms of intoxication.

The Family History. This is perhaps of more importance in connection with nervous diseases than in connection with those of any other system. By *neurotic heredity* we mean the fact that in certain families a tendency to the development of various forms of nervous disease exists, which may be manifested, however, only in certain members of a given generation. Various terms are employed, to indicate the nature of the inheritance. *Direct inheritance* means that the child suffers from exactly the same disease as its parent. If both parents have the same disease, the child is likely to have it more severely, and this is spoken of as cumulative inheritance. By *indirect inheritance* is meant the condition in which collateral ancestry and not the parents have had the disease. Both the parents of the child may appear to be healthy, and the grandparents have suffered from the same disease, and this is called atavistic inheritance. By *similar inheritance* is meant the occurrence in the offspring of a disease similar to that from which the parents have suffered. Examples of such diseases are Huntington's chorea, Goldflam's periodic paralysis, etc. By *dissimilar inheritance* is meant the development in the offspring of a form of nervous disease differing from that which existed in the parents, as an epileptic child born of parents suffering from neurasthenia, hysteria, or insanity. The indications of neurotic heredity are manifold. Inquiries must be made in regard to cases of insanity, to cases of epilepsy, to instances of suicide, to peculiarities of character, to criminal tendencies, to addiction to the use of drugs, such as alcohol or opium; to congenital deformities, or to congenital diseases, such as deaf-mutism, etc. Charcot has called attention to the fact that certain of the so-called rheumatic manifestations may occur in the antecedents of a patient suffering from nervous disease.

The History of Previous Diseases. This is of considerable importance. The infectious diseases are sometimes followed by peripheral

neuritis or lesions in the central nervous system, or they may produce an early tendency to arterio-sclerosis. It is of importance to know whether the fœtal existence of the patient was normal, and, if possible, to obtain data concerning the condition of the mother during this period. Inquiry should be made regarding the nature of the birth; the existence of infantile spasms, at what age they occurred, when they ceased, if at all, and if there was any suspected reason for their development. It should be noted when the child first walked, when it first was able to talk, the rapidity of its intellectual development and progress at school, whether the character was normal, if there were night terrors or nocturnal enuresis. In boys the physician should endeavor to discover if there is any history of severe injury, particularly to the head, whether the boy had the opportunity for free exercise or was restricted in this respect; if his habits were good; if he smoked early; if he was overworked at school or obliged to work hard during early adolescence. In the case of females the physician should inquire at what period puberty occurred, and whether there has been any difficulty with menstruation. The existence of luetic infection is often difficult to elucidate. Occasionally it will be admitted, but more frequently it is necessary to discover the fact by indirect questioning.

The History of the Disease Itself. As in other conditions, the patient should be questioned regarding the duration of the disease, its earliest manifestations, whether exacerbations and remissions have occurred, and the nature of its course. It is important to inquire for slight symptoms that are usually overlooked by the patient, such as the ocular disturbances, ptosis, paralysis of the external rectus in locomotor ataxia, a tendency to extravagance in paresis, the manifestations of nocturnal epilepsy, etc.

The Subjective Symptoms. The data obtained by inquiry include the subjective sensations of the patient. These are chiefly of two kinds—pain and paræsthesia. In addition, the patients sometimes complain of a general feeling of restlessness, of irritability, of inability to think consecutively, or various other forms of indefinite general and intellectual disturbance. Pain is, however, such an important symptom in general disease that it has been discussed in the section upon General Diagnosis.

Paræsthesiæ may be defined as subjective sensations, either resembling those normally occurring as a result of excessive stimulation of the sensory nerves, or of a peculiar nature. They are exceedingly various in their character, and may be sharply localized or indefinitely distributed. To them belong chiefly itching, tingling, formication, numbness, subjective sensation of heat or of cold, of moisture, of pressure, or of tearing or rending. Sometimes the paræsthesiæ are very slight in character, and may escape the notice of the patient until his attention has been directed to them; in some cases they become so severe as to cause intense suffering and temporary helplessness. They usually indicate some functional or organic disturbance of a nerve-trunk, and are, therefore, as a rule, limited to the distribution of some particular nerve. The functional forms, however, may be produced by external conditions, such as pressure upon the bloodvessels leading

to a local anæmia, exposure to cold, to heat, and the like. A peculiar type of this condition is known as meralgia paræsthetica, and is characterized by paræsthesiæ in the distribution of the external cutaneous nerve of the thigh. In this disease, and occasionally in other forms of paræsthesia, the subjective symptoms are associated with objective disturbances of sensation.

The Data Obtained by Observation.

These include nearly all the important symptoms of nervous disease, and are, therefore, of paramount importance. They are disturbance of sensation, of motion, of reflex action, of appearance and of contour, disturbances of the special senses, of the functional activity of the various organs of the body, and alteration of the condition of nutrition.

Sensation. New varieties of sensation appear to be discovered every year, and it is therefore tedious and sometimes impossible to analyze all that have been already described. Sensations may be described as those which are relatively simple—that is, involving but a single variety of perception, and those that are complex.

Simple Sensations. *Tactile sensation*, or the sense of touch, is usually spoken of as æsthesia. It is the ability to know when some external object has come in contact with the skin. *Hyperæsthesia* is an increased sensitiveness to contact; and *hypæsthesia*, decreased sensitiveness; *anæsthesia*, total loss of the ability to perceive objects touching the skin. No satisfactory instrument for the measurement of the touch sense has as yet been devised. In general, it may be tested either directly with the end of some hard, blunt object, or, when still acute, with a camel's-hair brush or cotton point. The patient should close his eyes, or, what is better, permit them to be bandaged, and should then be instructed to indicate by some word or motion the moment contact takes place. The investigator must be careful not to use force, and the instrument employed should not be so sharp nor so rough as to produce pain. From time to time the patient should be asked whether he were touched when contact has not been made, although some movement indicating the approach of the instrument to the skin has been performed. Frequently in prolonged examinations the attention becomes fatigued, and the patient no longer recognizes whether he is touched or not, and answers at random. *Hyperæsthesia* may occur in a variety of conditions. Its most common cause is functional exaltation or irritability of the nerves, which may occur in neuralgia or neuritis. It also occurs in organic disease of the cord, and is then limited to the area of distribution of the spinal segment just above the destructive lesion. This is spoken of as the zone of hyperæsthesia. It is also occasionally present in functional conditions, such as neurasthenia and hysteria, and may be merely the result of some local irritation of the skin. The degree of tactile perception varies considerably in different persons. *Hypæsthesia* may occur in a variety of conditions—in neuralgia, in partial lesions of the spinal cord, particularly disease of the posterior columns, and rarely in central lesions of various kinds, particularly those occurring in the parietal lobe, in the end of the posterior limb of

the internal capsule, and in the pons. It also occurs in functional nervous conditions, and is quite common among the insane. *Anæsthesia* results from solutions of continuity of the sensory nerves, from destructive lesions of the cord, or from central lesions. It is also the commonest form of hysterical stigma. Organic anæsthesia may be distinguished from functional anæsthesia by its distribution. If caused by nerve injury, it will exist in the region supplied by that particular nerve. If caused by disease of the spinal cord, the area of anæsthesia will be segmental in type—that is, bounded by two nearly horizontal lines passing about the body. In unilateral lesions of the spinal cord the anæsthesia is limited to the opposite side of the body. In central disease the anæsthesia is commonly unilateral, and corresponds to the paralyzed side, if paralysis is present. If due to a lesion of the cortex, however, it may be limited to one extremity, where it is usually associated with paralysis.

Pain sense, or *algesia*, is the ability to perceive pain of any kind. Various instruments have been devised for testing its intensity. Among the best is that suggested by Kulbin, consisting of a needle which is thrust into the skin for varying distances; the amount of pressure required and the degree of penetration being indicated on a scale. Even this, however, is far from accurate, and for clinical purposes it is sufficient to use a needle or pinch a small fold of skin between the finger-nails. In case of very pronounced disturbance of the pain-sense it is sometimes possible to use the actual cautery or to thrust a needle entirely through a thick fold of the skin. A faradic current is also frequently employed, and to a certain extent is accurate, if data can be obtained by comparing the healthy with the diseased side of the body. As, however, it appears that there is a special form of sensation for the induced current, its results cannot be relied upon implicitly. *Hyperalgesia* is increased susceptibility to painful impressions, so that the lightest contact may cause exquisite agony. It occurs in inflammation and in those conditions associated with hyperæsthesia. A variety of hyperalgesia is *tenderness*—that is, pain elicited by simple pressure. It is most frequently associated with local inflammation, and occurs along the course of the nerves in neuritis and neuralgia. *Hypalgesia*, or decreased susceptibility of pain, occurs as a result of partial lesion of the nerves, or of the central portion of the spinal cord, and, occasionally, as a result of focal lesions in the brain. It is also very common among idiots, immediately after epileptic attacks, and in cases of hysteria. Hypalgesia may also be acquired as a result of constant exposure to a mild form of irritation, as, for example, in those accustomed to going bare-footed. *Analgesia* is an exceedingly important symptom. It results from total destruction of the nerve; from disease of the central gray matter of the spinal cord, such as occurs in transverse myelitis, syringomyelia; in tumors of the cord; and from focal disease of the brain, particularly if situated in the parietal lobe, and the posterior limb of the internal capsule. It also occurs in a great variety of functional conditions, and may be general in the form of insanity known as primary stupor. It is a very common lesion in hysteria, and in this disease the area of distribution may assume the

most curious forms, being limited to one-half of the body, or tracing geometrical figures on various parts of the skin. It may also be produced by hypnotic suggestion. Organic analgesia is frequently associated with trophic changes, either as a result of the inability of the part to defend itself against irritation, or as a result of the intimate association of the sensory and trophic nerve-fibres.

Visceral pain may be elicited by strong pressure upon the testicles, ovaries, or breasts, or a violent blow upon the abdomen. It is usually characterized by intense prostration and nausea. Visceral analgesia occurs in some cases of locomotor ataxia and occasionally in hysteria.

The heat sense, or *thermoæsthesia*, enables us to recognize the difference in temperature between various bodies. It is usually tested by filling two test-tubes, one with hot and one with cold water, and applying them in irregular alternation to the region under investigation. The difference in temperature between the two tubes is a rough test of the delicacy of the sense. In health a difference of 1° C. can be recognized upon the more sensitive portions of the body (the anterior surface of the forearms, the skin of the face, and the chest). A rougher test is the use of metal and wooden objects. The former conduct heat more rapidly from the surface, and therefore give rise to a sensation of cold. The heat-sense is rather complicated, and is not yet thoroughly understood. There seem to be special points upon the skin where the nerves for heat and cold terminate. (Goldscheider.) There may be loss of perception for cold objects, while the perception for hot objects remains unimpaired, or the reverse may be present. Sometimes the patient calls all objects warm and at other times he calls them cold. *Hyperthermoæsthesia* is practically of no value as a clinical sign, for our methods of testing the delicacy of the sense are at present imperfect, and *hypothermoæsthesia* is also difficult to detect, and probably belongs to the category of conditions in which one of the sensations is more or less impaired. *Thermoanæsthesia*, or complete loss of the heat-sense, is very important clinically. It occurs in neuritis or destructive lesions of the nerve, and in central disease of the spinal cord, such as transverse or pressure myelitis, tumor, and especially in syringomyelia. As a result of being most frequently associated with cord disease, the thermoanæsthetic area is usually segmental. The heat-sense may, in connection with other forms of sensation, be diminished in functional nervous disease.

The above three forms of simple sensation are those usually regarded as of the greatest clinical importance. They may be equally affected, or one or two may be preserved and the others diminished or lost. The latter condition is known as *dissociation of sensation*. It occurs in neuritis, but is exceedingly rare. It also occurs in various forms of myelitis, particularly pressure myelitis. It is the most characteristic symptom, and for a long time was considered pathognomonic of syringomyelia. In this form of dissociation tactile sense is preserved, and the temperature and pain senses are lost. When the tactile sense is lost, and the pain sense still present, the condition is termed *anæsthesia dolorosa*.

Simple sensations of perhaps less clinical importance than the foregoing are *trichoæsthesia*, or the consciousness that a cutaneous hair has

been touched. This is really the sensation perceived when tactile sense is tested with the cotton point; the latter is felt very well upon the forearm, on the back of the hand, and not on the palm, where sensation is distinctly more acute. Von Bechterew calls attention particularly to the fact that trichoæsthesia and tactile sense are not equally delicate in various parts of the body. The former is most readily tested by touching the individual hairs with a small needle or cotton point. More elaborate apparatus of no particular value has, however, been devised.

The Sensation of Locality. When any part of the surface of the body is touched we can, under normal conditions, tell the location of the point of contact. This varies, however, considerably in various parts of the body, being more accurate on the lips and less on the skin of the back between the shoulder blades, where an error of from 6 cm. to 7 cm. is still within the normal limits. It may be very much disturbed without any loss of the delicacy of the touch sense. It may be tested by making contact with the finger or any blunt object. Another method formerly much used by clinicians, and still employed by psychologists, is the use of the *œsthesiometer*, an instrument consisting essentially of two points that can be placed at a measured distance from each other. It has been found that in normal persons these can be detected as two points at the tip of the tongue when separated only 1 mm.; but may still be felt as one on the back when separated as much as 65 mm. This method is extremely inaccurate, for the reason that it is difficult to apply the points with the same degree of force. Moreover, experiments have shown that the skin readily becomes educated and able to discriminate points much closer together than is normal for the part that is being tested.

Allochiria. This is a general term applied to the false localization of sensory stimuli. In some cases the sensation may be felt not at the point where it was applied, but at exactly the corresponding point on the opposite side of the body. This occurs particularly in hysteria. In organic disease of the spinal cord mistakes of localization are not uncommon—thus, in hypæsthesia of the arm, irritation at the hand may be referred to the shoulder, and the same is true of the lower extremity.

When there exists a hypæsthesia it is of course difficult for the patient to localize as accurately as is possible when sensation is normal.

The Electro-cutaneous Sense. This is really the degree of resistance to the irritation of the induced current. It varies considerably in different individuals, and in the same individual under different conditions and in different parts of the body. It is perhaps most delicate on the skin of the face, and least delicate on the back and the outer surface of the thighs. It is curiously affected in certain nervous diseases; thus, in the periodic paralysis of Goldflam it is almost completely abolished during the attack. In meralgia paræsthetica it is also, as a rule, greatly diminished. In all cases of muscular degeneration the electric current is better supported than when the muscles react. It is also greatly diminished when there is œdema of the skin or much subcutaneous fat. It sometimes persists, however, when tactile anæs-

thesia is present. In tetany it is greatly exaggerated (Erb's sign), and this constitutes one of the cardinal symptoms of the disease, and it is also increased in some of the functional nervous conditions. It is best tested by using a simple faradic battery, employing as the electrode for contact either the wire brush or the naked wire. No satisfactory system of measurement has as yet been devised, but it is of advantage to use invariably the same battery, and to note the position of the inner coil with reference to the outer one.

Pressure Sense. The clinical significance of this has not yet been determined. It is certain, however, that it undergoes considerable variation as the result of various pathological changes. It may be tested roughly by making various degrees of pressure with the finger or a blunt object upon the surface of the skin, the limb being so placed that it is impossible for the patient to make muscular resistance. It may be tested more accurately by using a series of little blocks that can be piled one on top of the other, or by filling a vessel more or less completely with shot or mercury.

Functional Modifications of the Various Forms of Sensation. *Delayed Sensation.* The perception of the various forms of stimulation that are appreciated in consciousness as sensations may be delayed for some time after the stimulus has been applied. This is spoken of as delayed sensation, and the interval may, in extreme cases, be several seconds. It is not known where this delay takes place, whether in the sensory bodies of the skin, or in the nerves, or in the central nervous system. This symptom is manifested particularly in tabes dorsalis, but may occur in functional nervous disease and in various forms of organic central disease. It has also been noted in peripheral neuritis. The delay can occur for one sensation alone, as the pain sense, even when tactile sense is normal.

Complex Sensations. These are probably very numerous, but only two have been so carefully studied that they are valuable for clinical purposes. These are the so-called position or muscular sense, and the stereognostic sense. By the *position or muscular sense* we mean the ability to perceive and recognize the position of the limbs or of the body—that is, whether, for example, the joints are in a state of flexion or extension, supination, pronation, or rotation; whether the spine is bent or erect, and the position of the head with reference to the trunk. It probably depends upon the complex co-ordination of the perceptions received from the muscles, joints, periosteum, tendons, and skin. It may be tested in a variety of ways. The patient should be instructed to close his eyes or have them bandaged; the finger is carefully grasped on either side and flexed or extended. After each movement the patient indicates its direction. After the fingers have been tested the same process is employed for the wrist, elbow, and shoulder. Similar methods may be used for the feet, and the head may be rotated to the right or the left, bent forward, laterally, or backward. Another method is to take one arm, bend it into some particular position, and instruct the patient to imitate the position with the other arm; the same thing being done with the legs; or the patient may be instructed to describe the position in which his arm has been placed, without attempting an imi-

tation. This sense is lost when for any reason there is total anæsthesia of the part, and may disappear as an isolated symptom in case of disease of the posterior columns or in the ataxia due to central lesions. By the *stereognostic sense* we mean the ability to recognize the shape, consistency, surface, and nature of any object placed in the hand or brought in contact with the skin of any part of the body. This sensation is most readily tested by directing the patient to keep the eyes firmly closed; then to select a number of small objects, such as a pencil, match-safe, coin, key, etc., and place them in his hand and direct him to name them or describe them. This sense depends upon a variety of perceptions. The size of the object is recognized by a combination of the locality and muscle senses; the nature of its surface by the tactile sense; its consistency chiefly by the pressure sense, perhaps aided by the pain-sense; its nature—that is, whether of metal, wood, or any other substance—largely by the temperature sense. The stereognostic sense is always abolished when tactile sense is absent. Occasionally in hysteria the patient may declare himself unable to perceive touch when the stereognostic sense is intact, but this is an exception. It may, however, be lost when tactile sense is still preserved, especially if the locality sense and the muscle sense have been greatly impaired. When due to organic causes its absence usually indicates a lesion in the parietal lobe or in the projection fibres coming from this region. It occurs frequently in hemiplegia, in cerebral monoplegia, and occasionally in peripheral palsy, involving two forms of sensation. It has also been observed as a transient symptom after brain shock without disturbance of any other sense.

Disturbance of Motility. These may be grouped under a number of heads. First, loss of power, which may be either partial, *paresis;* or complete, *paralysis.* Second, impairment of movement, *inco-ordination,* or *ataxia.* Third, closely allied to this, *tremor.* Fourth, excessive muscular movement, *spasm,* or *convulsions.*

Paralysis. This is a loss of power in the muscles. It may be true, in which the loss of power is due to some disease of the muscle itself or the nervous influence controlling it; or false, when it is due merely to an inhibition of the muscular function produced by a disease of the muscle or joint that causes pain upon movement. Paralysis is classified, according to the part affected, into *monoplegia,* when one extremity is involved; *hemiplegia,* when half the body is involved; *paraplegia,* when two symmetrical extremities are involved; *paraplegia cruralis,* if the legs are affected; *paraplegia brachialis,* if the arms are affected (this term is usually restricted clinically to paralysis of both legs); *diplegia,* when two extremities are affected without involvement of the trunk. Clinically, this is sometimes restricted, although incorrectly, to paralysis of both arms (*diplegia brachialis*) or of both sides of the face (*diplegia facialis*). *Crossed paralysis* is a term applied to paralysis of one side of the face and the opposite side of the body. *Local paralysis* is the term used when only small groups of muscles are affected. *Multiple paralyses* is employed when several parts of the body are involved at the same time. Paralysis is also classified, according to the cause, into *cerebral paralysis, spinal paral-*

ysis, neural paralysis, and *muscular* or *myopathic paralysis*. Paralysis is also classified, according to the type, into *spastic paralysis*, in which the muscle tone is increased and the reflexes are exaggerated, and contractures are either present or likely to ensue, and *flaccid paralysis*, in which the muscle tone is diminished, in which there is no resistance to passive movement, and the reflexes are abolished. Spastic paralysis is usually due to some lesion in the central motor neuron—that is, between the motor cortex and the terminations of the fibres of the pyramidal tracts in the anterior cornua of the spinal cord. The lesion, therefore, may be situated in the cortex, the corona radiata, the internal capsule, the pons, the pyramids of the medulla, and the lateral columns of the cord. Spastic paralysis must not be confused with the contractures that ensue after degeneration of the muscles, as in infantile palsy, neuritis, etc. In these cases the limbs are in a state of permanent flexion. The resistance to extension and to passive movement is not due to increased muscular tone, but to an actual shortening of the muscle and its tendons, which can only be overcome by rupture of one or the other. Flaccid paralysis may be produced by cerebral lesions, but is more commonly due to lesions of the peripheral motor neurons —that is, from the anterior cornua of the cord to the muscle itself. It may, therefore, be produced by destruction of the ganglion cells, by injury to the anterior roots, or the peripheral nerves, or disease of the muscle. Flaccid paralysis frequently occurs as the result of functional conditions—for example, it is the type of paralysis that is usually observed in hysteria. As the trophic centres influencing the muscle are either cut off or destroyed, atrophy of the latter usually takes place (*atrophic paralysis*), which is characterized by decrease in bulk, alteration of the electrical reactions, and fibrillary twitchings. *Monoplegia*, or paralysis of one limb, may be caused by small lesions in the cerebral cortex or the corona radiata. It is rarely produced by lesions of the internal capsule, where the fibres are placed closely together, or of the spinal cord, unless the gray matter of the latter is involved. It occurs in circumscribed forms of infantile paralysis, in lesions of the peripheral nerves, particularly the roots of the plexuses, but rarely in disease of the muscles alone, the lesions in this case being more widely distributed. Monoplegia also occurs in hysteria and in the pseudo-paralysis due to localized disease of the muscles or joints. *Hemiplegia* is commonly due to a lesion of the opposite side of the central convolutions. This lesion may either be extensive and destroy the motor portion of the cortex or corona radiata, or more circumscribed, involving the internal capsule, the crura, the pons, or the medulla. Spinal lesions, also, if unilateral, which is rare, and situated above the fourth cervical segment, may produce paralysis of the same side of the body. (*Vide* Brown-Séquard's syndrome.) In hemiplegia due to lesion of the cerebrum, the muscles of the trunk, and those supplied by the upper branch of the facial nerve commonly escape. The lower half of the face, the arm and leg, and the side of the body opposite the affected hemisphere are paralyzed. If due to lesion of the pons below the decussation of the facial fibres –that is, in the posterior half—the arm and leg of the opposite side and the lower half of the face on the

same side are paralyzed (*crossed paralysis, pontine palsy*). Lesions of the medulla ordinarily, in addition to the motor tracts, involve other important nuclei and tracts. *Spinal hemiplegia* is characterized by the absence of facial involvement. *Hysterical hemiplegia* can only be recognized in some cases by the discovery of the other stigmata of that disease. The form of paralysis in organic hemiplegia is ordinarily spastic, and usually in the course of time pronounced contractures occur. *Paraplegia cruralis* is usually produced by a lesion of the spinal cord. If this lesion is situated above the lumbar portion of the cord, the type of paralysis is spastic; if in the lumbar or sacral region, or involving the cauda equina, there is often abolition of the reflexes and flaccidity of some of the muscles. Paraplegia, therefore, occurs in transverse or pressure myelitis, in tumor of the spinal cord, in hemorrhage into the spinal cord, and as a result of traumatism. It is occasionally produced by multiple neuritis of the legs, particularly that form known as Landry's paralysis, or in alcoholic neuritis, by bilateral cerebral lesions, and occasionally as a functional condition. *Paraplegia brachialis* is a rare condition occurring chiefly as the result of a localized meningitis in the cervical enlargement, particularly pachymeningitis hypertrophica cervicalis. As the result of the destruction of the anterior roots there is atrophy and degeneration of the muscles, and the paralysis is flaccid. It may also occur in syringomyelia, and more rarely as a result of traumatic injury to both sides of the brachial plexus. *Diplegia facialis* is almost invariably the result of bilateral facial palsy—that is, either neuritis or an injury to the facial nerve after it leaves the medulla. The paralysis is, therefore, flaccid in type, characterized by the loss of the normal folds, and the inability to close the eyes and drooping of the corner of the mouth.

Multiple palsies are usually due to some general condition affecting the peripheral neurons—thus, in multiple infantile palsy the anterior cornua of the gray matter of the spinal cord are involved in various situations. The paralysis is usually flaccid and incomplete—that is, certain groups of muscles escape. In polyneuritis due to intoxication or *infection* there may be paralysis either of certain groups of muscles, particularly the extensors, or of the entire limb. This occurs most frequently in poisoning by lead, arsenic, and alcohol, or in infectious diseases, as beri-beri and diphtheria. The paralysis is nearly always flaccid; there is muscular atrophy, and the reactions of degeneration ultimately appear. *Local palsies* are usually due also to lesions of the peripheral neurons. Occasionally, however, a very small lesion in the cortex will produce this condition. They are commonly the result of some trauma injuring a single nerve-trunk. The paralysis is, of course, flaccid, and the reactions of degeneration are present.

A congenital absence of complete atrophy of the muscle gives rise to *myopathic paralysis*. In either case the diagnosis must usually be made by careful anatomical examination, as in the course of a very short time the patient learns to compensate the defect of the individual muscle by the excessive action of others in its neighborhood. The muscles most frequently affected by congenital absence are the pectorals, although many others also may be involved. Total atrophy

occurs in various myopathies, but with extreme slowness. In a special type of muscular atrophy (type of Duchenne-Aran) atrophy occurs in individual muscles or in small groups, and compensation is usually acquired for a considerable time until the progress of the disease renders it no longer possible.

Paresis is a term used to indicate partial loss of power in the voluntary muscles. In addition to the causes given for paralysis, it may be produced by exhaustion. Paresis is of two kinds—that in which the muscle is unable to exert its normal force at any time, and that in which the muscle may exert its normal force for a brief period and then rapidly becomes exhausted and insufficient. In the former there is some deformity, such as foot-drop or wrist-drop. In the latter the symptoms do not appear until some effort has been made. Paresis may also be temporary, as after fatigue; stationary, as in cases of injury to the central nervous system; or progressive, as in the myopathies. In the latter condition the muscles waste and lose their power, but reactions of degeneration do not occur, and there are no fibrillary twitchings. Ultimately, the condition may go on to absolute paralysis. The power of the muscle may be tested very accurately by means of the dynamometer. This consists of a steel spring with a staff on one side and a sliding index on the other. The patient compresses the spring in the palm of the hand, and the amount of pressure is indicated in pounds or kilogrammes upon the index. By various mechanical devices the dynamometer may also be employed for the other muscles of the body. Care should be taken when it is used that the patient is not permitted to throw his weight against it. In using the instrument it is chiefly important to regard not so much the absolute power as the difference between the two sides, the degree of muscular force normally present varying very greatly in different individuals. Clinically, it is often sufficient to have the patient squeeze the physician's hand first with one hand and then with the other; even moderate differences being readily detected by this means. The patient may also be instructed to resist passive movements, such as the extension of the flexed arm; the flexion of the extended arm; the lateral movement of the head; the opening of the eyelids, or the various movements of the lower extremities.

Intermittent claudication is a term applied to indicate the occurrence of transient, partial, or complete paresis or lameness. Sometimes the patient will suddenly be unable to continue locomotion, and fall to the ground; at others, one limb will become weak, causing a pronounced limp and necessitating the aid of a crutch; while in other instances there is simply discomfort upon continued locomotion. This symptom occurs in various forms of functional nervous diseases; thus the periodic paralysis of Goldflam, meralgia paræsthetica, and as an idiopathic condition in diabetes and arterio-sclerosis.

Disturbances of movement, characterized by excessive or perverted muscular activity, consist of ataxia, tremor, and spasm. By *ataxia* is meant the inability to co-ordinate perfectly—that is, to give each muscle its due share in the performance of any action. As a result the movements are irregular and imperfect. Various types of ataxia

have been distinguished: *Spinal ataxia*, in which the disturbances of motion are more pronounced when the eyes are closed, and which is due to disease of the posterior columns; *cerebellar ataxia*, in which the disturbances are equally severe when the eyes are opened or closed, but disappear when the patient lies down; *cerebral ataxia*, in which there is loss of muscular sense and marked persistent inco-ordination of movement, occurs as a result of injury to the parietal lobe; *pseudo-ataxia*, due to the weakness of certain groups of muscles, so that they do not properly oppose the action of other groups. Finally, there is a form of ataxia apparently due to anæsthesia of the skin and loss of the muscular sense, in which the patient is able to perform movements perfectly as long as he can watch the part with the eye, but as soon as the eyes are closed the ataxia appears Ataxia may be tested in a variety of ways. Ataxia of the upper extremities may be recognized by directing the patient to touch the tip of the nose with the tip of the forefinger, or to extend the arms and bring the tips of the forefingers together with a rapid motion. In health, after one or two trials, either of these movements can be done perfectly. When ataxia is present they are carried out awkwardly, and the forefingers are only brought in contact with each other or the tip of the nose after several irregular coarse oscillations. The ataxia of the legs may be tested by requesting the patient, lying upon his back, to touch some object held above his feet with one of the toes, or to bring the heel of one foot against the knee of the other. When the patient is erect the ataxia may be tested by getting him to place the feet together, when there may be some swaying that is usually very markedly increased when the eyes are closed. If the ataxia is very slight it may be necessary to get the patient to stand on one foot with the eyes closed, or to attempt to step backward under the same conditions. Under these circumstances a considerable swaying occurs that is more pronounced than the swaying noticed in a normal person attempting to perform the same movements. If the ataxia is at all pronounced it produces a characteristic disturbance in the gait. (See Ataxic Gait.) Ataxia of the head is difficult to detect. Some observers contend that a peculiar form of grimacing, whenever the patient attempts to move the lips or the eyes, or whenever the muscles of the face express some emotion, is an ataxic condition due to overaction.

Tremor. This is a disturbance of motion characterized by an oscillation of the part or parts involved. Tremor may be of various kinds. It may be fine or coarse, constant or irregular. It may disappear upon voluntary effort or only be apparent when motion is attempted (*intention tremor*). It may be the result of paralysis, *paralytic tremor;* of poisoning, *toxic tremor;* of some functional nervous disease, as the *hysterical tremor;* or spasm of the muscle, *spasmodic tremor;* or it may occur as a family peculiarity without any discoverable cause, *hereditary* or *idiopathic tremor*. Tremors are also classified as rapid, in which the movements occur more than five times per second; and slow, in which the oscillations may occur at intervals of several seconds. Nearly all forms of tremor are increased by placing the muscles upon a stretch. Tremor can usually be recognized by simple inspection. In some cases

it is necessary to use peculiar methods of detecting it. Ordinarily it is sufficient, in order to detect tremor of the fingers, to get the patient to extend them forcibly and keep them in that position. If the tremor, however, is exceedingly fine, its effect may be exaggerated by attaching long, light rods to the fingers, such as straws. This procedure is often exceedingly useful in cases of tremor of the head or the feet. Tremors may be recorded by attaching to the part affected rods whose ends are furnished with a pencil or stylet which writes upon a moving roll of paper. If a chronograph marks off seconds or fractions of a second at the same time, it is possible to measure very accurately the rate of oscillation. A more convenient method consists in allowing the patient to put the trembling part, for example, the hand, upon a small drum which conveys each movement to an oscillatory stylet that marks upon a piece of smoked glass or paper. Seconds should be marked at the same time. Persistent fine tremor occurs particularly in paralysis agitans. In this the movements in the fingers are those of flexion and extension and of opposition in the thumb, and it has, therefore, been spoken of as the *pill-roller's tremor*. It also occurs not infrequently in exophthalmic goitre and as hereditary or idiopathic tremor. Irregular tremors occur as a manifestation of ataxia, often with cerebral lesions (the paralytic tremor), and after intoxications, as alcohol and tobacco. The hysterical tremor may be either irregular or regular. Its character is largely influenced by surrounding circumstances; thus if the hysterical patient be in the hospital ward and have an opportunity of seeing a case of paralysis agitans, the tremor peculiar to that condition is often closely reproduced. Ordinarily, however, the hysterical tremor, being the result of voluntary and variable effort, is irregular. Intention tremor occurs particularly in multiple sclerosis. In this condition no tremor is observed while the parts are at rest, but as soon as voluntary motion is attempted a violent tremor ensues, and continues until the effort ceases. Such a tremor can be particularly well elicited by asking the patient to convey a glass of water to his mouth. The movements become more and more violent as the lips are approached, and frequently more or less of the water is spilled. It may also be tested by asking the patient to touch with the forefinger some object. It will be observed, as the finger approaches, that the oscillations become more vigorous and wider. Intention tremor may, of course, be present in other parts of the body. Generalized tremors are spoken of as convulsions or convulsive movements (*q. v.*).

Muscular Spasm. By this is meant a condition in which the muscle is involuntarily but forcibly contracted, either persistently (tonic or tetanic spasm) or rhythmically (clonic spasm). *Tonic spasms* are characterized by the vigorous contraction of the muscle, which becomes hard and painful. If only one group of muscles is affected, as, for example, the calf, the joint controlled by this group is placed in the position normally assumed when they are active. If all the muscles of the limb or even antagonistic groups are affected, the flexors usually overcome the extensors. This, however, is not invariably the case. When all the muscles of the body are involved, the powerful muscles of the back usually arch the spinal column, and there is a more or less

severe opisthotonos. Tonic spasms can usually be diagnosticated by simple inspection. They occur particularly in tetanus, strychnine-poisoning, and hysteria, and in these conditions may often be produced by peripheral irritation. Localized spasms in the upper extremities may occur as a result of disease of the cord above the cervical enlargement, or of the brain, producing a spastic condition of the muscles. This is rare. A more common type is the peculiar form of spasm seen in tetany, consisting in the closure of the fingers and the opposition of the thumb, giving rise to the so-called *obstetrical hand*. Spasms in certain individual muscles of the hand or arm occur in the occupation neuroses. Spasms of the lower extremities are also occasioned by the various conditions giving rise to spasticity of the muscles. An idiopathic form of spasm not infrequently occurs in the calf muscles, particularly on awakening. It appears to be of no clinical significance. Hysterical spasms are of various types. The tonic forms may affect a single limb or even a single group of muscles, and may persist for long periods of time, giving rise either to extension or persistent flexion of the limb. In the latter case shortening may ultimately ensue and cause persistence of the deformity. General hysterical spasms usually can be recognized by the fact that the patient assumes some extraordinary posture, as opisthotonos, pleurosthotonos, and emprosthotonos. These spasms are often precipitated by pressure upon some sensitive point (hysterogenic zone, ovaria), and may sometimes be abolished by pressing upon the same or a corresponding portion of the body. A peculiar form of localized tonic spasm is that occurring in the masseters, known as *trismus*. The *myotonic reaction* is frequently spoken of as a form of tetanic spasm. It consists of a sudden, persistent contraction of the muscle or groups of muscles with which some voluntary movement has been attempted. It occurs, as far as is known, only in Thomsen's disease. *Clonic spasms* are of various types. They may affect a single extremity, half the body, or, in rare cases, the whole body. The movements are usually rhythmical, and vary greatly in different cases. The most frequent causes of clonic spasms are the injuries to the brain. Focal irritation in the motor region will produce at first a spasm in the part innervated by that area. If the irritation is sufficiently strong, or acts for a sufficiently long time, its influence will extend to the adjacent areas in the cortex, and a general unilateral or bilateral convulsion will ensue. This is the so-called epileptiform attack. If the local spasm is distinct and precedes by some time the development of the general twitching, it is spoken of as *focal*, or *Jacksonian epilepsy*. As a result of the violent irritation in the brain, unconsciousness usually ensues, but not invariably. Clonic convulsions may possibly be of local origin, although this is exceedingly doubtful. Ankle clonus, however, and patellar clonus bear a certain resemblance to this symptom of disease. A localized form of clonic spasm, due to peripheral irritation in all likelihood, is *facial tic*, characterized by occasional or successive lightning-like contractions of the muscles of the face. Functional convulsions, particularly those occurring in hysterical patients, are very frequently clonic in character. Often there will be a preliminary tetanic spasm, followed in a short time by the development of clonic

movements. These assume various forms, the commonest being perhaps beating with the limbs, throwing of the head from side to side, and lateral or antero-posterior movements of the body. The attitudes and movements express fear, threat, ecstasy, eroticism, or other emotional states.

Allied to the clonic spasms, but bearing also close affinity to tremors, are the irregular movements that occur in chorea and athetosis. The typical movement of *chorea* is an irregular innervation of groups of muscles that appears to be voluntary in character, but that is not under the control of the patient, is much more rapid, as a rule, than a voluntary movement, and recurs at very frequent intervals. Choreic movements may be mild, or so severe that they produce irregular contortions of the body, causing the patient to throw himself or herself from side to side, and often producing severe bodily injuries and even death by exhaustion. *Athetosis* is a name given to a peculiar, slow, irregularly rhythmical movement of the extremities, generally spoken of as *worm-like* in character. It is ordinarily most marked in the fingers. In movement these are gradually extended until they form almost a right angle with the back of the hand, and then slowly flexed and extended again, each finger moving more or less independently of the others. At the same time there is movement at the wrist-joint, the elbow, and sometimes of the trunk. The limbs may be affected, giving rise to a curious, staggering gait in which the patient seems ever to be about to lose his equilibrium, but maintains it almost by a miracle. Frequently the muscles of the face are involved, giving rise to curious, irregular grimaces and more or less disturbance of speech or *dysarthria*. The movements are usually continuous. Athetosis is a very common sequel to cerebral lesions occurring in early childhood.

The term *convulsion* is used to designate general spasm with loss of consciousness. It is often employed, however, to indicate general clonic spasm of the whole body, even if consciousness be still present. This use is undesirable, and should be avoided. General convulsions invariably indicate some disturbance in the brain. If this is organic, it may be either some chronic disease with occasional exacerbation of cortical irritation, or some acute injury or some disease, such as meningitis. If it is some functional disturbance, it may be hysteria or epilepsy. (The latter is, of course, usually due to organic lesions.)

The term MUSCULAR TONE means that condition of the voluntary muscles of the body by which they are maintained in a state of tension sufficient to enable them to respond promptly to nervous innervation. Muscular tone varies slightly under normal conditions. It is less in profound fatigue, and when the attention is distracted by external objects; it is more marked when the patient concentrates his attention upon the part being tested. It is invariably diminished after lesions of the peripheral motor neuron, in cases of profound cachexia, in coma, and during anæsthesia. It is also generally decreased in lesions of the posterior columns of the spinal cord. It is increased in lesions of the central motor neuron without involvement of the peripheral neuron, in neurasthenia, hysteria, and in conditions affecting the brain as a whole, such as meningitis, brain tumor, etc. It must be remembered that

flaccid paralysis does not necessarily imply diminished muscle tone; thus in the early stages of hemiplegia the muscles are completely relaxed, but, nevertheless, the reflexes are usually increased. There are two methods of testing this quality: First, passive movements; second, the tendon reflexes. In the former the limb to be tested is grasped firmly, and, if flexed, is suddenly but not too forcibly extended, or, if extended, is flexed. If the muscle tone is normal there may be a transient, involuntary resistance at first, but this disappears very soon, and then the limb may be moved in any position with comparatively slight effort. Any of the joints may be tested independently in this manner. It is important to inform the patient what is to be done and what is to be tested. In children, in the ignorant, and in the insane it is often almost impossible to overcome the tendency to voluntary resistance, which is usually increased by the anxiety produced by the examination. Occasionally it is necessary to take some measures to distract the attention, such as giving the patient a sum in arithmetic to perform, requesting him to look at the ceiling or some particular object, or engaging him in conversation. Increase of the muscle tone is determined by increased resistance to passive movements. This may be so great that it is almost impossible to bend the limb at any of the joints, or so slight that it is difficult to discriminate it from the normal condition.

The exaggerated forms are usually spoken of as *spasticity* of the muscles, and when associated with paretic or paralytic conditions the term *spastic paralysis* is employed. Diminution of the muscle-tone is usually difficult to detect by passive movements alone. When it is entirely lost the limb is spoken of as *flail-like*. The joints seem to have no tendency to remain in one position. If the limb is shaken, with every movement they pass from extension to flexion, or *vice versa*. Under these circumstances the passive movements are entirely unresisted, the only effort necessary being that required to overcome the weight of the limb itself.

The Tendon Reflexes. These were first described by Westphal in connection with the reflexes of the knee. They consist essentially of a rapid twitch or succession of twitches in the muscle when the tendon by which it is attached to some bony part is struck a sharp blow. There is some difference of opinion regarding the true nature of the stimulus required to produce them. According to Gowers, it is a simple extension of the muscle, and he, therefore, uses the term *myotatic phenomenon*. Sternberg, on the other hand, believes that he has shown that they are the result of vibrations in the tendon, which are communicated by it to the muscle. Others contend that they are pure reflexes produced by the mechanical action of the blow upon the nerve-fibres in the tendon itself. It is certain, at any rate, that more factors are required than the mere tone of the muscle, and that afferent impulses to the spinal cord and efferent impulses from it are necessary to the development of the reflex; and that it is furthermore profoundly influenced by higher centres that usually have an inhibitory action (upper reflex arc). The question is complicated by the fact that in certain cases the reflexes may be elicited by tapping the bony parts, such

as the periosteal reflexes; by irritating the skin overlying the muscles, as the cutaneous reflexes; or by tapping upon the fascia or the belly of the muscle itself. In general, it may be said that all conditions producing increased muscular tone produce exaggeration of the reflexes, and that all conditions diminishing muscular tone diminish the reflexes. In marked contradiction to this, however, are the facts that attention to the reflex, being tested, will diminish or abolish it completely, whereas distraction of the attention, which ordinarily diminishes muscular tone, increases the force of the reflex. Moreover, in certain forms of profound coma, where the muscle tone appears to be at a minimum, the reflexes appear to be often greatly exaggerated. Thus, in uræmia and diabetic coma, I have been able on several occasions to detect exaggeration of the reflexes when the limbs were flail-like in their relaxation.

The individual reflexes of the head are practically limited to the *chin-jerk*. This is elicited by having the patient open his mouth slightly, then a flat object, such as a tongue depressor, or the handle of a spoon, is placed upon the teeth of the lower jaw and sharply tapped with the finger or hammer. Under normal circumstances there will be a slight upward jerk of the chin. It may also be elicited with less discomfort to the patient by placing the finger beneath the lower lip and upon the mental prominence and striking it sharply with the hammer. This does not always result in a reflex under normal conditions, but is quite satisfactory for the purpose of testing pathological exaggeration. The chin-jerk is nearly always increased in neurasthenia and hysteria, and is sometimes present in profound coma. In the conditions of general spasticity that are occasionally met with in severe infectious disease it is also usually exaggerated. Its absence does not appear to be of any pathological significance. Allied to the tendon or peritoneal reflexes is the phenomena known as *Chvostek's sign*. This occurs only in tetany, and consists of a sudden lightning-like twitching of the muscles of the face, particularly the elevators of the angles of the lip and the muscles of the eyelids. It is elicited by striking the skin below the zygomatic arch just in front of the ear with the hammer. It was formerly supposed that this was due to mechanical irritation of the trunk of the facial nerve, but the same phenomenon can also be elicited by striking over the malar bone or in the region of the infra-orbital foramen. No tendon reflexes have as yet been discovered for the muscles of the trunk.

In the arms the most important are the *bicipital*, *tricipital*, and the *supinator reflexes*. The bicipital reflex is best obtained by allowing the patient to rest the perfectly relaxed arm upon some support, for example, the arm of the investigator in a semi-flexed position. The finger or thumb is then placed upon the tendon of the biceps, and struck a sharp blow with the hammer or the finger, as in percussing. In nearly all normal cases a slight twitching or distinct contraction of the biceps can be obtained in this manner. Sometimes it is possible, by resting the arm upon a support, to see the tendon distinctly and to strike it directly, but this is usually much less satisfactory. The tricipital reflex is readily obtained by holding the arm semi-flexed and relaxed, and then striking just above the olecranon process of the ulna.

The supinator reflex is obtained by striking the radius just above the styloid process. These reflexes are particularly distinct in hemiplegia, upon the paralyzed side. They also occur in the general conditions above mentioned. Their absence is of no pathological significance, as it is often impossible to obtain them in normal individuals. In addition a reflex may be obtained by striking the bodies of the extensor muscles of the forearm, giving rise to extension of the fingers. A form of *wrist clonus* occasionally occurs that may be elicited by suddenly flexing the wrist-joint either dorsally or ventrally, and holding it in the cramped position. The *hypothenar reflex* is the contraction produced in the palmaris brevis by pressure upon the pisiform bone. It does not appear to be dependent upon any diseased condition. Tapping upon the bodies of the muscles sometimes gives rise to a sharp contraction. This is particularly observed in connection with the shoulder muscles (Strümpell) and pectoral muscles. An important reflex, the *abdominal reflex*, is elicited by drawing the end of a blunt object obliquely across the skin of the abdomen downward and outward or upward and inward, the object being to make it cross the line of the intercostal nerves as nearly as possible at a right angle. This produces contraction in the muscles innervated by these nerves, and is due to the stimulation of their cutaneous distribution. It may be exaggerated in functional nervous conditions, and is diminished in cases of hemiplegia and anæsthesia on the anæsthetic sides. Its absence at some particular point occasionally serves as an additional factor in the localization of lesions of the spinal cord. Various reflexes, probably periosteal or fascial in nature, may be produced by tapping upon the spinous processes of the ilium. As far as is known, they are not of any clinical value.

The reflexes of the lower extremities are the most important of all. The first discovered, the *knee-jerk*, is invariably present in health, and by its delicacy and constancy is the most valuable reflex for clinical purposes. It may be elicited in a variety of ways. Perhaps the best method is to have the patient lie upon his back; then placing one hand under the knee it should be lifted several inches from the surface of the bed or table until the leg and thigh form an obtuse angle of about 120°. Then with the finger, the side of the hand, the edge of the stethoscope, or the percussion hammer[1] it is struck a sharp blow. The patellar tendon should be struck between the lower edge of the patella and the tuberculum of the tibia. The stroke should be delivered with moderate force, and, according to the practice of most clinicians, a single blow is sufficient, but sometimes the reflex is more certainly elicited if several strokes are given in quick succession. The most obvious and vigorous contraction occurs in the quadriceps of the same side, causing the leg to be tipped upward suddenly and giving rise to the name knee-jerk. In addition, the adductors of the same side nearly always contract slightly, and occasionally the flexor muscles—

[1] There are various forms of these—one with a heavy metal head and short, wooden handle, the end of the metal head being covered with leather; another, composed of a wedge-shaped piece of rubber set in a light metal handle; the latter is probably the better.

that is, the biceps, the semi-tendinosus, and the semi-membranosus—also contract. Frequently the adductors of the opposite side contract very slightly in health, and sometimes quite vigorously in diseased conditions (*crossed-reflex*). Other methods of obtaining this reflex are to allow the patient to sit on a low chair with the leg extended forward, until it forms a blunt angle with the thigh, with the heel resting upon the ground. The patellar tendon is then struck as before. Clinically it is usually sufficient when the patient is sitting in an ordinary chair to have one leg thrown over the other, and hanging loosely and freely. Occasionally it is difficult, on account of extreme relaxation of the muscles, to stretch the tendon sufficiently to obtain the reflex by this method, and Gowers suggests that under these circumstances the legs should be completely flexed upon the thighs. It is often difficult to discover the tendon, either on account of deformity of the joint or because of an excess of fat tissue. In one case that I have observed, in which extensive arthropathies existed, the knee-jerk was present, but obtained with great difficulty, on account of the distortion of the parts. The patellar tendon reflex, therefore, is a multiple muscular reflex, producing phenomena of the opposite side, the so-called *bilateral reflex*. It is said to be invariably present in health, but its intensity varies considerably, and in some apparently healthy persons without any evidence of disease of the spinal cord it is extremely difficult to elicit. Under these circumstances it is necessary to use various procedures in order to make it evident. These consist either in requesting the patient to look at the ceiling, in order to distract the attention, or to perform some violent muscular effort, such as an attempt to pull the hands apart when they are clasped together, to squeeze the dynamometer, etc. Under these circumstances the knee-jerk, if obtained, is spoken of as *reinforced*. It is always important to have the muscles completely relaxed, and to prevail upon the patient not to think of what is being done. The knee-jerk is sometimes rendered more pronounced by emotion, and sometimes inhibited, as, for example, by fright. The arc of the knee-jerk is situated in the first lumbar segment of the cord, but probably occasionally deviates slightly from this position, being either higher or lower. The knee-jerk is, therefore, invariably increased in any disease of the pyramidal tracts above this point. It is diminished in disease of the efferent or afferent fibres. Its absence in tabes dorsalis was noted early, and has long been considered evidence of disease of the posterior columns. Closely allied to the knee-jerk in its clinical significance and mode of occurrence is the *patellar reflex*. This is elicited usually by placing the finger transversely above the patellar, pushing the bone forcibly down, and then striking the finger with the hammer. Ordinarily a distinct, pronounced contraction of the quadriceps alone is produced. In order to elicit this reflex the leg must be extended and relaxed. *Patellar clonus* occasionally occurs, and is obtained by placing the thumb and forefinger on the upper edge of the patella and pushing it forcibly downward and keeping it in that situation. If clonus occurs it will be characterized by a series of rapid contractions of the quadriceps, resulting in a vertical oscillation of the patella. It occurs in disease of the spinal cord, and

not infrequently in conditions of increased tonicity in general infectious diseases.[1]

In general it may be said that the mechanical effort is dependent upon the condition of the nutrition of the quadriceps and the amount of interference of the opposing muscles. Exaggeration of the knee-jerk is characterized by a more vigorous effort or more extensive contraction of the surrounding muscles. The latter, indeed, may, by the involvement of the flexors, diminish the excursion of the leg. Sometimes in cases of profound emaciation, as in cachexia, although the knee-jerk is increased and the muscle apparently contracts vigorously, its power is so greatly diminished that it is unable to move the leg. Elaborate mechanisms, therefore, that have been devised for measuring the knee-jerk do measure in fact only the amount of movement of the foot, and are practically worthless. They consist essentially of an arc of a circle whose radius is approximately equal to the length of the leg. Either a pencil or a small readily movable index is placed against the foot, and the knee-jerk is measured by the number of degrees marked off on the scale. It is manifest that comparisons are only valuable when the blow is of exactly the same force, and then only when the experiments are performed upon the same individual within a limited period of time. In order to obtain a constant force of blow various instruments have been devised, the simplest being weights dropped through a paper cylinder upon the patellar tendon, and the more complicated having springs for their motive power. Tendon reflexes may also be obtained by tapping upon the hamstring tendons. They are of no particular value. Tapping upon the inner condyle of the tibia often produces contraction of the adductor muscles, but this is not, as a rule, as pronounced as the contraction produced by the percussion upon the patellar tendon. Next in importance to the patellar reflex is the *Achillis tendon reflex*, which consists of the contraction of the gastrocnemius and soleus muscles when the Achillis tendon is struck. It is most readily elicited by lifting the entire leg from the bed or table, and holding it by the ball of the foot, which is gently pressed upward. The tendon is thus moderately stretched, and may be struck directly. In nearly all healthy individuals this reflex is present, but is absent in some, and its absence is apparently of no clinical significance. Exaggeration may be indicated in moderate cases by the more forcible extension of the foot. In more pronounced cases it gives rise to a peculiar and characteristic phenomenon, known as *ankle clonus*. This may be elicited by tapping the tendon once vigorously or several times in succession when the leg is held in the manner described, but is more readily produced by slightly flexing the leg and thigh, then grasping the ball of the foot firmly, flexing it dorsally with considerable force, and holding it in that position. When ankle clonus exists there will be violent vibratory oscillations of the foot, as long as the pressure upon the sole is continued, that vary from two to three up to five or

[1] Dr. Mills has devised an ingenious instrument, consisting of a metal ring with a curved handle, by which the patella may be drawn downward and the jerk or clonus more certainly elicited.

ten movements per second. There is usually a rhythmical increase and decrease in the rapidity, without absolute cessation at any time. Occasionally, in very mild cases, the clonus after a few movements becomes weaker, and rapidly disappears. Ankle clonus is supposed to indicate the existence of a lesion above the second lumbar segment of the spinal cord that seriously interferes with the function of the pyramidal tract. For a long time there has been doubt as to whether it occurs in functional disease, but it seems now to be established that it does. Its occurrence in functional conditions is, however, of such extreme rarity that when it is present organic disease should always be suspected. It is most characteristic in spastic paraplegia, either due to transverse myelitis, to lateral sclerosis, or to syringomyelia. It also occurs after lesions in the motor regions of the brain. It can sometimes be elicited by supporting the weight of the leg upon the toe. Under these circumstances it develops spontaneously in organic conditions, and sometimes in fatigue, cold, or exhaustion. It may also be produced in normal persons who continue for a sufficient length of time voluntary oscillatory movements of the foot supported in this manner. A *pseudo-ankle clonus* has been described as characterized by a few irregular oscillations that soon cease. It occurs in functional disease and occasionally among malingerers. Tapping upon the tendon of the great toe occasionally produces a slight contraction of that member. The other reflexes of the lower extremities are *front tap*, dorsal extension of the toes upon percussion of the anterior surface of the tibia, and the *toe reflex*—that is, slight flexion of the toes when the skin of the sole is irritated. This, according to Babinsky, is replaced by a dorsal flexion of the toes when the pyramidal columns are involved, and disappears in tabes dorsalis. The *plantar reflex* properly belongs to the group of cutaneous reflexes. It is characterized by the involuntary withdrawal of the foot when the sole is irritated. It is of course absent in cases of anæsthesia, and is greatly exaggerated in functional nervous conditions, occasionally giving rise to a peculiar general tremor of the leg or even of the whole body. It is best elicited by drawing a blunt object (pencil, handle of a stethoscope) across the surface of the foot.

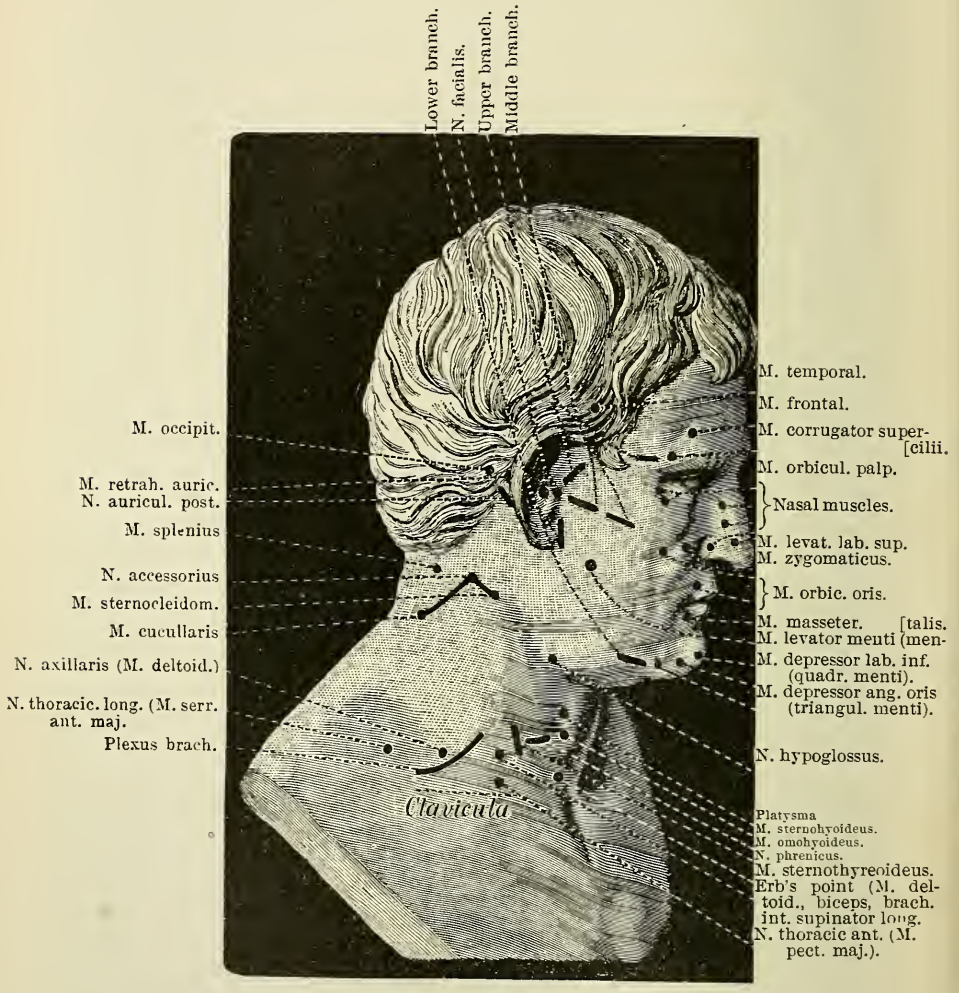
Allied to the reflexes is the so-called *paradoxical contraction* of Westphal. This consists in a persistent spasm of the muscle when its two attachments are suddenly brought closer together. It is most frequently observed in the peroneal muscles of the leg, and may be elicited by suddenly flexing the foot dorsally. It occurs most frequently in various functional conditions, and has also been observed in paralysis agitans.

Next to the functional conditions of the muscles, which is indicated by the degree of motility that they possess, we are interested in the state of their *nutrition*. It may be suspected that this is impaired when fibrillary contractions or atrophy are present.

Atrophy of the muscles may usually be detected by simple inspection. If only certain groups are involved, the latter will appear more or less distorted. It is always, however, important to measure the injured limb and compare it with the sound side if the affection is unilateral. When due to general conditions, such as the muscular dystrophies or

polyneuritis, it is sometimes more difficult to be certain of its existence. A general atrophy of the muscular system also occurs in cachectic states, such as the cachexia of carcinoma. *Fibrillary twitchings* occur in muscles undergoing degenerative changes. They are characterized

FIG. 226.



Motor points for the head and neck. (SAHLI.)

by the sudden, spasmodic contraction of individual fibres in the mass of the muscle itself, giving rise to a curious trembling of the overlying skin and a peculiar sensation to the palpating hand, as if minute waves were passing through the muscular substance. They often occur spontaneously, and in degenerating muscles may be elicited by slight mechan-

ical stimuli, such as cold, percussion, or shock. Fibrillary twitchings may also occur in healthy muscles that have either been chilled (tremor or shivering) or subjected to severe fatigue.

The most reliable method of diagnosis is by an *electrical examination.* For this purpose we use two types of apparatus. The galvanic current is produced by the galvanic battery, consisting of a number of cells, each containing an electro-positive and electro-negative element and filled with battery fluid. Long wires are attached to the battery, through which the current flows when they are brought in contact or the circuit closed, and ceases when they are kept apart or the circuit opened. The free end of the wire toward which the current flows from the cell is called the anode, and the free end from which the current passes to the cell, the cathode; then, if any substance is introduced between these ends of the wire, closing the circuit, the current passes through it from the anode or positive pole to the cathode or negative pole. It is customary to introduce into the circuit for measuring the amount of electricity employed a galvanometer, which is graduated in milliampères.[1] As it is important to employ a definite number of milliampères, the apparatus is also provided with a rheostat, which renders it possible by the introduction of a greater or less degree of resistance to regulate the amount of electricity passing through the body. The free ends of the wire are, for medical purposes, supplied with electrodes. These consist essentially of metal disks or plates to which the wire is attached, provided with a wooden or hard rubber non-conducting handle. As the resistance normally offered by the skin is greatly reduced if it be moistened, the ends of the electrodes are covered with cotton or gauze and moistened by immersion in either plain or salt water. The area of the cross-section of the electrode may vary considerably. Ordinarily, it is customary to have one very large electrode, from 50 to 100 square centimetres in area, and one exactly 3 square centimetres in area. (Stintzing's standard electrode.) In addition, for therapeutic purposes, it is customary to have for the galvanic and faradic apparatus a wire brush and various special electrodes for application to the more inaccessible portions of the body. If a muscle or nerve is to be investigated the large electrode is thoroughly moistened and placed over the back or the sternum. It is not advisable to place it over the neck nor to allow the patient to hold it in the hand. The current is so arranged that this large electrode is at first the anode and the small electrode the cathode. The cathode is now placed over the muscle or the nerve to be stimulated, locating it, if possible, exactly over the most sensitive (electrically) point. This is most readily determined by comparison with the figures on pages 990 *et seq.* The circuit should be open and the rheostat so placed that the minimum amount of current flows through the body. The circuit is

[1] One milliampère equals 0.001 of an ampère. The ampère is the unit adopted for the measure of the amount of current. It is determined by dividing the unit of electromotive force, one volt—that is, 0.9 of the amount of current liberated by a freshly filled Daniell cell, divided by 1 ohm—that is, the amount of current required to overcome a unit of standard resistance, or a column of mercury 1.06 metres in length and 1 square millimetre in cross-section.

now rapidly opened and closed, while the cathode is kept in position and the rheostat gradually moved around until the current is strong enough to produce a slight twitching of the muscle. This will first occur at the making of the circuit, and is spoken of as cathodal closing contraction, or CCC. The current should now be slightly increased, and by means of a switch the small electrode converted into the anode and the other into the cathode. It will soon be observed that a contraction takes place both at opening and closing the current. This is spoken of as the anodal closing contraction, or ACC, and the anodal opening contraction, or AOC. If the small electrode be again made

FIG. 227.

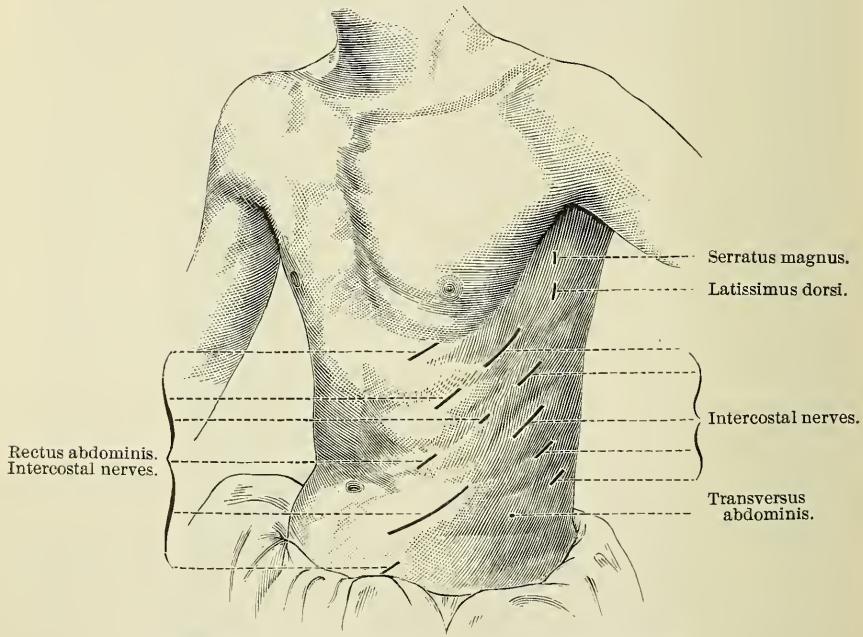


Diagram of the motor points of the trunk. (From VON ZIEMSSEN.)

the cathode, it will be found that there is a vigorous contraction when the current is closed, but none when it is opened. Finally, if the current is made still stronger, it will be found that the closure of the current produces at the cathode no longer a simple lightning-like contraction, but a prolonged cramp of the muscle, spoken of as cathodal closing tetanus, or CCTe. The contraction produced by both opening and closing the current at the anode is now much stronger than before, and there will probably appear a slight contraction at the opening of the cathode, the cathodal opening contraction, or COC—that is to say, with gradual, increasing current the order of contraction is as follows in a normal muscle: cathodal closing contraction, anodal closing contraction, anodal opening contraction, cathodal closing tetanus, cathodal

opening contraction. Under ordinary circumstances the healthy muscle contracts suddenly and relaxes almost immediately. Various modifications of these phenomena occur in diseased conditions, and there are considerable quantitative changes between the different muscles in health. Thus, in the muscles of the face contraction is always more

FIG. 228.

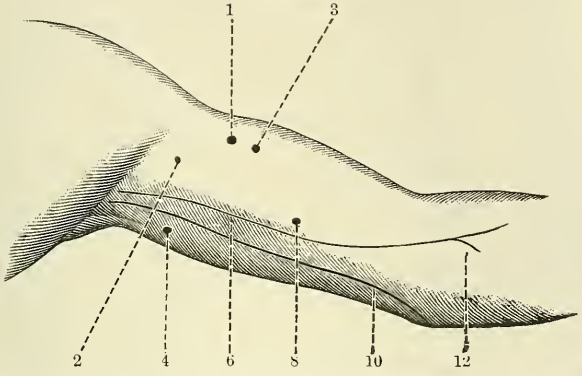
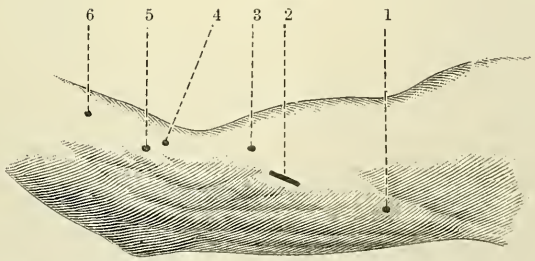


Diagram of the motor points of the arm, under side. (From VON ZIEMSSEN.)

1. Musculo-cutaneous nerve. 2. Musculo-cutaneous nerve. 3 Biceps. 4. Internal nerve of triceps. 6. Median nerve. 8. Brachialis anticus. 10. Ulnar nerve. 12. Branch of median nerve to the pronator teres.

rapid than in those of the thigh, and can be elicited with much weaker currents. In disease we recognize three types of alteration: First, quantitative changes; second, quantitative qualitative changes; third, pure qualitative changes. Before discussing these it is necessary to describe the *faradic apparatus*. This consists essentially of a coil of wire through which flows an electric current, that forms the core for a

FIG. 229.



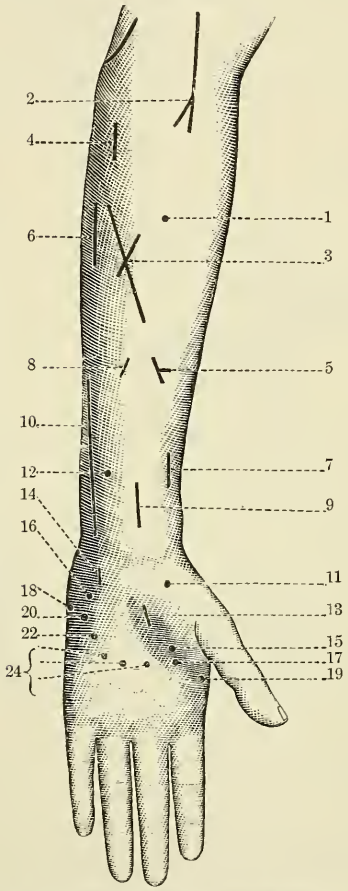
Motor points of the arm, outer side. (From VON ZIEMSSEN.)

1. External head of triceps. 2. Musculo-spiral nerve. 3. Brachialis anticus. 4. Supinator longus. 5. Extensor carpi radialis longior. 6. Extensor carpi radialis brevior.

second coil not attached to it. If, now, the current passing through the inner or primary coil is interrupted, there will be generated, at each opening of the current, a current in the outer or secondary coil, going in the opposite direction, and, at each closure, a current going in the same direction. This is usually the stronger, and, if the interruptions

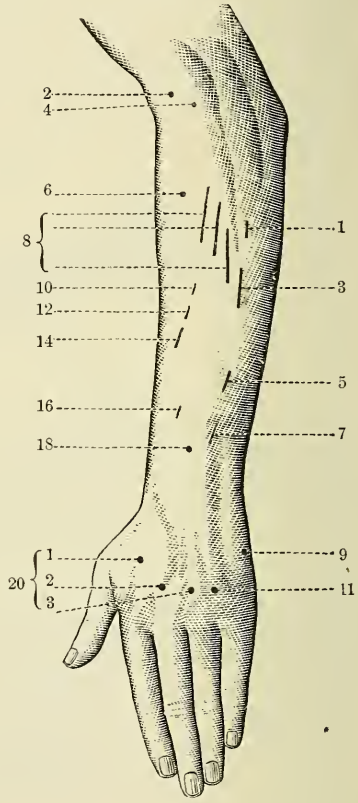
are sufficiently rapid, dominates the reversed current. The ends of the secondary coil are attached to the electrodes. The strength of the current is altered by moving the inner coil away from the secondary coil.

FIG. 230.



Motor points of forearm, inner surface.

FIG. 231.



Motor points of forearm, outer surface.

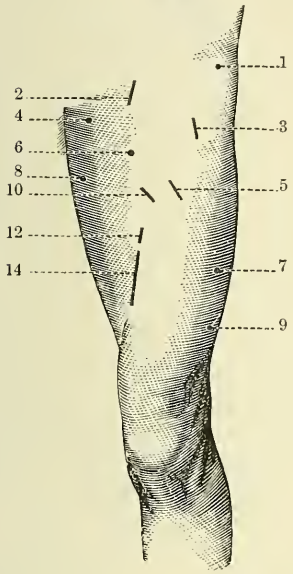
(From VON ZIEMSSEN.)

FIG. 230.—1. Flexor carpi radialis. 2. Branch of the median nerve for the pronator teres. 3. Flexor profundus digitorum. 4. Palmaris longus. 5. Flexor sublimis digitorum. 6. Flexor carpi ulnaris. 7. Flexor longus pollicis. 8. Flexor sublimis digitorum (middle and ring fingers). 9. Median nerve. 10. Ulnar nerve. 11. Abductor pollicis. 12. Flexor sublimis digitorum (index and little finger). 13. Opponens pollicis. 14. Deep branch of ulnar nerve. 15. Flexor brevis pollicis. 16. Palmaris brevis. 17. Adductor pollicis. 18. Adductor minimi digiti. 19. Lumbricalis (first). 20. Flexor brevis minimi digiti. 22. Opponens minimi digiti. 24. Lumbricales (second, third, and fourth).

FIG. 231.—1. Extensor carpi ulnaris. 2. Supinator longus. 3. Extensor minimi digiti. 4. Extensor carpi radialis longior. 5. Extensor indicis. 6. Extensor carpi radialis brevior. 7. Extensor secundi internodii pollicis. 8. Extensor communis digitorum. 9. Abductor minimi digiti. 10. Extensor indicis. 11. Dorsal interosseus (fourth). 12. Extensor indicis and extensor ossis metacarpi pollicis. 14. Extensor ossis metacarpi pollicis. 16. Extensor primi internodii pollicis. 18. Flexor longus pollicis. 20. Dorsal interossei.

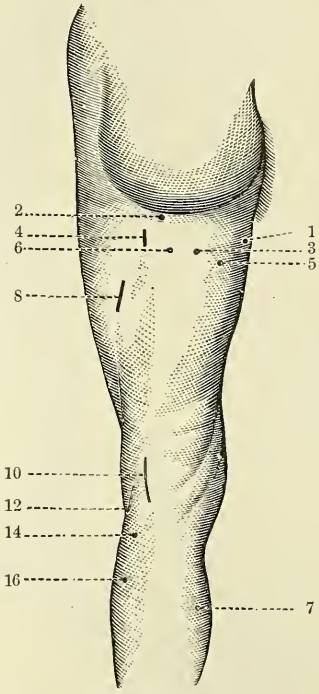
This is spoken of as the distance between the coils, and is measured in inches or centimetres. It is manifest that this method for measuring is not absolute, but its value must be determined for each particular machine. This can only be done by the physiological test—that is, measuring the force required to produce contractions in some muscles and then comparing it with the known value for this muscle obtained by a standard machine, and obtaining in this way the ratio. The current

Fig. 232.



Motor points of thigh, anterior surface.

Fig. 233.



Posterior surface.

(From Von Ziemssen.)

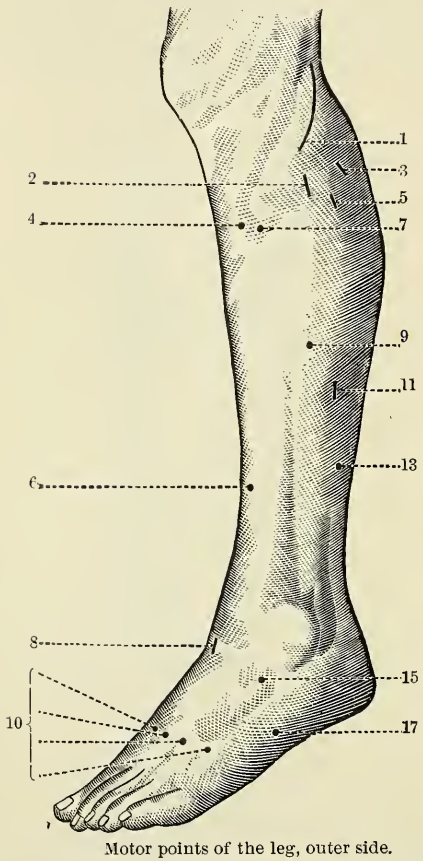
Fig. 232.—1. Tensor vaginæ femoris (branch of superior gluteal nerve). 2. Anterior crural nerve. 3. Tensor vaginæ femoris (branch of crural nerve). 4. Obturator nerve. 5. Rectus femoris. 6. Sartorius. 7. Vastus externus. 8. Adductor longus. 9. Vastus externus. 10. Branch of crural nerve to quadriceps extensor cruris. 12. Crureus. 14. Branch of crural nerve to vastus externus.

Fig. 233.—1. Adductor magnus. 2. Inferior gluteal nerve for gluteus maximus. 3. Semi-tendinosus. 4. Great sciatic nerve. 5. Semi-membranosus. 6. Long head of biceps. 7. Gastrocnemius (internal head). 8. Short head of biceps. 10. Posterior tibial nerve. 12. Peroneal nerve. 14. Gastrocnemius (external head). 16. Soleus.

is, of course, increased when the secondary coil is directly over the primary one and diminished when the primary coil is withdrawn. As the current in the secondary coil is oscillatory—that is, going first in one direction and then in the other—it is not theoretically possible to speak of an anode and a cathode. Practically, however, the current going in the same direction as that of the primary coil is the stronger, and a difference does exist between the two ends of the wire, which are

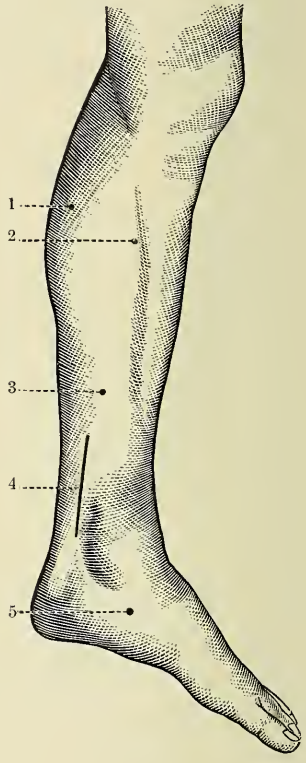
usually spoken of, therefore, as cathode and anode. A contraction produced by the faradic stream is always tetanic in health, as there are a series of stimulations constantly passing through the muscle.

Fig. 234.



Motor points of the leg, outer side.

Fig. 235.



Inner side.

(From Von Ziemssen.)

Fig. 234.—1. Peroneal nerve. 2. Peroneus longus. 3. Gastrocnemius (external head). 4. Tibialis anticus. 5. Solens. 6. Extensor longus pollicis. 7. Extensor communis digitorum longus. 8. Branch of peroneal nerve for extensor brevis digitorum. 9. Peroneal brevis. 10. Dorsal interossei. 11. Soleus. 13. Flexor longus pollicis. 15. Extensor brevis digitorum. 17. Abductor minimi digiti.

Fig. 235.—1. Gastrocnemius (internal head). 2. Soleus. 3. Flexor communis digitorum longus. 4. Posterior tibial nerve. 5. Abductor pollicis.

Alterations in the Reactions of the Muscles and Nerves to Electricity. Reactions of Degeneration. *Quantitative alterations* consist in increase or decrease of the susceptibility of the muscles or nerves to electrical action. They may be determined in case the lesion is unilateral by comparison with the normal side of the body; in case the lesion is bilateral, only by comparison with a standard table, such as has been furnished by Stintzing. If the deviation

from the normal is slight, the error has very likely been produced by variation or alteration in the resistance of the skin. Quantitative increase in the electrical reaction occurs chiefly in tetany, for which disease it is almost pathognomonic, and has been spoken of as Erb's sign. It occurs also occasionally in the early stages of hemiplegia, in paralysis of the facial nerve, and has been noted in certain cases of tabes dorsalis. Diminished electrical irritability occurs in all the forms of idiopathic muscular dystrophy. It also occurs in those forms of atrophy due to lesion of the central motor neuron without involvement of the peripheral motor neuron. It also occurs in those atrophies secondary to disease of the joints and loss of functional activity on the part of the muscle. Diminished reaction may occur in hysteria and profound neurasthenia, and has been observed in some cases of locomotor ataxia, and even in some cases of progressive spinal muscular atrophy of exceedingly slow course. It also occurs in certain nervous diseases whose nature is not yet understood, as in Goldflam's periodic paralysis, although it is to be noted that there are other alterations in the electrical reactions in this disease. The *quantitative qualitative reaction* consists, first, of a diminution of the reaction of the muscle or the nerve to the faradic current, and its diminution or exaggeration to the galvanic current, with distinct alteration of the order in which the various forms of galvanic irritation produce contractions. Cohn discriminates three types of this form of degeneration: First, the complete reaction, mild in character, and terminating in recovery; second, the complete reaction, severe and incurable; and, third, a partial reaction. He gives the following table illustrating the various stages of these three forms:

TOTAL REACTION OF DEGENERATION.

Moderate Form.

	Indirect stimulation (nerve).		Direct stimulation (muscle).[1]	
	F.	G.	F.	G.
1st stage, 1–8 days.	Diminished.	Diminished.	Diminished.	Diminished.
2d stage, 2–15 weeks.	Lost.	Lost.	Lost.	Increased, contraction slow. AOC>CCC.
3d stage, 6–30 weeks.	Returning.	Returning.	Returning.	Diminishing contraction more rapid. AOC = or > CCC.
4th stage, later.	Subnormal.	Subnormal.	Subnormal.	Subnormal, no qualitative changes
Progressive Incurable Form.				
1st and 2d stages.	As first and second stages above.			
3d stage, after 6 weeks.	Lost.	Lost.	Lost.	Diminished or lost. AOC>CCC.

[1] By direct stimulation is meant the application of the electrode to the muscle itself. By indirect stimulation is meant the application of the electrode to the motor nerve-trunk. The latter term is employed because irritation of the nerve can only be detected by the activity of the muscle, and the stimulation of the latter is, of course, in this mode of application, indirect.

Partial Reaction of Degeneration.

	Indirect stimulation (uerve).		Direct stimulation (muscle).	
	F.	G.	F.	G.
1st stage, 1–8 days.	Normal or diminished.	Normal or diminished.	Normal or diminished.	Normal or diminished.
2d stage, 2–5 weeks.	Normal or diminished.	Normal or diminished.	Normal or diminished.	Increased, contraction slow. AOC<CCC.
3d stage. 6–12 weeks.	All normal or progressive form.			
3d stage, 6 weeks.	Diminished or lost.	Diminished or lost.	Diminished or lost.	Diminished or lost. Contraction still slow. AOC<CCC.

The following points in these tables need explanation. The faradic reaction is similar to that which occurs in the normal muscle, but requires a much stronger current to produce it. The galvanic reaction of the nerve is similar to that obtained under normal conditions, excepting that a stronger current is required. The contraction is lightning-like and disappears instantly. The direct galvanic stimulation of the muscle, however, produces a worm-like contraction very different from that observed in the normal muscle, and is ascribed to the direct stimulation of the muscle itself and not to the stimulation of thet erminations of the motor nerves. This often occurs with a much weaker current than is normally required to produce contraction in the muscle. It will also be observed that the cathodal closing contraction is no longer the first to appear, but it is replaced by the anodal opening contraction, and this is followed by the anodal closing contraction, cathodal closing contraction occurring only with relatively strong currents. If regeneration occurs muscular contractions occur in response to weaker faradic currents, and by direct galvanic stimulation they become more lightning-like in character. Gradually the cathodal closing contraction appears in response to weaker currents, and finally occurs before the anodal opening contraction. If recovery does not take place, direct galvanic stimulation requires stronger and stronger currents, and there is no increase in the rapidity of the contraction. The cathodal closing contraction disappears, and finally only the anodal contraction remains, which is exceedingly slow and worm-like. When the muscle-tissue has been completely replaced by connective tissue all reactions naturally cease. The partial reaction of degeneration is very similar to the mild, complete form. Recovery, however, occurs, as a rule, very rapidly. The reaction of degeneration may be used for determining the prognosis of the case. When after the sixth week the muscle does not respond as readily as before to direct galvanic stimulation, and the cathodal closing contraction becomes equal to or greater than the anodal opening contraction, the prognosis is exceedingly favorable. Particularly the increased rapidity of the contraction is of great significance. If, on the other hand, after from six to twelve weeks no change has occurred and the anodal still precedes cathodal contraction, and both are worm-like in character, the prognosis is doubtful. Months, however, may elapse before the muscle gradually begins to regain its normal

character. The voluntary contractions of the muscle, as a rule, persist after the reaction of degeneration has become manifest, unless, of course, there has been total destruction of the peripheral motor neurons. Often in cases of peripheral neuritis it will be observed that the reaction of degeneration is present in muscles that are apparently healthy, but which, when the process is progressive, subsequently atrophy. When regeneration occurs voluntary motion will have been almost completely restored long before the muscle has become entirely normal, and it may often reappear before any improvement can be detected in the electrical reactions. In testing these reactions the following points are to be noted: First, the reaction of the nerve to faradic and galvanic electricity; second, the reaction of the muscle itself. It is particularly important to be certain that only the muscle under investigation is affected by the electrical current. Sometimes it will be impossible to accomplish this, but ordinarily it can be done sufficiently well to enable us to secure positive results. It must be remembered, however, that the reactions of degeneration often occur in the muscles of limbs that have been injured, or are found in limbs in which some of the groups of muscles have already undergone atrophy, and thus altered the anatomical relations. Under these circumstances mistakes are very likely to arise. Sometimes valuable information can be obtained by stimulating a nerve-trunk and observing whether all the muscles innervated show normal or impaired contractility. Quantitative and qualitative reactions of degeneration occur primarily as a result of disease of the peripheral motor neuron. They are, therefore, found in all diseases of the spinal cord that affect the anterior cornua or the motor roots, and in all diseases of the medulla that affect the motor nuclei or their roots; therefore, in acute and chronic antero-poliomyelitis, progressive spinal muscular atrophy, in bulbar palsy, in transverse myelitis, syringomyelia, tumor of the cord, and as a result of chronic forms of meningitis, or disease of the vertebral columns pressing upon the roots. They are also found in all forms of peripheral neuritis, either the toxic, the infectious, or the traumatic, and in all cases of solution of continuity of the nerves. They occasionally occur in the so-called idiopathic muscular dystrophies, but in these they are exceptional. They are also found in a few cases after cerebral lesions.

Atypical Types of the Reaction of Degeneration. Only two of these are important. First, the *myotonic reaction*, consists of the persistence of the muscular contraction after the electric stimulus has been removed. This occurs either with the faradic or the galvanic current, but the order of contraction to the various forms of stimulation of the latter is not altered. This reaction is pathognomonic of Thomsen's disease—myotonia congenita. It is more likely to occur as a result of stimulation of the muscle itself than of stimulation of the nerve. Second, the *myesthenic reaction* is characterized by the rapid exhaustion of the muscle or the nerve, so that relaxation may take place while the faradic current is still being employed, and if the muscle is stimulated successively several times, it loses its power to contract or requires a stronger current. It occurs in periodic family paralysis. Remak and Marino have described a peculiar form of reaction which they name the *neuro-*

tonic reaction. It consists of the persistence of the contraction only after stimulation of the nerve.

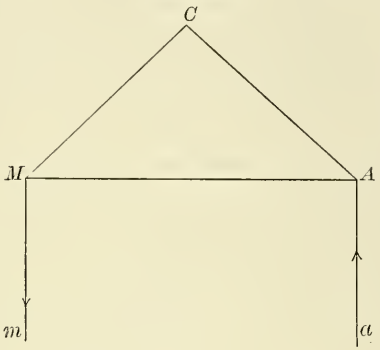
Disturbances of Speech. These may be divided into two groups: *aphasia*, the disturbance of the central nervous mechanism controlling speech, writing, and mimicry; and *anarthria*, the disturbance of the peripheral motor mechanism of speech.

By aphasia is meant the loss or impairment of the ability to understand spoken, written, or mimic language, and to express thoughts by the same means. It is ordinarily divided into two forms: *motor aphasia*, or the inability to innervate the motor apparatus for speech, while the sensory or perceptive functions are intact; and *sensory aphasia*, or the inability to recall or understand words, although the ability to produce sound is preserved. A variety of other forms, however, have in the course of time come to be recognized. Oppenheim recognizes the following five varieties: (1) *Motor aphasia.* This consists of the loss of power to speak, with persistence of the understanding of spoken, written, and mimic speech. This is the first form of aphasia in which it was possible to locate with accuracy the portion of the brain involved. The lesion is cortical or subcortical, and involves the foot of the third frontal convolution on the left side. The symptoms are variable according to the extent and destructiveness of the lesion. (2) *Sensory aphasia.* The perception of sound as such is preserved, but there is inability to recognize the significance of words, although spontaneous and occasionally voluntary speech is preserved. The lesion is usually found in the auditory centre—that is, the first temporal convolution on the left side. The symptoms may be variable, alexia being often present. (3) Pure *alexia*, or word blindness. In this, although sight is preserved and objects may be recognized, the ability to understand written or printed language is lost. Spoken speech is still understood, voluntary speech and writing possible, and occasionally written words may be read if the patient is permitted to trace the letters with a pencil or the finger, recognizing each one as it is formed. The lesion is usually found in the left occipital lobe on the external surface, but sometimes involves the gyrus angularis. (4) Pure *agraphia*, or the loss of power to write, all the other qualities remaining normal. Lesions have been found in the left upper parietal lobe. (5) *Optic aphasia.* In this objects may be seen and recognized, but it is impossible for the patient to find the proper name for them. If the objects are recognized by some other sense, as, for example, hearing or touch, the name may be recollected instantly. The lesion is usually found at the junction of the first temporo-sphenoidal and the occipital lobes. This form is frequently a symptom in otitic abscess. Loss of the stereognostic sense may also be regarded in some respects as an aphatic manifestation.

In order to explain aphasia, it has been customary, since the time of Wernicke, to employ the diagram given in Fig. 236. In this the triangle, *A C M*, represents the intra-cerebral paths and centres for the mechanism of speech, and the lines *Aa* and *Mm* the peripheral apparatus. In this diagram *A* represents the centre for auditory perception; *M* the centre for the emission of motor impulses; and *C* the concept centre, in which the intellect analyzes the impressions received

and from which the directing influence for the choice of language is transmitted to the motor centre. *Aa* represents the auditory nerve; *Mm*, the motor nerves to the pharynx, tongue, and lips. Auditory impressions may, therefore, be transmitted along *Aa* to *A*, thence

FIG. 236.



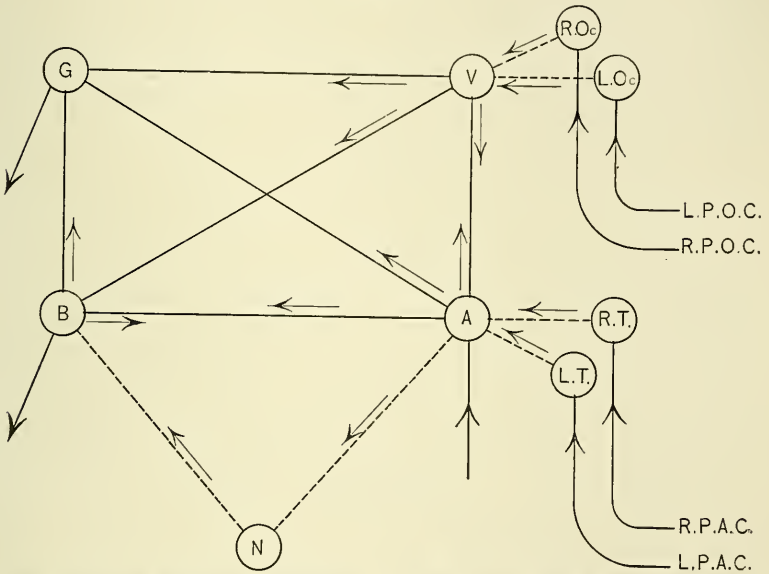
directly to *M*, and thence to the larynx. This is the mechanism supposed to be involved in ordinary mechanical speech—that is to say, the mechanical repetition of spoken words. The auditory impressions may, however, pass from *A* to *C*, there be analyzed or understood, and then transmitted to *M*, either in the same or altered form. This constitutes the intelligent repetition of spoken speech. If the alteration of form is considerable, or if, without immediate auditory impressions, impulses are transmitted from *C* to *M*, voluntary or intelligent speech is said to occur. Although this diagram probably does not accurately represent the conditions existing in the brain, it has been found that the varieties of aphasia that can be theoretically deduced from it correspond more or less closely to those that may be recognized in actual practice. These varieties are as follows: Destruction of the motor centre, *M*, gives rise to the so-called cortical motor aphasia with the following symptoms: Loss of (1) voluntary speech; (2) repetition; (3) reading aloud; (4) voluntary writing; (5) writing from dictation. There are preserved (1) the understanding of speech; (2) the understanding of writing; (3) the ability to copy writing. Destruction of the auditory centre, *A*, gives rise to cortical sensory aphasia. There are lost (1) the understanding of speech; (2) the understanding of writing; (3) the ability to repeat speech; (4) the ability to write from dictation; (5) the ability to read aloud. There are preserved (1) voluntary speech; (2) voluntary writing; (3) the ability to copy writing. A lesion in *C* would give rise to cortical apperceptive aphasia. The symptoms of this form would differ very slightly from those due to interruption of the tracts supplying it. The centre is probably complex and its parts are widely distributed. The speech disturbances of general paresis are possibly due to its partial destruction. Lesions of the various tracts of fibres connecting the different centres with each other or with the periphery also produce symptoms. Lesions between *A* and *M* produce the symptom known as *paraphasia*. (1) Voluntary

speech; (2) repetition of speech; (3) the understanding of spoken and written language; and (4) the ability to copy writing are all preserved. The only symptom, therefore, of this condition is the misuse or false pronunciation of words. Thus, objects may be misnamed, one word used in place of another, different syllables of the words misplaced (literal paraphasia), or the words jumbled in a sentence (verbal paraphasia). There is usually also *paragraphia*—that is, a similar disturbance of written language; *paralexia*, manifest when the patient attempts to read aloud, and sometimes the symptom known as *agrammatism*—that is, the misuse of cases, moods, or tenses. Paraphasia, however, occurs also in certain general diseases of the brain, and is practically always present if the intrinsic tracts concerned in speech are disturbed. Interruption of the tract uniting *C* and *M* causes transcortical motor aphasia. There are lost (1) voluntary speech and (2) voluntary writing. There are preserved (1) the understanding of speech; (2) the understanding of writing; (3) the ability to copy; (4) the ability to repeat words; (5) the ability to write from dictation; (6) the ability to read aloud. The most characteristic symptom is the inability of the patient to remember words, although he is able to repeat them fluently. The interruption between *A* and *C* gives rise to transcortical sensory aphasia. There are lost (1) the understanding of speech; (2) the understanding of writing. There are preserved (1) voluntary speech; (2) voluntary writing; (3) the repetition of speech; (4) reading aloud; (5) writing from dictation. Both voluntary speech and writing are usually affected by the paraphasia common to the interruption of the intrinsic tracts. It differs from the preceding form particularly in the fact that words spoken upon repetition or written from dictation are not in the least understood by the patient. In this form communication with the patient, even by gestures, is often impossible. Finally, lesions may occur in the tracts uniting the centres concerned in speech with the periphery. Lesions in the tract *Mm* give rise to subcortical motor aphasia. There are lost (1) voluntary speech; (2) repetition of speech; (3) the ability to read aloud. There are preserved (1) the understanding of speech; (2) the understanding of writing; (3) the ability to copy; (4) voluntary writing; and (6) writing from dictation. This is, of course, the purest form of motor aphasia. Interruption of the tract *Aa* gives rise to subcortical sensory aphasia. There are lost (1) understanding of speech; (2) the repetition of speech; (3) the ability to write from dictation. There are preserved (1) voluntary speech; (2) voluntary writing; (3) understanding of writing; (4) reading aloud; and (5) copying.

This theoretical classification with groupings of symptoms is susceptible to modification in actual pathology by a variety of conditions. The most important modification is that produced by the existence of possible lesions of other centres concerned in speech. Thus, the share taken by the visual receptive and apperceptive centres is of great importance in all persons who have been taught to read. They are necessarily concerned also in the production of writing. It is not, however, possible to represent them by a diagram as we have represented auditory and motor speech, for it appears that impulses from

the visual centres must pass through the receptive centre for speech, or *A*, before being transferred to the arm centre or the speech centres. The same is true for tactile impressions. These are of importance chiefly in blind persons who have been taught to read with their fingers, in whom, indeed, they may equal in importance the rôle of the visual centres in normal persons. Various complicated diagrams have been devised for the purpose of exhibiting the influence of all these centres upon speech, and Mills has introduced an additional naming centre, situated in the third temporal convolution, in which

FIG. 237.



A, auditory centre (centre for word-hearing); V, visual centre (centre for word-seeing); N, naming centre (centre where percepts are given in name); B, motor speech centre in Broca's convolution (regarded by Broadbent as a propositionizing centre); an utterance centre—motor centre—is also required to complete the motor side of the speech process, if the view is accepted; G, graphic centre; R. Oc., primary cortical visual centre in the right occipital lobe; L. Oc., primary cortical visual centre in the left occipital lobe; R. P. O. C., optic centres at the base of the brain, right side; L. P. O. C., optic centres at the base of the brain, left side; R T., primary cortical auditory centres in the right temporal lobe; L. T., primary cortical auditory centres in the left temporal lobe; R. P. A. C., auditory centres at the base of the brain, right side; L P. A. C., auditory centres at the base of the brain, left side.

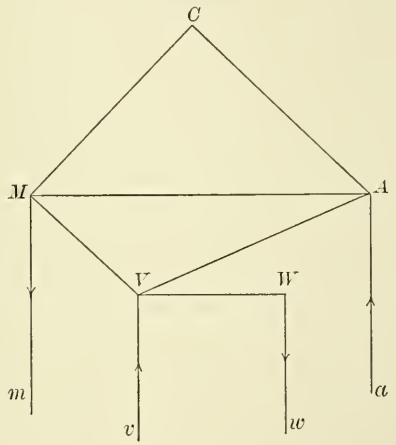
perceptions are given the names that properly belong to them. His diagram is one of the most satisfactory of all the more complicated diagrams representing the speech function (see Fig. 237), but, unfortunately, it is not yet possible to deduce from it theoretically the symptoms that actually occur. Another source of error is the fact that lesions may be only partially destructive, or may be so large as to involve two or more tracts or centres at the same time. Under these circumstances the symptoms become very complex, and it is often impossible to determine the extent of the physiological disturbance that has been produced. Usually, however, the localization of these lesions is not diffi-

cult, on account of the predominance of certain characteristic localizing symptoms.

It will be obvious from this description that it is necessary in each case of aphasia to test a variety of functions. These can best be examined as follows: 1. Voluntary speech. If the patient is able to answer questions intelligently or makes spontaneous intelligent remarks to the physician, voluntary speech is preserved. Voluntary speech may, however, exist and the remarks of the patient be nevertheless unintelligible when there is an extreme degree of paraphasia. 2. The ability to repeat words. This may be tested by merely saying a word or several words and getting the patient to repeat them. Mechanical speech, whose centre is supposed to be located in the speech area of the right hemisphere, may also be tested by requesting the patient to repeat some well-known series—such, for example, as the names of the days of the week, the alphabet, the numbers, or the months. Sometimes familiar songs may be remembered and spoken when it is absolutely impossible for the patient to make an intelligent answer. Under striking emotional conditions epithets or oaths may also be employed. The ability to repeat words may sometimes be present when it is impossible to determine it on account of the existence of transcortical sensory aphasia. Under these circumstances it is impossible to make the patient understand what he is expected to do. 3. Reading aloud. It must not be forgotten that in some cases this symptom is masked by defects of vision. If possible, the eyes should always be examined and the patient be given his glasses if he has been in the habit of using them. It is advantageous to use large type, such as the headlines of newspapers. 4. Voluntary writing. This symptom may be masked by the existence of right hemiplegia and inability to write with the left hand. 5. Writing from dictation. As in the repetition of speech, this symptom may be masked by the inability of the patient to understand what he is expected to do. 6. Copying. Errors of vision should again be excluded as well as paralysis and other motor disturbances of the arm. 7. The understanding of speech. This is perhaps one of the most difficult of all aphatic symptoms to determine. The patient is usually requested to perform some simple action, such as putting out the tongue, touching the ear with the hand, etc. This may be perfectly performed, but more complex commands or long statements may not be understood. It is supposed that this is perhaps due to incompleteness of the lesion, or to a general disturbance of intellect, such as must occur in any case of aphasia, in a more or less pronounced degree. It is, therefore, important to attempt if possible to converse with the patient, getting him to reply by gestures, or writing, according to his ability, and gradually to employ more and more complex statements. In cases of marked paraphasia the improper use of words in the replies may lead to the belief that the patient does not understand what is said to him, when, as a matter of fact, word perception is perfect. 8. Understanding of writing. This is subject to the same errors as the understanding of speech, and, in addition, the possibility of visual defect. 9. The existence of paraphasia. This, of course, can only be detected when either voluntary speech or the ability

to repeat words is present. Under these circumstances it may be recognized when it is only slight in degree by getting the patient to repeat words of many syllables, such as "incomprehensibility," or sentences of several words. Disturbances of writing, apart from disturbances of speech, may also occur. These may be better understood by a consideration of Fig. 238, in which the writing centres are added to the speech centres. It will be seen from this that there may be destruction of *W*, or *agraphia*. There are lost (1) voluntary writing; (2) copying; and there is preserved the ability to read. Destruction of *V*, or *cortical alexia*. This is characterized by the loss of (1) the recognition of written words; (2) voluntary writing. Speech may be intact. Destruction of *W V*, or *conduction agraphia*. There is lost (1) voluntary writing; (2) voluntary copying. There is preserved ability to read—that is to say, it corresponds exactly to the preceding form. Under such circumstances paragraphia may exist in this type. *Transcortical agraphia.*

FIG. 238.



There is lost voluntary writing. There is preserved (1) mechanical copying; and (2) reading. *Transcortical alexia.* There is lost the ability to read. There is preserved (1) voluntary writing; (2) copying. Finally, there may be interruption of the tracts to the periphery, giving rise to *subcortical agraphia.* There are lost (1) voluntary writing; (2) copying. There is preserved reading. Paragraphia never occurs in this form. *Subcortical alexia.* There are lost reading and copying. There is preserved voluntary writing. All of these forms may coexist with the various types of aphasia. In testing the patient for alexia the following symptoms should be examined: (1) Voluntary writing (see above); (2) writing from dictation (see above); (3) copying; and (4) the recognition of letters either spoken or written. In testing patients for voluntary writing with the left hand, it must be remembered that many aphasics give *mirror writing.* The following terms are also used in connection with aphasia: *aphrasia*, the inability

to form sentences with words; *dysphasia*, the imperfect formation of sentences; *apraxia*, the total loss of speech.

By ANARTHRIA is meant a disturbance in the peripheral motor mechanism of speech, as a result of disease of the nuclei in the medulla or of the peripheral nerves arising from them. This may vary in degree from complete aphonia, or loss of power to make sounds and words, which occurs in bulbar paralysis, or the aphonia of laryngeal paralysis, in which whispering speech is still preserved, to merely the imperfect pronunciation of certain consonants, as a result of local paralysis or paresis of the lips or tongue. Anarthria may be permanent or temporary, or, in cases of slight paresis, recurrent, giving rise to intermittent claudication of speech. It is best tested by directing the patient to repeat letters of the alphabet, to count, or to repeat words with long syllables and difficult consonants, as "artillery," "extraordinarily," etc. Allied to anarthria, but perhaps the result of certain functional disturbance, are *stuttering* and *stammering*. In the former, if the patient attempts to speak, there is inhibition of motion for a longer or shorter interval, and then the word may be pronounced with explosive violence, and the following words of the sentence spoken normally. In *stammering* there is frequently repetition of the first two or three consonants of the word, particularly if these happen to be labials. Stuttering and stammering are sometimes associated with defective intelligence. Finally, there are a series of disturbances of speech in which intellectual derangement is apparently the chief factor. These may perhaps be forms of aphasia due to partial destruction of the concept centre or centres. Among them may be mentioned the inability or unwillingness to speak, that occurs in the mutism of the insane; a tendency to excessive speech, *logorrhœa;* the omission of syllables, particularly characteristic of general paresis; difficult words, such as those mentioned above, being pronounced imperfectly, as "arlry" for "artillery," or even less accurately. *Scanning* speech, in which the words are separated by considerable intervals, and are spoken with a peculiar drawl and a descending cadence. It is particularly characteristic of multiple sclerosis, but may occasionally occur in general paresis. Other forms are: *explosive*, or *staccato* speech, and a peculiar, slow, drawling utterance, occasionally termed *bradylalia*, that occurs in certain states of mental depression. *Echolalia* occurs almost exclusively in imbeciles, and is characterized by the repetition of all sounds heard.

DISORDERS OF NUTRITION, or TROPHIC CHANGES, are lesions produced in tissues as a result of defective or altered innervation. They may be classified clinically into superficial trophic changes affecting the skin and its appendages, etc., and deep trophic changes affecting the muscles and joints. Among the superficial trophic changes of the mild form may be included vasomotor disturbances. In a strict sense flushing and the dead finger of Raynaud's disease are trophic alterations, but it is not certain what parts of the central nervous system are involved in order to bring them about. More severe are the various eruptive disorders that occur, particularly a herpetic eruption along the course of the nerve (*herpes zoster*). This occurs chiefly along the intercostal nerves, but may also occur along the other nerves of the

body, such as those of the face. It is characterized by the appearance of numerous vesicles surrounded by a congested zone and limited strictly to the distribution of the nerve or nerves involved. It occurs in neuralgias, in chronic neuritis, and in some cases as a result of an injury to the ganglion of the posterior spinal root. Among the milder trophic disturbances are the graying or falling out of the hair in the distribution of some particular nerve and the alterations in the nails. The latter are characterized by an increased brittleness, the formation of longitudinal ridges, and an excessive slowness of growth, which may be best detected by staining the nail at its root with nitric acid and comparing the amount of growth with that observed in a normal nail. These trophic disturbances in the nail occur in general cachectic states, but they are usually slight. They are more pronounced in lesions of the peripheral nerves supplying the fingers and toes, and also occur in destructive lesions of the spinal cord in the lumbar or cervical enlargement, such as syringomyelia and pachymeningitis cervicalis hypertrophica. More severe lesions are those due to the combination of defective resistance and secondary infection. These are chiefly the forms of panaritis observed in syringomyelia and characterized by the formation of an abscess at the root of the nail, which breaks down, leaving a chronic ulcer that heals very slowly, usually with the loss of the nail. In leprosy, in either the nodular or neural forms, and in Morvan's disease, somewhat similar changes also occur. Atrophy of the subcutaneous tissue with loss of elasticity of the skin is also a characteristic form of trophic disturbance. The part is shrunken, the finger-tips become pointed, the skin is dry and glossy or glazed, and the cutaneous bloodvessels, especially the veins, are distended. This occurs in destructive lesions of the peripheral nerves, and particularly in myelitis or destructive lesions of the spinal cord. An analogous change sometimes occurs in the teeth. These either become carious very rapidly and are destroyed, or become loosened in their sockets and fall out painlessly. The latter symptom is characteristic of the early stage of tabes dorsalis. There is also a tendency to the formation of chronic ulcers in the affected parts as a result of trifling injuries. Finally, severe lesions of the central nervous system may give rise to gangrene. This is characterized by the rapid destruction of the skin and underlying parts in regions subjected to the most trifling injuries, such as pressure. The part first becomes red, then a slight abrasion is formed upon the surface, followed by ulceration and the conversion of the surrounding tissue into a gangrenous mass, black and offensive. The usual situation is upon the back, just over the sacrum or to either side of it. It is called bed-sore, or *decubitus*. Bed-sores may also appear upon the hips, the knees, the heels, the shoulders, or, in fact, almost any part of the body. They are ordinarily the result of myelitis, in which they progress rapidly, and are more extensive than in any other condition. They may also occur, however, in cases of profound cachexia or exhaustion, and as a result of prolonged unconsciousness and of lack of attention in mental disease. Gangrene of the skin may also occur in hysteria. The mechanism of this is not clearly understood, but it is supposed to be due to vasomotor disturbances. Other severe cutaneous

lesions are the deep ulcerations that occur in various parts of the body, particularly the feet (*mal perforante*). These have been noted in tabes dorsalis, in syringomyelia, and also in hysteria. Finally, destructive lesions of the extremities with loss of the fingers may occur in Raynaud's disease, in syringomyelia, and in leprosy. *Trophic lesions of the deeper parts* involve the joints and the muscles. Trophic lesions of the joints, or *arthropathies*, are characterized by the enlargement of the joint involved, usually the knee, proliferation of the bone, relaxation of the ligament, so that the mobility of the joint is much greater than normal, and, for example, in the knee, there may be considerable lateral motion as well as flexion and extension. The joint surfaces become rough and give rise to a grating upon palpation. Curiously enough, aside from the undue mobility, the function of the joint remains relatively good, and the patient is often able to walk upon a knee that bends laterally almost to a right angle. There is usually little pain. These arthropathies may also assume the atrophic instead of the hypertrophic form—the *arthrite sêche* of the French. In this case the ends of the long bones atrophy and luxation commonly occurs. The frequency with which the different joints are affected is, according to Gowers, as follows: Knee, 45; hip, 20; shoulder, 11; tarsus, 8; elbow, 5; ankle, 4. In addition, the fingers and the ends of the ribs may show these alterations.

Alteration of the contour of the body occurs, as a whole, in various nervous diseases. In acromegaly the bones of the feet, hands, and face are greatly enlarged; there is usually slight kyphosis, and the soft parts become thickened, the whole appearance being extremely characteristic. In myxœdema the subcutaneous tissues are thickened, giving the subject the appearance of enormous obesity. In the various forms of amyotrophy, particularly the spinal type, the patient becomes extremely emaciated; alteration of the shape of the head occurs in hydrocephalus, the enlargement being globular, and the face, by contrast, very small; in microcephaly the cranium is greatly reduced in size, and the face appears more prominent and rather of an animal type. Occasionally, in the various chronic lesions associated with idiocy and epilepsy, there may be marked asymmetry of the skull. Sometimes an intracranial tumor will also produce a local distortion. Alterations in the expression or appearance of the face are produced by exophthalmic goitre, which is readily recognized, on account of the marked prominence of the eyes and the swelling of the neck. In facial tic the lightning-like contractions of the muscles on one side of the face, occurring at more or less frequent intervals, are extremely characteristic. In facial paralysis in the early stage the absence of folds on one side of the face, the drooping corner of the mouth, and partially opened eyelid are typical of the condition. In the later stage contractures may occur, causing the mouth to be drawn up and the eye to be kept partially closed with accentuation of the normal folds of the skin. Mimic paralysis—that is, failure of one side of the face or of both sides to assume an expression in accordance with the language or the feelings of the patient—occurs in lesions of the optic thalamus, and perhaps as a result of partial injury to the facial nerve. Stolidity of expression—that is, immo-

bility of the facial muscles—occurs in paralysis agitans. Finally, in various mental diseases the expression of the features may more or less closely indicate the type. Thus the mournful countenance of the melancholic, the excited, eager aspect of the maniac, or the furtive, anxious expression of the paranoiac, have all been described. It must not be forgotten, however, that temporary emotional states may give rise to the same manifestations. The Mongolian type of the features—that is, slightly oblique eyes and high cheek bones—seems to be characteristic of a certain form of idiocy. The reason for its occurrence is not known. Alterations in the posture of the body occur in a great variety of diseases. The spinal column may be permanently bent and ankylosed in rhyzomyelic spondylosis. This may also be associated with ankylosis of the large joints. The position and gait in every case are quite characteristic. Angular deformity of the spine occurs in Pott's disease. Lateral curvature frequently occurs in the various forms of muscular dystrophy and in Friedreich's ataxia. The presence of a large, fluctuating tumor at the base of the spinal column over the lumbar or sacral region is indicative of spina bifida, the lesion being, of course, congenital, and in this case there is often an extensive growth of hair upon the skin covering the tumor.

Changes in the Extremities. Various alterations in the contour of the arms are produced by muscular atrophy. The most characteristic is the flattening of the shoulder-joint that occurs as a result of the wasting of the deltoid and the peculiar appearance of the hand produced by the wasting of the thenar and hypothenar muscles. In the latter the thumb assumes a position parallel to the fingers, which is only characteristic, however, when it involves the metacarpal bone as well as the phalanges (*ape-hand*). The position of the hand is affected in paralysis of the extensors, giving rise to wrist-drop in injury to the radial and to the ulnar nerves. If the latter is involved the interossei muscles are paralyzed, so that the proximal phalanges can no longer be flexed, and the extensors gradually pull them backward until they are perpendicular to the dorsum of the hand (*main en griffe*). Enlargement of the hands, as a whole, occurs in acromegaly and in pulmonary osteoarthropathy. Mutilation of the fingers is frequently characteristic of syringomyelia, Morvan's disease, Raynaud's disease, and leprosy. (See Trophic Changes.) The alterations produced by muscular disease in the lower extremities are analogous to those that occur in the upper extremities. In addition, however, there is a peculiar alteration produced by pseudo-hypertrophic muscular atrophy, in which the limbs appear to be of Herculean development. Enlargement of the feet, as a whole, occurs in the same conditions as does enlargement of the hands. Deformities of the feet are much more common as a result of contractures following anterior poliomyelitis, which gives rise to the various types of club-foot. Certain nervous diseases frequently cause deformity of the knee and hip-joints, particularly syringomyelia, which gives rise to a form of dry arthritis of the hip; and tabes dorsalis, producing the tabetic arthropathies. (See Trophic Lesions.)

Mental Disturbances. These are of most varied kinds. They may be divided into disturbance of consciousness and disturbance of

intellection. Disturbances of consciousness may be of various degrees. The mildest form is called *apathy*. The patient lies quietly, makes no voluntary attempt to commence a conversation, shows no interest in his surroundings, and only answers if spoken to. A more severe state may be spoken of as *lethargy* or *stupor*. The term *coma* implies that it is impossible to arouse the patient by any means, and at the same time the condition resembles more or less closely actual sleep. The reflexes are usually preserved, and there is a certain degree of perception to painful impulses, manifested by the withdrawal of the part irritated. *Unconsciousness* is, of course, a condition that cannot be sharply differentiated from this. The term is ordinarily applied to conditions that do not resemble natural sleep. The patient may lie quietly, but the breathing is stertorous; the eyes may be open; all the muscles may be relaxed or various types of spasm may be present. These conditions occur in the intoxications, infections, poisonings, and as a result of severe injury to the head. A peculiar type of coma, known as *coma vigil*, is characterized by complete relaxation of the patient, whose eyes, nevertheless, remain open and appear to observe that which transpires around the bed. The mildest form of disturbance of intellect consists in impairment of memory, or *amnesia*. This may be restricted to the memory of certain things only, as the names of certain classes of objects or certain groups of words. It may also be restricted to loss of memory for certain definite periods of time, which may occur as a result of severe injury or disease during or about this period. If the memory is lost for the period preceding the traumatism, the condition is spoken of as antero-active amnesia; if for the period following, retro-active amnesia. Memory is commonly impaired in old age, and often as a result of chronic cerebral disease, particularly in paralytic dementia. General impairment of the intellect is spoken of as *imbecility* or *idiocy*. In its milder forms imbecility consists in diminution of the reasoning powers, so that the patient is unable to form accurate judgments. In its severer grades, and particularly in the more pronounced forms of idiocy, intellectual activity may appear to be absolutely abolished, life being merely a mechanical process not under control of the reason. Both conditions are usually associated with alterations in the substance of the brain, either in the form of hydrocephalus or of the various scleroses associated with epilepsy. Exaltation of the intellectual functions associated with excitement, and more or less violence is usually spoken of as *delirium*. This may be severe or mild. It is characterized by a tendency to talk or to be noisy, and by great restlessness. Delirium occurs in many of the acute infectious diseases, particularly in meningitis. Among the commoner symptoms of intellectual disorder usually grouped under the term insanity are exaltation, or *mania*, depression, or *melancholia*, and delusional states, or *paranoia*. By *mania* is meant excessive intellectual activity, characterized by a tendency to be noisy, to be active, fondness for singing, shouting, swearing, or punning. There is usually, also, in the acute forms a rapid loss of weight and decrease in the physical powers, while the patient believes himself to be in the most admirable and exceptional condition. Mania occurs as a nervous disease and as the result of inflammations of the brain-substance in *acute delirium*.

It occurs in the exacerbations of general paresis and in diseased states of unknown etiology that are denominated by the term itself. In *melancholia* the expression of the patient is mournful, he is commonly quiet, sits with his head cast down, refuses to speak, to eat, or to take any interest in what goes on about him. Often he weeps or groans constantly, and when persuaded to talk, expresses an acute sense of his manifold sins and the hopelessness of salvation, or will complain of misfortunes that have not befallen him. Melancholia occasionally occurs in general paresis, particularly in patients whose vitality has been exhausted by excesses. It also occurs as one of the varieties of insanity. The term *paranoia* is used by different authors in very different senses. In general, it may be said that the majority imply by it the existence of delusions or false ideas that have, among themselves, a certain logical sequence, or, as the term is, are organized. Thus a paranoiac may believe that he is being persecuted by a certain person, and be able to give reasons why his persecutor should torment him. It must not be forgotten that occasionally these delusions may be true in fact, although none the less symptoms of the mental condition. When there is merely a false idea it is spoken of as a *delusion*. If the person complains of certain physical impressions, such as non-existent sounds, visions, odors, or tastes, the term *hallucination* is generally employed.

Localization of Lesions of the Nervous System. In a diagnosis of diseases of the nervous system, particularly those that are the result of focal lesions, it is usually far more important to determine the situation of the lesions than the nature of the pathological process. The nervous system may be regarded physiologically as a collection of neurons. By *neuron* is meant a nerve-cell and all its processes to their ultimate ramifications. The processes are of two kinds: the so-called protoplasmic processes, which are relatively short, thick, and branched, and appear to resemble in many respects the protoplasm of the nerve-cell itself; and the axis-cylinder, a long, slender process that in its course gives off at regular intervals still more slender branches, the collaterals, and at its termination usually breaks up into a small tuft of fibres that surround some other ganglion cell. An exception to the latter rule is formed by the axis-cylinders of the motor cells that run to the muscles, and end in tufts of fibres distributed to peculiar terminations in the muscle-fibres. The axis-cylinders, at a certain distance from the nerve-cell, usually become surrounded by myelin sheaths, and constitute the nerve-fibres which make up the greater bulk of the central nervous system (the white substance), and practically all of the peripheral nervous system. Neurons with similar functions are usually grouped together, the aggregation of the cells forming a nucleus, and of the fibres a bundle or system. The gray matter is largely composed of these groups of ganglion cells or nuclei. Physiology has shown, although not absolutely conclusively, that the axis-cylinders convey impulses from the cell, and the protoplasmic processes convey impulses or nutriment to the cell. In the cell itself these impulses are modified or altered in some as yet unknown manner. At present the course and functions of comparatively few of the groups of neurons are known.

Those that have been most accurately studied may be divided into the sensory neurons, conveying impulses from the peripheral nervous system, and the motor neurons, conveying impulses from the central nervous system to the muscles. The *sensory neurons* commence in the various sensory corpuscles, in the skin, and organs. They pass through the peripheral nervous system to the posterior roots of the spinal cord, and here they enter the cells in the ganglia of the posterior roots. From these cells a fibre emerges that for a short distance is continuous with the entering fibre, and then leaves it and continues along the posterior root of the spinal cord. Here it divides into two branches, an ascending and a descending branch. Of the function of the latter nothing certain is known. Some of the ascending branches pass into the lateral posterior column (Burdach), and at a higher level into the median posterior column (Goll). Those entering the cord in the upper dorsal and cervical regions, however, do not pass into the median posterior column, but continue in the lateral posterior column to a nucleus in the medulla. Both columns end respectively in the nucleus cuneatus and the nucleus gracilis. These two nuclei may be looked upon as indicating the termination of the peripheral sensory neurons. These two groups of fibres probably convey only touch and muscular sensations. The fibres conveying pain and temperature sensations apparently pass up the cord through the central gray matter, but their central termination is not yet definitely known. From the ganglion cells in the two nuclei in the medulla, axis-cylinders arise that pass toward the brain and form a mass of fibres known as the filet. In the medulla these are situated on either side of the median line, lying between the olivary bodies. They continue to occupy the central regions of the pons in its posterior part, but anteriorly they gradually spread out until they form a narrow band, placed horizontally, just below the gray matter surrounding the aqueduct of Sylvius. They then enter the tegmentum of the crus, and the majority lose themselves in the ventral nucleus of the optic thalamus. They constitute the second chain of sensory neurons. It is probable that from the optic thalamus, and from the other nuclei in which perhaps fibres of the filet terminate, other axis-cylinders arise which pass through the corona radiata to the sensory areas in the cortex. These sensory areas will be discussed in connection with the cortical localization.

Destructive lesions in the peripheral sensory nerves produce total anæsthesia of the part supplied. Partial lesions may produce partial anæsthesia or even dissociation of sensation. Irritative lesions of the peripheral nerves produce severe pain, usually referred to the part supplied by the nerve, and there are also sensitive points or general tenderness over the nerve trunk. Certain forms of irritative lesion produce partial alteration of sensation, which is usually spoken of as paræsthesia (*q. v.*). Trophic changes in the skin often occur. Lesions of the posterior roots also produce total anæsthesia. If the lesion is on the peripheral side of the ganglion there are in addition trophic changes in the part supplied. If the lesion lies between the ganglion and the spinal cord, the anæsthesia is total, but trophic changes do not occur. Lesion of the ganglion itself usually produces anæsthesia and atrophic

changes, if complete; if partial, the symptoms are variable. In some cases herpes zoster along the course of the nerve has been observed. Irritative lesions of the posterior roots produce fulgurant pains in the limbs, or a feeling of constriction in the trunk. They may also be the cause of visceral crises. Destructive lesions of the posterior columns of the spinal cord produce more or less tactile anæsthesia and loss of the muscle sense. As a result of the latter there is ataxia. Lesions of either of the two central sensory neurons produce various forms of anæsthesia, depending upon their extent. According to our knowledge of

FIG. 239.

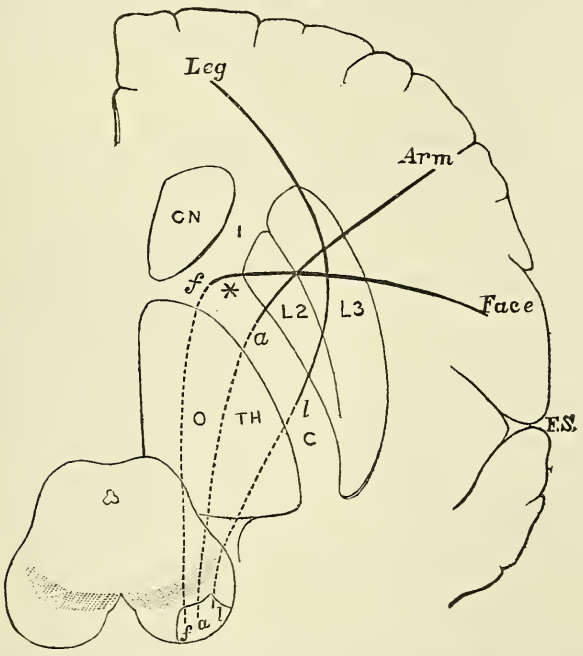


Diagram to show the relative positions of the several motor tracts in their course from the cortex to the crus.

The section through the convolutions is vertical; that through the internal capsules, I C, horizontal; that through the crus is again vertical. CN, caudate nucleus; O TH, optic thalamus; L2 and L3, the middle and outer parts of the lenticular nucleus; *f a l*, face, arm, and leg fibres. The words in italics indicate the corresponding cortical centres. (GOWERS.)

this subject, destructive lesions, such as hemorrhage or aneurism in the posterior portion of the posterior limb or the internal capsule, or destructive lesions of the optic thalamus, are usually associated with hemianæsthesia on the opposite side of the body. At times, tactile sense is preserved and only the pain sense lost. As a rule, however, all forms of sensation are more or less affected.

The *motor neurons* consist of two groups, the central and peripheral neurons. The central motor neurons commence in the motor portion of the cortex. They then pass through the corona radiata to the internal capsule, where they form a large band of fibres occupying the knee

and the anterior two-thirds of the posterior limb. (See Fig. 239.) The fibres for the face occupy the knee and anterior third of this portion. Next come the fibres for the arm, then those for the leg, and, finally, the fibres for the trunk. From the internal capsule the fibres pass into the crura cerebri, where they lie beneath the substantia nigra, occupying about the middle of each crus. The fibres for the face and cranial nerves lie internal to those for the extremities and trunk. From here they pass to the ventral portion of the pons, where they are broken up into small bundles by the association of fibres of the two cerebellar hemispheres. These reunite and form the pyramids in the anterior portion of the medulla, which decussate in the first cervical segment and pass down the cord as the lateral pyramidal columns. (See Fig. 240.) A few of the other fibres, however, do not decussate

FIG. 240.

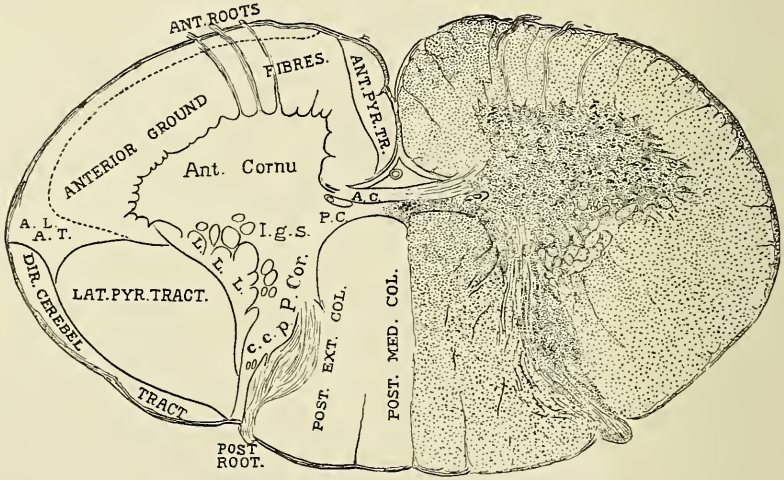


Diagram showing the different tracts of the cord. (GOWERS.)

at this time, but pass downward in the direct pyramidal columns, which decussate through the anterior commissure of the cord at lower levels. The fibres for the cranial nerves decussate, as a rule, in the neighborhood of the nuclei for these nerves, and by this means we are able to locate with considerable accuracy the situation of lesions in the pons and medulla. The fibres for the oculomotor nerves decussate in the tegmentum and the nuclei around the aqueduct of Sylvius. The fibres for the facial decussate in the anterior portion of the pons. From this point downward fibres are continually crossing the median raphe to the nuclei of the various motor cranial nerves until the main decussation—that is, in the first cervical segment. It follows, therefore, that if a lesion occurs in such a position that it affects the fibres of one of the cranial nerves after they have crossed the median line, at the same time involving the undecussated fibres of the pyramids, we will have the syndrome known as a *crossed paralysis*—that is, the muscles supplied by the affected cranial nerves will be paralyzed on the same side

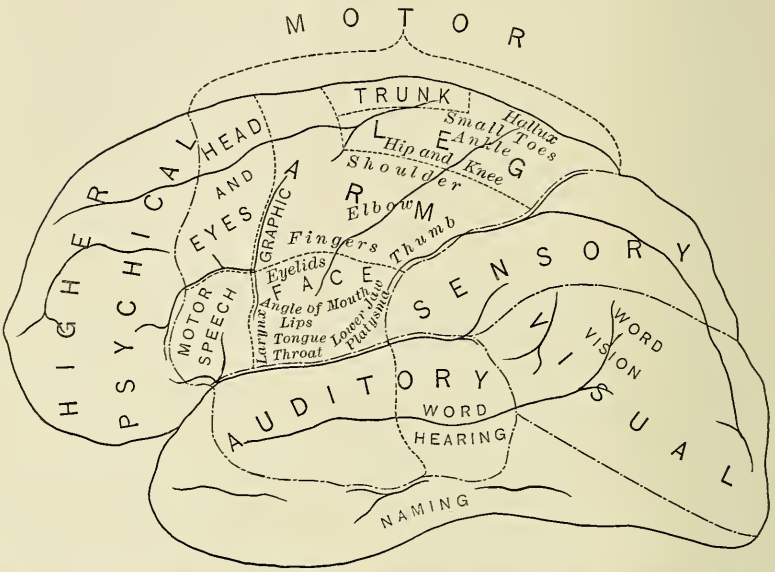
as the lesion, and the rest of the body on the opposite side. (See Lesions of the Cranial Nerves.) The *peripheral motor neurons* commence in the cells of the anterior cornua of the spinal cord, passing out through the anterior roots, and reach the muscles through the peripheral nerves.

The functions of these two neurons are apparently not identical. The central motor neurons convey impulses from the cortex to the cells of the anterior cornua, by which the latter are stimulated to produce muscular movement. At the same time they seem to possess an inhibitory influence by means of some form of constant activity, so that while they are intact the reflexes are restrained, and the muscles do not become spastic. Upon the nutrition of the muscles they apparently have no influence whatever, or at least act only indirectly by causing paralysis. The peripheral motor neurons control directly muscular activity. By their continuous action they maintain muscle tonus, and when unrestrained by the influence of the upper neurons produce a condition of spasticity. While they and the sensory neurons forming the arc are intact, reflex action persists. They also control in some mysterious way the nutritional changes in the muscles. Destructive lesions of the lower neurons—that is, of the peripheral nerves involving the motor fibres of the anterior root and of the ganglion cells in the cornua—cause paralysis and degenerative changes in the muscles. Irritative lesions cause spasms; these are usually tonic in character, and either momentary (as in facial tic) or more rarely persistent (tetanic). The muscle tonus is lost, and, therefore, the paralysis is flaccid in character and the reflexes are abolished. Destructive lesions in the central motor neurons, on the other hand, produce paralysis of the muscles, but their nutrition is not impaired, their muscle tonus is increased until they become spastic, and the reflexes are exaggerated. Irritative lesions of the central nervous neurons produce, as a rule, clonic spasms. These may be limited to the part irritated, as occurs in some form of central softening in the motor region, or become generalized. (See Convulsions).

Cortical Localization. The origins of the motor neurons and the terminations of the sensory neurons are, as will be seen from this description, in the cortex of the brain. It is, therefore, of considerable importance to be able to locate the portions of the cortex that have to do with these functions. As a result of experimental work and of the repeated examination of pathological specimens, a considerable amount of knowledge has been acquired upon this subject. The motor regions, indeed, are marked out with accuracy, and some of the regions for the reception of impulses from the organs of special sense are also certainly known. The cortex of the brain has been divided into various regions that are referred to certain fissures that are quite constant in position. The most important of these is the fissure of Sylvius. It separates the temporo sphenoidal lobe below from the frontal and parietal lobes above. Around its posterior extremity there winds the convolution known as the gyrus angularis. Next is the Rolandic fissure, passing from the superior longitudinal fissure to the fissure of Sylvius, with which it forms an acute angle. It separates the frontal from the pari-

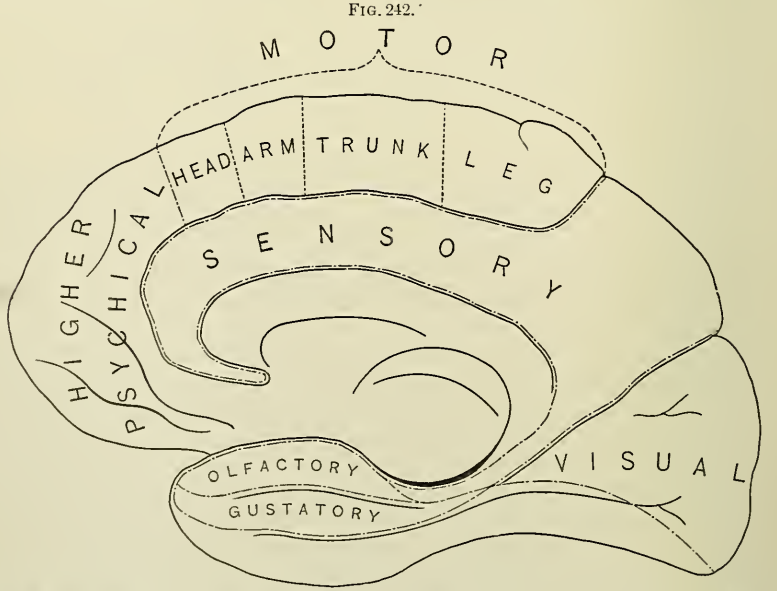
etal lobe, and lies in the midst of the motor region of the cortex. In front of it is the ascending frontal convolution, and behind the ascending parietal convolution. These two contain nearly all the motor centres. The third prominent fissure is the occipito-parietal. It is

FIG. 241.



Cortical centres and areas of representation on the lateral aspect of the hemicerebrum. (MILLS.)

FIG. 242.



Cortical centres and areas of representation on the mesial aspect of the hemicerebrum. (MILLS.)

best defined on the median surface of the brain, but can be traced for a short distance on the convex surface. It separates the parietal from the occipital lobe. On the median surface it unites at an acute angle with the calcarine fissure, the two enclosing between them the triangular convolution that is known as the cuneus. (See Fig. 241 and Fig. 242.) The motor centres are so arranged that those for the face are in the lowest portion of the motor region, those for the arms just above them, those for the legs around these, and those for the trunk in the posterior termination of the ascending parietal convolution, along the margin of the superior longitudinal fissure. These centres do not represent particular muscles, but particular forms of movement, involving frequently the simultaneous contraction of several muscular groups. It is not known how sharp their limitations are, but it is supposed that the central portion of the focus is most exclusively devoted to its function, while at the periphery this fades gradually into the surrounding centres. The motor region for speech was first discovered by Broca, in 1861. It occupies the posterior portion of the third frontal convolution and the lower part of the ascending frontal convolution. The termination of the sensory neurons is not yet conclusively determined. It seems likely that some of them terminate in the motor region, and others in the upper portion of the parietal lobe. It is probable that different forms of sensation are represented by different areas upon the cortex, but at present our knowledge of this subject is uncertain. The stereognostic sense appears to be situated in the parietal lobe—that is, lesions in this locality will cause its loss without disturbance of tactile sensation. As it has been shown that this sense is largely dependent upon muscular and localization senses, it is likely that the fibres concerning these terminate in the parietal lobe. It is to be noted that although it is the general rule that fibres from one hemisphere ultimately pass to the opposite side of the body, this is by no means invariably the case. Certain muscles, such as those of the trunk, apparently are innervated from both sides of the brain—that is, bilaterally—so that if one centre is destroyed the other assumes its functions, and no paralysis ensues. It also appears possible, in certain instances, for the centre of one hemisphere gradually to learn to perform the functions of the centre of the other hemisphere when the latter has been destroyed. This is seen most clearly in cases of the destruction of the speech centre on the left side, when, if the patient is still young, the speech centre on the right side may assume all its duties.

The Centres for Reception of Special Senses. The cuneus of the median surface of the occipital lobe appears to receive directly the fibres from the optic tract. When it is destroyed there is bilateral contralateral hemianopsia. The pupillary reflexes are, however, preserved, so that light impulses must exert some activity at a point in the chain of neurons between this and the eye, probably in the anterior quadrigeminal bodies. The centre for audition is situated in the temporo-sphenoidal convolution. Destructive lesions produce deafness in the ear of the opposite side, or at least impairment of hearing, which, as a rule, rapidly disappears. The centres for smell and taste have been placed respectively in the uncinate and fornicate convolutions.

The evidence for these localizations is very strong, but is not yet absolutely conclusive. It is doubtful whether irritative lesions in any of the centres for special sense are responsible for hallucinations.

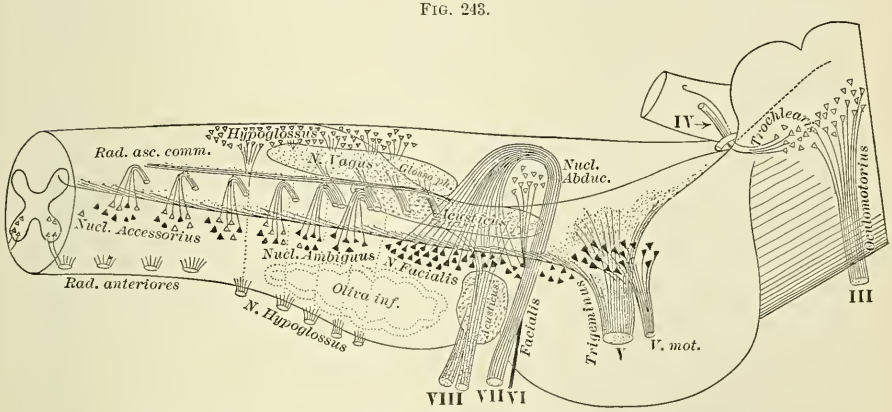
The functions of the *frontal lobes* are not well known. It has been supposed that they are the seat of intelligence, but there has never been adequate proof of this belief. Lesions of the frontal lobes may, therefore, exist without giving rise to any symptoms that lead to a suspicion of their presence. On the other hand, the patients may exhibit various intellectual disturbances, but, on the whole, none that are characteristic, and perhaps these symptoms do not occur more frequently as a result of disease of this part than when some other part of the brain has been affected. It has been claimed that there is a certain degree of intellectual impairment; that the patient, while not insane or even eccentric, becomes incapable of exercising the same degree of judgment and comprehension that he formerly possessed. It has been claimed, also, that a peculiar form of insanity, characterized by progressive dementia associated with a manifestation of self-contentedness, occurs only in lesions of this part, and it has been given the term moria. The production of a tendency to make puns has also been described to lesions in this region. It does not always occur, but, on the other hand, it may occur as an early manifestation of insanity without gross lesion or in connection with the lesions of other parts of the brain. The most important symptoms, of course, are those due to the involvement of the adjacent motor centres. The one most frequently affected is the speech centre in the third frontal gyri, and as a result aphasia is a common associated symptom, particularly if the lesion is situated in the left hemisphere. The other motor centres may, however, be involved and produce characteristic symptoms.

The functions of the *basal ganglia* of the brain are as yet insufficiently known to enable us to diagnose lesions situated in them with certainty. Lesions in the *lenticular nucleus* may be entirely latent. In some cases they appear to have produced sensory disturbances, but even this is doubtful. Ordinarily, the only symptoms they produce are those resulting from pressure upon the surrounding parts, such as the internal capsule. The *optic thalamus* appears to receive fibres from many parts of the cortex. Its relation to the fillet has already been mentioned, and lesions in this region frequently produce sensory disturbances. The pulvinar appears to be one of the three basal ganglia associated with the optic tract, and when it is destroyed there is usually bilateral contralateral hemianopsia. There is some doubt, however, whether this is not due to the involvement of the neighboring structures, either the fibres of the optic tract passing just beneath it or of the geniculate bodies. Nothnagel and v. Bechterew have called attention to the fact that certain localized movements on the part of the muscles of the face, particularly those concerned in the expression of the emotions, are more or less completely abolished by destruction of the optic thalami. The existence of this mimic paralysis has, in a few cases, led to the correct diagnosis of thalamic lesion. The *anterior corpora quadrigemina* apparently form one of the intermediate stations for the optic tract, the fibres from the nerve ending in them, and new

neurons commencing that possibly form the fibres of the optic radiation. They are apparently the situations in which the arch of the pupillary reflex is completed. The *internal geniculate ganglia* and the *posterior corpora quadrigemina* appear to be associated with hearing.

Lesions in the *pons* and *medulla* produce, as a rule, characteristic symptoms that make it possible to locate them with considerable accuracy. This is due to the fact that the nuclei of the cranial nerves are situated in these two portions of the brain, and also that they form the great source of communication between the cerebrum and the spinal cord, containing both motor and sensory fibres. (See Fig. 243.) The nucleus of the oculomotor nerve is found surrounding the anterior portion of the aqueduct of Sylvius, just beneath the anterior corpora quadrigemina. Numerous groups of cells have been separated which are supposed to belong each to a different muscle. Destructive lesions cause partial or complete ophthalmoplegia, according to the extent of the lesion. There is, therefore, abolition of the pupillary reflex. Just

FIG. 243.



Relative location of the nuclei of the different cranial nerves. (EDINGER.)

behind it, and beneath the posterior corpora, is a small group of cells for the pathetic nerve. The nucleus of the trigeminus is situated in the anterior portion of the pons, just to the outer side of the fillet, the motor group of cells lying inside the sensory group. The Gasserian ganglion receives the peripheral branches of this nerve and corresponds to the spinal ganglia. In addition the nerve receives a bundle of fibres from the lower portion of the medulla. Disturbances of the nucleus produce anæsthesia on the same side of the face, involving the conjunctiva and the mucous membrane of the mouth. There is loss of taste in the anterior two-thirds of the tongue, and there is some disturbance of smell in the nostril on the same side. At the same time the pterygoid muscles are paralyzed and mastication is imperfect. Irritative lesions cause tic douloureux. This may also be the result of disease of the ganglion. The nucleus of the *abducens* lies in the posterior portion of the pons, just beneath the floor of the fourth ventricle. Destructive lesions cause internal strabismus. The nucleus of the *facial nerve* is

found in the posterior portion of the pons, lying slightly behind and to the median side of the nuclei for the trigeminus. The fibres from this nerve pass out first forward, then downward and backward, and arise from the lateral surface of the medulla at its anterior extremity, passing forward over the pontine cerebellar tubercles. Destructive lesions cause paralysis of the same side of the face, usually involving the upper branch. (See Hemiplegia.) Irritative lesions cause facial tic. The nucleus of the *acusticus* is found in the anterior portion of the medulla oblongata, just beneath the floor of the fourth ventricle, lying just above the superior olivary body. Lesions produce nerve or mental deafness on the same side. The nuclei of the *vagus* and the *glosso-pharyngeal nerves* are apparently in the jugular and petrosal ganglia—that is to say, they are sensory nerves, and correspond to the sensory fibres entering the spinal cord. From these ganglia fibres pass into the medulla oblongata at its lateral aspect, and end in a nucleus in the floor of the fourth ventricle. The motor nucleus of the vagus is supposed to be the nucleus ambiguus, situated just posteriorly to the olive in the posterior portion of the floor of the fourth ventricle. Close to the median line is the *hypoglossal nucleus*. Its destruction produces paralysis and degenerative atrophy of the corresponding side of the tongue.

The functions of the *pons* are merely those of the centres and tracts it contains, and therefore the symptoms are dependent upon the situation and greater or less amount of destruction that the lesions produce. On account of the decussation of the central fibres for the facial nerve in this region, crossed paralysis is usually considered pathognomonic of pontine disease. The functions of the *medulla* are also largely dependent upon the nuclei and tracts it contains. As it contains the centres for the pneumogastric and some of the centres or tracts of fibres for respiration, lesions in it are ordinarily followed very promptly by death. Lesions of the *restiform bodies*—that is, the lower portion of the medullary peduncle to the cerebellum—are frequently associated with nystagmus, and may cause the symptoms of cerebellar ataxia. As the medulla contains the nuclei of the motor nerves to the pharynx, larynx and mouth, paralysis of the muscles in this region is spoken of as bulbar palsy.

The *cerebellum* is supposed to be concerned in co-ordination and the maintenance of the equilibrium. The hemispheres may, however, be extensively diseased without giving rise to any symptoms. If the middle lobe is affected the characteristic manifestations are disturbance of equilibrium and inco-ordination. The gait resembles that of a drunken man, nystagmus is frequent, especially in cases of tumor. Giddiness and vomiting sometimes occur, but are, however, of no localizing value. The knee-jerk is often absent, but sometimes increased and sometimes variable. If the pyramidal tracts are pressed upon it is always increased, and there is then weakness in the extremities. As a result of pressure there may be paralysis of the cranial nerves, difficulty in articulation, and occasionally epileptiform convulsions. If the medullary peduncle is affected by an irritative lesion, quite characteristic symptoms result. These are forced movements—that is to say, the patient may have an irresistible tendency to fall

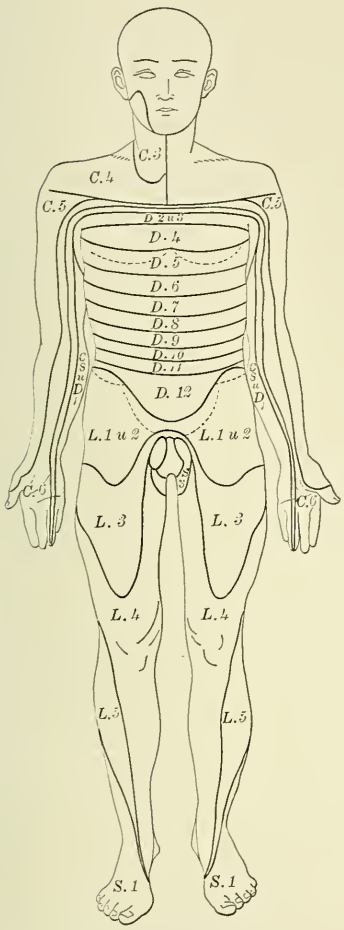
toward or lie upon one side. There are no symptoms diagnostic of disease of the superior or middle peduncles. Disease of one side of the pons may cause symptoms similar to those of cerebellar trouble.

Localization of Spinal Lesions. The spinal cord may be regarded in two ways: First, as the pathway between the peripheral nervous system and the brain, containing the tracts running from the brain to the motor nerves, and from the sensory nerves to the brain; second, as a number of groups of ganglion cells arranged in horizontal layers or segments. These segments are usually classified according to the nerve-roots that spring from them. There are, therefore, eight cervical, twelve dorsal, five lumbar, and five sacral segments of the cord. The white matter of the spinal cord is divided into two regions: the antero-lateral part, extending from the median fissure to the posterior horns, and the posterior part, lying between the posterior horns. The antero-lateral part contains the motor fibres or pyramidal tracts, whose functions have already been described. In addition, there are certain fibres that pass downward whose functions are not certainly known. The gray matter of the cord is divided into the anterior and the posterior horns. It is composed of nerve-cells and nerve-fibres. The nerve-cells in the anterior horns form a large group, which send their axis-cylinders into the anterior roots, and comprise the peripheral motor neurons. In the posterior horns, in the dorsal region, there is a group of cells on the inner side known as the column of Clarke, which apparently have something to do with equilibration. Other cells, whose functions are not definitely known, are also found in the posterior cornua. The gray matter also contains a large number of nerve-fibres, some of which pass transversely and apparently are concerned in reflex action; others ascend, and convey to the brain the sensations of pain, heat and cold. Each segment of the cord innervates and receives sensory impressions from an approximately corresponding segment of the body, and contains the lower reflex arcs. The motor and reflex functions of the various segments are shown in the table and the sensory functions in Fig. 244 and Fig. 245.

TABLE OF MOTOR AND REFLEX FUNCTIONS OF THE SEGMENT OF THE SPINAL CORD. MODIFIED FROM GOWERS AND MULLER.

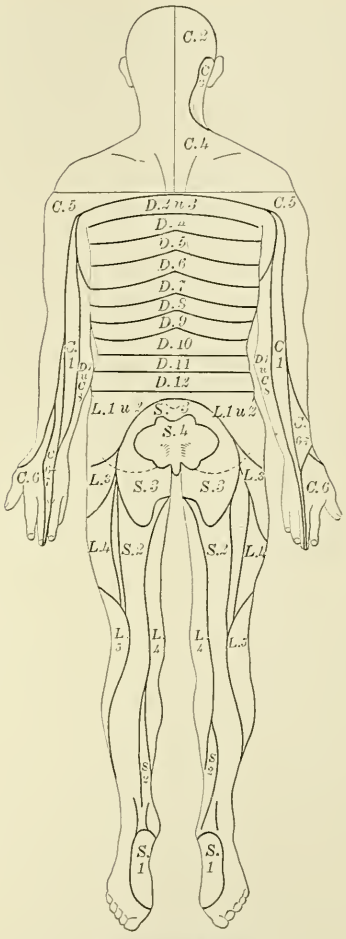
Segments.	Motor innervation.		Reflex centres.
C.			
1	Small rotators of head		
2	Depressors of hyoid	Scaleni.	
		Lev. ang. scapulæ.	
3	Diaphragm	Cucullaris.	
4	Platysma (?)		
5	Deltoid		
	Biceps		Dilatation of the pupil, sensory part. (?)
	Coraco brachialis		
	Supinator longus		
	Spinati		Scapular.
	Serratus major		
6	Pectoral major (clav.)	Pronators	
		Triceps	
7	Flexors of wrist and fingers	Extensors of wrist and fingers	Tendon reflexes of the muscles of the arms.
	Pectoralis (costal)		
	Subscapularis		
	Latissimus dorsi	Muscles of hand	
8	Teres major		Dilatation of pupil, motor part. (?)
		Extensors of thumb	
D.			
1			
2			
3			
4			Epigastric.
5			
6	Intercostal muscles		
7		Erectors of spine	
8			
9			
10	Abdominal muscles		
11			Abdominal.
12			
L.			
1	Quadratus lumborum		
2	Ileo psoas		
	Cremaster		Cremasteric.
3	Sartorius		
	Pectineus		Knee-jerk.
	Adductors		
4	Quadriceps		
	Gracilis		Gluteal reflex.
	Obturator	Gluteal	
5	Adductors		
	Flexors of knee		
S.			
1	External rotators of thigh		Achillis tendon reflex.
2	Extensors of foot		Plantar reflex.
	Tibialis anticus		
	Peroneal muscles		
3	Perineal and anal muscles		Centres for the bladder and rectum.
4			
5			

Fig. 244.



(From Oppenheim.)

Fig. 245.



(From Oppenheim.)

General Symptomatology of Lesions of the Brain. Lesions of the brain may be irritative or destructive. The former, if affecting the motor tract, produce clonic spasms. If destructive, they produce paralysis without atrophy, and cause increase in the muscle-tone by the removal of the influence of the superior arc and exaggeration of the reflexes. All these changes occur in the muscles of the opposite side of the body. Irritative lesions are most likely to be extra-cerebral —that is, pressing upon the cortex. Lesions in the brain-substance are usually destructive, and, therefore, cause paralysis. As motor fibres are distributed over a considerable area of the cortex, lesions in this region, if circumscribed, are likely to cause monoplegia. If involving the area for the face, the upper branch of the facial nerve, which is innervated from both sides, is rarely involved. Aphasia only occurs if the left side is destroyed. Lesions in the corona radiata near the

cortex usually cause monoplegia; if near the internal capsule, hemiplegia is more common. Lesions in the internal capsule almost invariably cause hemiplegia. If the knee and anterior portion of the posterior limb are involved, hemiplegia without sensory changes results. If they also affect the posterior third of the posterior limb, sensory disturbances are present, and there is likely to be hemianopsia. Lesions in the anterior portion of the anterior limb produce no recognizable symptoms, and are termed latent. General disturbances of the brain may be caused by increase of the intracranial pressure. This may be brought about by growths, traumatism, œdema, or inflammation. There is usually headache, delirium or coma, and vomiting. If the process is of slow development, a certain amount of adaptation may occur, and only the headache and vomiting may be present. The former is occasionally sharply localized. In addition, if the pressure be long continued, there is œdema of the optic nerve. (See Disorders of the Special Senses.)

General Symptoms of Disease of the Spinal Cord. These depend upon the segment of the cord and upon the nerve-tracts involved. Lesions are spoken of as transverse if they involve the whole cord, unilateral if they involve but one side, and focal if they involve only a circumscribed portion. Transverse lesions may be produced by inflammation, by pressure either by a tumor or as a result of deformity of the vertebral column (Pott's disease). Transverse lesions above the fifth cervical segment usually cause death by paralysis of the diaphragm. If the patient survive there is paralysis of all four extremities and total anæsthesia of the body. There is also paralysis of the bladder and rectum and abolition of the cutaneous reflexes, and, in the majority of cases, of the tendon reflexes. Transverse lesions between the fifth cervical and the first dorsal segments produce atrophy and degeneration of certain muscles of the arm, according to their situation. There is spastic paralysis of the legs and total anæsthesia of the body as far up as the part that transmits sensation to the lowest intact segment. There is paralysis of the bladder and rectum, abolition of the reflexes whose arcs are found in the segments involved, and sometimes exaggeration of all the tendon reflexes that are completed in the lower segments. The cutaneous reflexes are abolished. Lesions of the dorsal region produce spastic paraplegia and paralysis of the bladder and rectum. The arms escape entirely, and respiration is not disturbed. The anæsthesia extends up to the segment involved. Lesions in the lumbar region produce atrophy and degeneration of certain groups of muscles in the legs, with paralysis and disturbances of sensation, distributed according to their extent. The situation of a lesion may be roughly determined by a study of the reflexes. If the lesion involve the segments concerned in any of these, they are, of course, abolished. If the lesion is above them, they are sometimes exaggerated; if below them, they are ordinarily not involved. Lesions of the conus terminalis and the cauda, as they involve the large number of nerve-roots, produce a complexity of symptoms. There are irregular areas of anæsthesia corresponding to the posterior roots involved, and atrophy and degeneration of the muscles supplied by the anterior roots. The bladder and rectum usually are affected.

Unilateral Lesion of the Spinal Cord (the syndrome of Brown-Séquard). This produces paralysis of the same side and anæsthesia of the opposite side, both symptoms extending as far upward as the region supplied by the segment that has been affected. Disturbance of sensation is not total. There is tactile anæsthesia, analgesia, and loss of temperature-sense on the side opposite the lesion, but persistence of the muscular sense, which, however, is diminished or lost on the same side as the lesion. Disturbance of motion is complete. Atrophy and degeneration occur in the muscles supplied by the involved segment; below this there is spastic paralysis, with increase in the reflexes. Above the paralytic area there is a zone of hyperæsthesia that has never been satisfactorily explained. The commonest cause of unilateral lesion is traumatism, particularly bullet and stab wounds. Occasionally the symptoms develop in the early stages of syringomyelia or as a result of tumor or hemorrhage of the spinal cord. Focal lesions in the spinal cord produce various symptoms, according to their situation. Inflammations involving the gray matter are commonly spoken of as poliomyelitis. They usually attack the anterior cornua and involve only the peripheral motor neuron—that is, they produce paralysis, atrophy, and degeneration of the muscles. Inflammatory lesions in the white matter are spoken of as leukomyelitis. They produce symptoms according to the tracts they involve.

The Cranial Nerves. The olfactory, optic, oculomotor, pathetic, abducens, auditory, and glosso-pharyngeal have already been described in connection with the special senses. The *trigeminal nerve* takes its origin from the centres in the pons and medulla already described. Destructive lesions of the motor portion cause paralysis of the pterygoid muscles. If they are unilateral it is impossible for the patient to move the mouth toward the opposite side when the lower jaw is protruded. It is to be assumed that atrophy and degeneration of these muscles occur, but their electrical examination is practically impossible. Irritative lesions produce cramp known as *trismus*. This is, of course, usually due to central disease. The sensory portion of the trigeminus supplies the skin of the face and the mucous membranes of the cavities of the head. The distribution of the three branches is shown in Fig. 246. Irritative lesions produce tic douloureux; destructive lesions, anæsthesia in the distribution of the part affected. The *facial nerve* arises from the nuclei in the posterior portion of the pons. These are probably double, each supplying a separate branch of the nerve, and the superior nucleus is innervated from both sides of the cerebrum. It is the motor nerve for the muscles of the face, and supplies the temporal, masseter, the orbicularis palpebrarum, the muscles of the lower part of the face, the muscles of the palate, and the platysma myoides. Unilateral destructive lesions produce paralysis of the muscles of the face (Bell's palsy). This can be recognized by the disappearance of the folds, drooping of the corner of the mouth, and the inability to close the eye. In addition there may be loss of taste and hyperacusis in the ear on the same side. Occasionally there is deviation of the tongue, the palate is oblique, and the uvula is pulled toward the sound

side. If the peripheral portion of the nerve is involved, usually both the upper and lower branches are affected, and the paralysis is general. If the lesion is central the upper branch commonly escapes, or, at least, instead of being paralyzed, is only paretic. Moreover, in central lesions lying above the pons the opposite side of the body is paralyzed. Secretion of saliva on the same side is diminished or abolished. This may be tested on the sublingual glands by raising the tip of the tongue, carefully drying the sublingual space and getting the patient to inhale some pungent substance, such as acetic acid or musk. The saliva will immediately appear on the sound side, but will fail to appear on the other. In facial paralysis it is impossible for the patient to masticate on the diseased side, because the food collects between the cheek and the gums. It is also impossible for him to whistle. Saliva freely dribbles from the drooping corner of the mouth, and as it is impossible to contract the orbicularis palpebrarum the eye remains open even in sleep (*lagophthalmus*), and the corneal reflex is abolished or imperfect. When the patient attempts to close the eye the ball rolls upward and outward. In addition, the palatine reflex also disappears. In facial paralysis of long standing contractures may occur. In all cases the muscles show either partial or complete reactions of degeneration. Irritative lesions of the facial nerve cause spasm of the facial muscles, usually spoken of as facial tic. The *vagus* nerve supplies motor fibres to the larynx, sensory fibres to the lungs, and inhibitory fibres, probably sensory in nature, to the heart. It also probably sends sensory fibres to the gastro-intestinal tract. Destructive lesions of the vagus produce, if unilateral, unilateral paralysis of the vocal cords, interference with deglutition, and transient tachycardia. The laryngeal changes are most characteristic. (See Chapter I., Part II.) Irritative lesions produce spasm of the glottis, with dyspnœa or aphonia. The *spinal accessory* nerve is the motor nerve for the trapezius and part of the sternocleidomastoid. Destructive lesions of this nerve are the chief cause of torticollis. The *hypoglossal* nerve is the motor nerve for the tongue, and is, therefore, concerned in chewing, swallowing, and speaking. Unilateral destructive lesions produce paralysis of one-half of the tongue, which is protruded toward the paralyzed side, with atrophy and degeneration of the muscle. Fibrillary twitchings are usually present. The functional disturbance, however, is slight, and the patient may complain of no discomfort. Bilateral paralysis produces, however, very severe symptoms. The tongue lies flaccid in the mouth, it is impossible to protrude it, or even to move it from side to side. Mastication is impossible and swallowing exceedingly difficult. Speech is at first seriously affected, but, as a rule, the patient in time learns to compensate the lingual palsy. Paralysis of the tongue as a result of central lesion almost never occurs.

General Diagnosis of Nervous Diseases. It is necessary to study the patient according to some fixed plan, otherwise its complexity and the numerous investigations that it is necessary to make render a thorough examination almost impossible. It is true, of course, that in actual clinical practice diseases will be met whose clinical symptoms are so characteristic that the diagnosis can be made almost by

inspection alone, and a prolonged examination will only be useful for the purpose of excluding or detecting possible complications. On the other hand, certain cases will occur that almost defy diagnosis, on account of the multiplicity and contradictory character of the symptoms. In general it may be said that, aside from the history and the subjective symptoms, the physician will meet with four groups of signs: disturbance of sensation, disturbances of motility, atrophic and degenerative lesions, and disturbances of intelligence. These should be taken up in the following order: 1. Disturbance of *intellect*. It is often possible to detect this, when it exists, by simple conversation with the patient. It may be indicated by the history, or, on the other hand, the history and the behavior of the patient may exclude it altogether. 2. Disturbances of *motion*. It is well to study first the more patent alterations. Thus the patient should be told to move the arms and legs, in order to detect paralysis; he should be requested to walk, in order to study the gait; he should be directed to perform some fine, co-ordinated movement, in order to detect possible ataxia; and to put the muscles in a state of tension, in order to exaggerate a possible tremor. Following this the individual movements should be carefully examined. It must be remembered that, whether the lesion is in the central or peripheral nervous system, disturbance of motility is manifested only in the muscles themselves, and the investigations, therefore, should commence with these—that is to say, it is not desirable to test the motor functions of each particular nerve, but rather of each particular group of muscles, and to deduce from the changes found in them the nerve or segment involved. The following table from Sahli gives a classification of the muscles of the extremities, according to their functions, with their nerve-supply:

TABLE OF THE VOLUNTARY MUSCLES GROUPED ACCORDING TO THEIR FUNCTIONS, WITH THEIR NERVOUS SUPPLY. (FROM SAHLI.)

UPPER EXTREMITY.

A. MOVEMENTS OF THE SHOULDER-BLADE.

1. *Elevators of the shoulder.*

Middle part of the cucullaris (N. accessorius).
Rhomboidei (N. dors. scapul., 5th cervical nerve).
Levator scapulæ (2d and 3d cerv. nerv. and N. dors. scap.).
Upper portion of the pectoral major (Nn. thorac. ant., 5th and 6th cerv. nerves).

2. *Depressors of the shoulder.*

Pectoralis minor (Nn. thorac. anterior).
Lower portion of the latissimus dorsi (N. subscapularis).
Lower portion of the pectoralis major (N. thorac. ant.).

3. *Adduction of the shoulder.*

Lower portion of the cucullaris (N. accessor.).
Upper portion of the latissimus dorsi (N. subscapularis).

4. *Abduction of the shoulder.*

Upper third of the pectoral. major (N. thor. ant.).
Serratus anticus major (N. thorac. longus, 6th, 7th, 8th cerv. nerv.).

B. Movements of the shoulder-joint.

1. *Elevators of the arm.*
   (*a*) Laterally, deltoid (N. axillaris).
   Vertically, serratus anticus major (N. thorac. longus).
   (*b*) Anteriorly, anterior portion of the deltoid (N. axillaris).
   Coracobrachialis (N. musculocutaneous).
   Biceps (N. musculocutaneous).
   (*c*) Posterior portion of the deltoid (N. axillaris).

2. *Adduction of the arm.*
   Pectoralis major (N. thorac. anticus, 5th and 6th cerv. n.).
   Latissimus dorsi and teres major (N. subscapularis).
   Infraspinatus (N. suprascapular, 5th and 6th cerv. n.).
   Teres minor (N. axillaris).
   These muscles also depress the arm.

3. *Internal rotation.*
   Subscapularis (Nn. subscapulares).

4. *External rotation.*
   Infraspinatus (N. suprascapularis).
   Teres minor (N. axillaris).

C. Movements of the elbow.

1. *Flexion.*
   Biceps (N. musculocutan.).
   Brachialis (N. musculocutan.).
   Supinator longus (N. radialis).

2. *Extension.*
   Triceps (N. radialis).

3. *Supination.*
   Supinator brevis, Supinator longus } (N. radialis).

4. *Pronation.*
   Pronator quadratus, Pronator teres } (N. medianus).
   Supinator longus (N. radialis).

D. Movements of the wrist-joint.

1. *Flexion.*
   Flex. carpi radialis (N. medianis).
   Flex. carpi ulnaris, Palmaris longus } (N. ulnaris).

2. *Extension.*
   Extensor radialis longus and brevis, Extensor ulnaris } (N. radialis).

3. *Abduction.*
   Flexor carpi radialis, Radialis longus and brevis } (N. medianis and N. radialis).

4. *Adduction.*
   Extensor ulnaris and flexor carpi ulnaris (Nn. radial. and ulnar).

E. Movements of the fingers.

1. *Flexion.*
   Flexor digitor. sublim.; flexion of the 2d phalanx (N. median).
   Flexor digitor. prof.; flexion of the terminal phalanx (Nn. median, ulnar).
   Interossei and lumbrical muscles, flexion of the proximal phalanx (Nn. ulnaris, median.)

2. *Extension.*
   Extensor dig. comm. (N. radialis).
   Interossei and lumbrical muscles (N. ulnar, N. median).

F. Movements of the thumb.

1. *Flexion.*
   Flexor pollicis longus and brevis (N. median).
2. *Extension.*
   Extensor pollicis longus and brevis (N. radialis).
3. *Abduction.*
   Abductor pollicis long. (N. radialis).
   Abductor pollicis brev. (N. median).
4. *Adduction.*
   Adductor pollicis (N. ulnaris).
5. *Opposition.*
   Opponens pollicis, Adductor pollicis brev. } (N. median).

G. Movements of the little finger.

1. *Flexion.*
   Flexor communis digitorum profundus and sublimis (N. median and N. ulnaris).
   Flexor brevis minimi digiti (N. ulnaris).
2. *Extension.*
   Extensor minimi digiti proprius (N. radial.).
3. *Abduction.*
   Abductor minimi digiti (N. ulnaris).
4. *Opposition.*
   Opponens minimi digiti (N. ulnaris).

Lower Extremity.

A. Movements of the hip-joint.

1. *Elevation of thigh.*
   Iliopsoas (N. plexus lumbalis).
   Rectus femoris, Sartorius } (N. cruralis).
2. *Depression of thigh.*
   Glutæus maximus (Nn. glut. inf. and ischiadicus).
   Flexors of the knee (N. ischiadicus).
3. *Internal flexion.*
   Glutæus med. and minim. (N. glut. super.).
4. *External rotation.*
   Quadratus femoris, Obturator int. and Gemelli } (N. ischiadicus).
   Obturator ext. (N. obturat.).
   Pyriformis (Plex. ischiad.).
   Iliopsoas (Plex. lumbal.).
   Glutæus max. (N. glutæus inf.).
5. *Adduction.*
   Adductores (N. obturator).
   Pectineus (N. crural and obturat.).
   Gracilis (N. obturator).
6. *Abduction.*
   Glutæus med. and min. (N. glut. sup.).

B. Movements of the knee-joint.

1. *Flexion.*

Sartorius (N. cruralis).
Gracilis (N. obturat.).
Semitendinosus, Semimembranosus, Biceps } (N. ischiad.).
Popliteus (N. tibial, N. ischiad.).

2. *Extension.*

Quadriceps (N. cruralis).

C. Movements of the ankle-joint.

1. *Dorsal flexion.*

Tibial antic., Extensor commun. dig. long. } (N. peron. prof.).

2. *Plantar flexion.*

Gastrocnemius, Soleus } (N. tibial).
Perineus long. (N. peron. superficial).

3. *Adduction.*

Tibial postic (N. tibial).
Tibial ant. (N. peron. prof.).

4. *Abduction.*

Peroneus long., Peroneus brevis, Comm. dig. long } (N. peron. prof.).

5. *Elevation af the inner side of the foot.*

Tibial ant. (N peron prof.).
Tibial post. (N. tibial).

6. *Elevation of the outer side of foot.*

Peroneus long. and brev., Peroneus tertius } (N. peron. superf.).

D. Movements of the toes.

1. *Flexion.*

Flexor comm. digit. long. and brev., Interrossei and lumbricales } (N. tibial).

2. *Extension.*

Extensor comm. digit. long. and brev. (N. peron. prof.).

3. *Adduction.*

Interossei plantares (N. tibial).

4. *Abduction.*

Interossei dorsales (N. tibial).

E. Movements of the great toe.

1. *Flexion.*

Flexor hallucis long. and brev. (N. tibial).

2. *Extension.*

Extensor hallucis long. and brev. (N. peron. prof.).

3. *Adduction.*

Adductor hallucis (N. tibial).

4. *Abduction.*

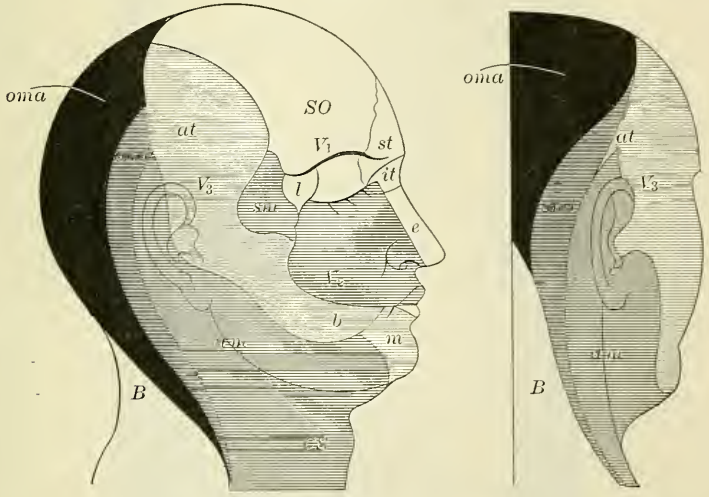
Abductor hallucis (N. tibial).

F. Movements of the small toe.

1. *Flexion.*
   Flexor minimi digit. (N. tibial).
2. *Abduction.*
   Abductor minimi digit. (N. tibial).
3. *Opposition.*
   Opponens minimi digit. (N. tibial).

Each movement should be tested by requesting the patient to perform it first unimpeded, and then against resistance. (For functions of motor cranial nerves, see page 1025.) 3. Sensory disturbances. As in testing the motor disturbances, there is first obtained a rapid orientation of the sensory condition of the patient. For this purpose it is customary to touch with the finger or a blunt object both sides of the

Fig. 246.



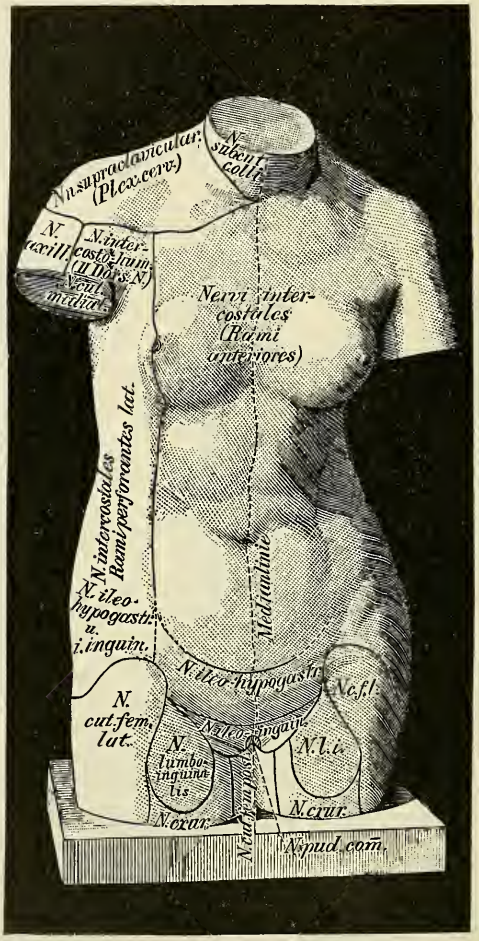
Cutaneous nerves of the head and face.

V_1, V_2, V_3, first, second, and third branches of the trigeminus; *S O*, supra-orbital: *l*, lachrymal *st*, supratrochlear; *it*, infratrochlear; *e*, ethmoidal; *sm*, malar; *at*, auriculo-temporal; *b*, buccinator; *m*, mental; *am*, auricularis magnus; *oma* and *omi*, occipitalis major and minor.

face, the arms, the legs, and both sides of the body. If the patient declares that there is no difference in the sensory perceptions, tactile anæsthesia may be temporarily excluded. The same regions are tested for pain and temperature-sense, and it is often desirable to test the muscle-sense at the same time, although this properly belongs to disturbances of motility. It is often possible, in testing sensation, to decide whether the lesion is peripheral or central by its distribution. If it affects the spinal cord it will be segmental in type. (See Fig. 244 and Fig. 245.) If it affects the peripheral nerves, the area or areas will correspond to the cutaneous distribution of the nerve or nerves involved. (See Fig. 246 *et seq.*) 4. The cutaneous trophic changes occur

particularly in the form of panaritis of glossy skin or of bed-sores. Trophic changes in the joints occur especially in the knee, shoulder, and hip. Trophic changes in the muscles may occur in any part of the body. They are, of course, nearly always associated with distinct paralysis. Having obtained a rough idea of the condition of the patient, it is then

FIG. 247.

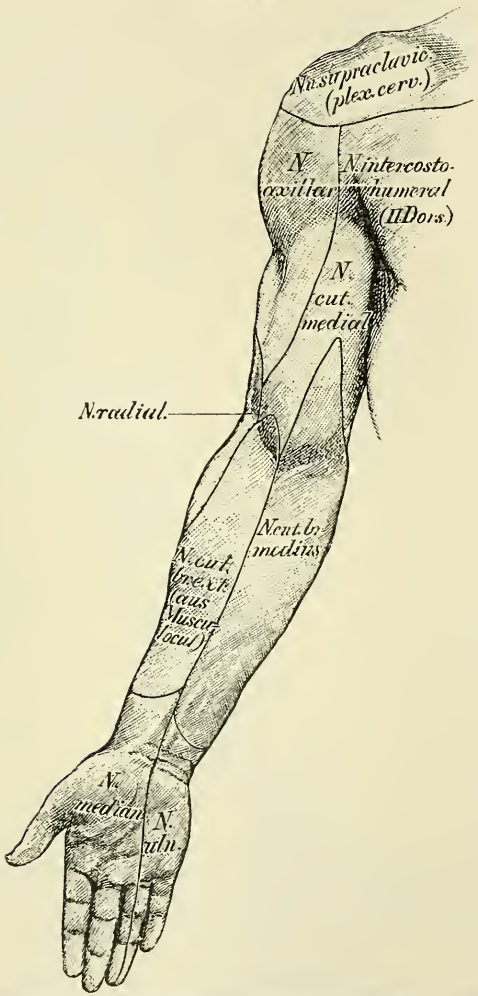


Cutaneous nerves of the anterior surface of the trunk. (SAHLI.)

necessary to make a more minute examination. 1. The various functions should be carefully studied, particularly those of the cerebral nerves. These should be taken up in order and all their functions tested. 2. It is important to note the reflexes, especially those of the eye, and the tendon and cutaneous reflexes of the body and

extremities. 3. The position, station, and gait. 4. The disturbances of speech. 5. The condition of the individual muscles and nerves of

FIG. 248.



Cutaneous nerves of the anterior surface of the arm. (SAHLI.)

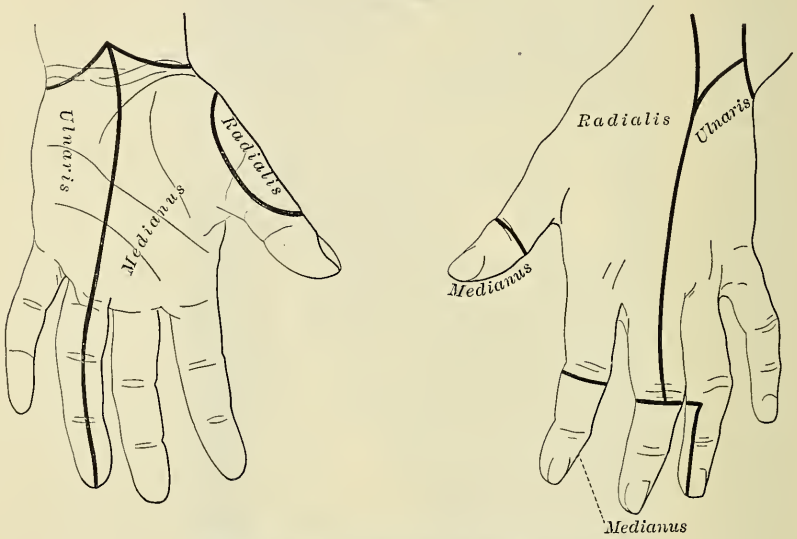
the body. The diagnosis must then be made by the study of the symptoms elicited. It should be, if possible, both topical and pathological, although it is not always possible to make the latter.

SPECIAL DIAGNOSIS OF DISEASES OF THE NERVOUS SYSTEM.

The semeiological classification of nervous diseases presents many difficulties. Many of the diseases that are closely analogous in their

symptoms are widely different in their pathology or etiology, and many diseases present such variations in their symptom-complex that at one period they could properly be placed in one group and at another period elsewhere. In general, it may be said, however, that the diseases of the peripheral motor neurons differ so widely from those of the central motor neurons that they can be classified as two separate groups, and in a third group would come the diseases of the sensory neurons. Combinations of these three groups, producing on their part rather clearly marked complexes of symptoms, may then be described, and finally the general and local diseases of the brain and

FIG. 249.



Distribution of the cutaneous nerves in the hand.

cord. An entirely separate group, characterized by peculiar symptoms, are the so-called functional nervous diseases, or the neuroses. It must be admitted, however, that this group, as a result of more accurate methods of investigation, is growing rapidly smaller.

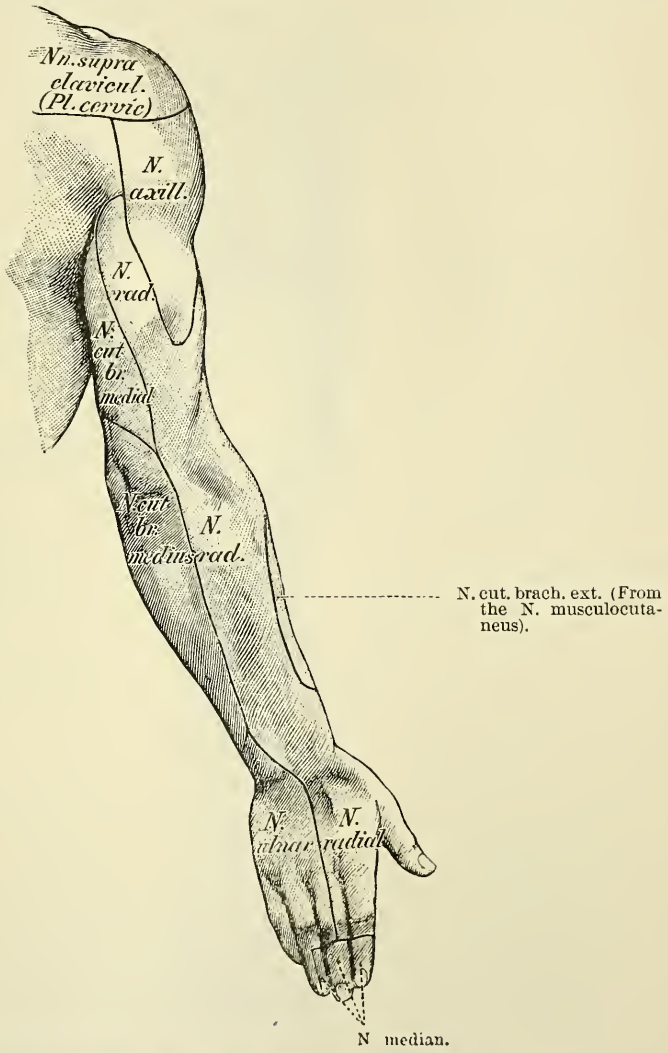
Diseases of the Peripheral Motor Neurons and the Muscles.

DISEASES CHARACTERIZED BY PURE MOTOR DISTURBANCE.

Progressive Muscular Atrophy. Two forms are recognized — the scapulo-humoral type of Erb and the facio-scapulo-humoral type of Dejerine-Landouzy. In the former the disease commences in the muscles of the shoulder, especially the pectorals and the latissimus dorsi. Next the adjacent muscles are involved, followed by the muscles of the arms, thighs, and finally the muscles of the calf. There is gradual loss of power corresponding to the atrophy of the muscles, but reactions of degeneration do not occur. As a result of the

wasting, peculiar alterations occur in the configuration of the body—that is, the shoulder-blades become prominent, lordosis occurs, and, as a result of the weakness of the glutei, it may be necessary for the

FIG. 250.



Cutaneous nerves of the posterior surface of the arm. (SAHLI.)

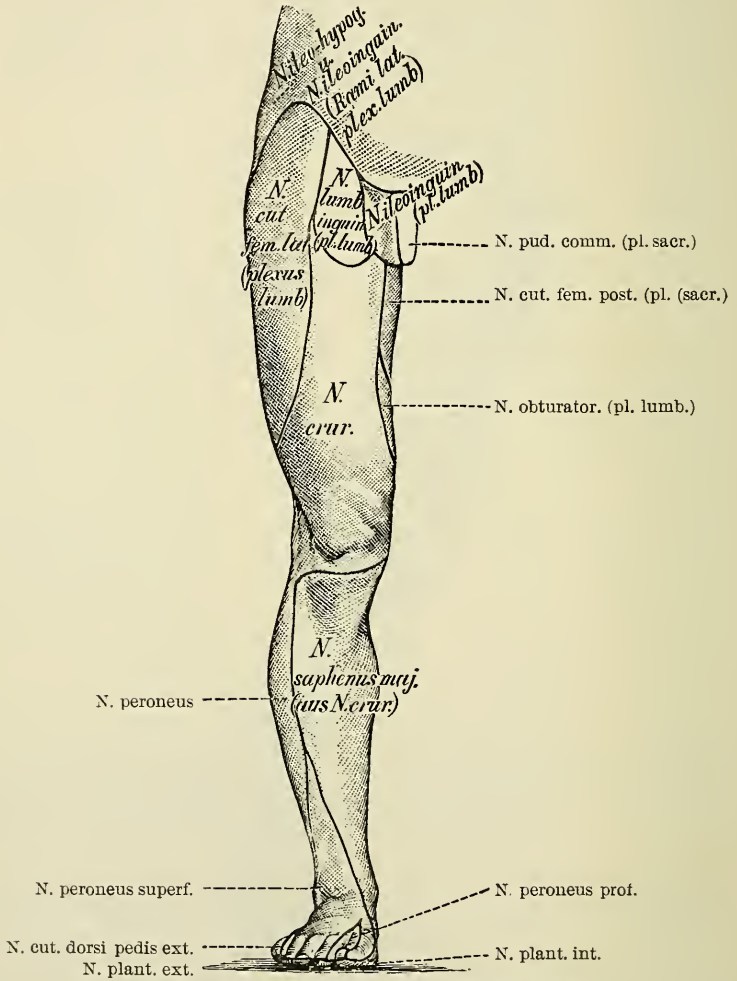
patient to arise, as in the following form, by climbing up his legs. The gait, as a result of the atrophy of the quadriceps, is waddling in character. The disease usually presents itself about puberty.

In the latter type the symptoms are essentially the same, excepting that the first muscles to undergo atrophy are those of the eyelids and

mouth. This form usually commences about the third or fourth year of life.

Pseudo-hypertrophic Muscular Paralysis. The disease usually commences in the muscles of the calves. These become greatly enlarged, hard, and there is great loss of power. Other muscles of the legs are

FIG. 251.

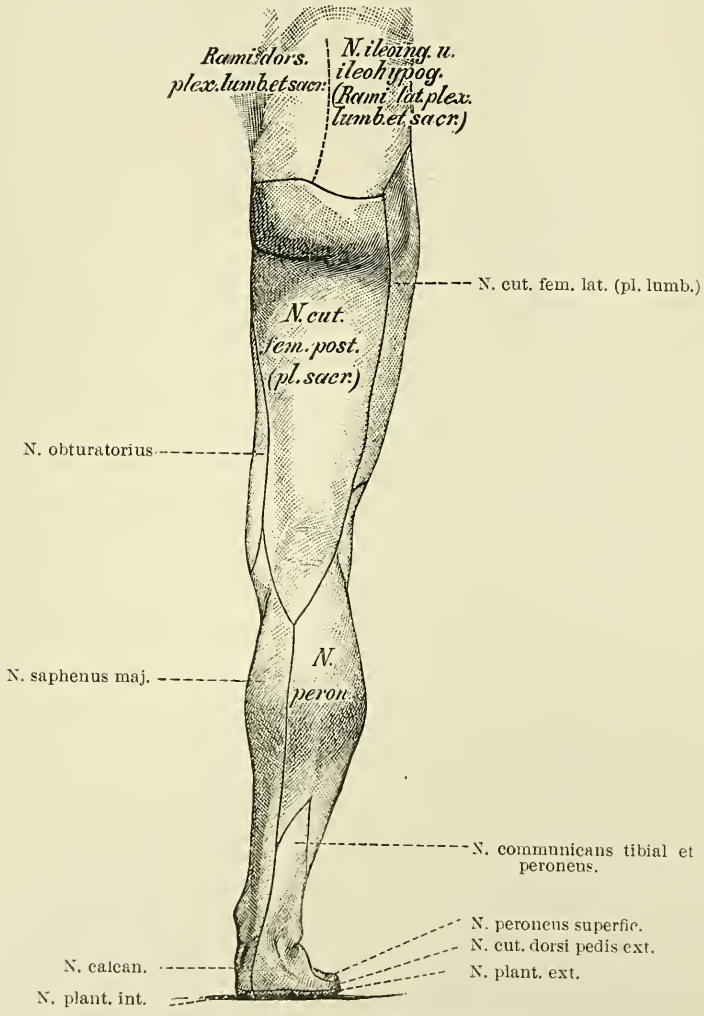


Cutaneous nerves of the anterior surface of the leg. (SAHLI.)

next involved; then those of the back, and perhaps the arms. Not all the muscles that undergo atrophy show a preliminary hypertrophy. The electrical reactions remain normal, and the loss of power is due merely to the atrophy of the true muscle substance. The gait is waddling, and the patient is unable to arise from the ground, except by getting upon the hands and knees and then gradually climbing up

his legs. There is usually lordosis or scoliosis, and occasionally contractures occur, leading to formation of club-foot. In all these three forms of disease the course is slowly progressive.

FIG. 252.



Cutaneous nerves of the posterior surface of the leg. (SAHLI.)

DISEASES CHARACTERIZED BY MOTOR DISTURBANCE, WITH DEGENERATIVE CHANGES IN THE MUSCLES.

Progressive Muscular Atrophy Consecutive to Disease of the Nerves. (The Charcot-Marie-Hoffmann type; the peroneal type of Gowers.) The first muscles affected are those of the feet and hands, usually in the former, the peronei, the extensors of the toes, and the small muscles

of the foot; in the latter, the interossei and the muscles of the thenar and the hypothenar eminences. The affected muscles show distinct fibrillary twitchings and usually the characteristic reactions of degeneration to the electrical current. These reactions of degeneration are also present in the nerves. There is usually a coarse, irregular tremor, and the atrophy of some of the muscles with contractures of others give rise to various deformities, such as the *ape-hand*, the *main en griffe*, or, if the foot is first affected, to foot-drop. Later the foot assumes a position of equino valgus or varus. In this disease there is sometimes involvement of the sensory fibres, and the patients may complain of slight paræsthesia or even of pain. Hypæsthesia is also occasionally present. In a form of this disease described by Dejerine under the title of *Infantile Hypertrophic and Progressive Interstitial Neuritis*, there are, in addition to the above changes, the symptoms of locomotor ataxia—that is, Romberg's symptom—lancinating pains, atactic gait, and even disturbance of the pupillary reflexes. The nerve-trunks become enlarged and can be felt beneath the skin.

Progressive Spinal Muscular Atrophy. (Type of Duchenne-Aran.) The disease commences usually in the muscles of the hand, particularly in those of the thenar eminences, giving rise to the formation of the ape-hand. The interosseous spaces become deeper, the fingers become gradually weakened, and ultimately become fixed in a semi-flexed condition—incomplete *main en griffe*. The muscles show fibrillary twitching and give the reactions of degeneration to the electrical current. Usually the process is bilateral. As the disease progresses it next involves the muscles of the shoulder, especially the deltoids, and later the muscles of the upper arm, and then of the forearm. Finally, the muscles of the back become involved, and even the lower extremities. Sensory disturbances are never present. The emaciation is extreme, but total paralysis occurs only very late in the disease.

Acute Anterior Poliomyelitis. This is really an infectious disease, commencing with chills and fever and characterized by the rapid appearance of flaccid paralysis in one or more limbs. The onset is usually sudden, and the paralysis may occur before the development of the general symptoms. The legs are more frequently involved than the arms; the muscles are usually affected in functionally similar groups, such, for example, as the flexors of the upper arm, and then very rapidly begin to undergo contractures. These produce deformities, particularly various forms of club-foot, scoliosis or lordosis, and contractures of the hand. The disease usually occurs in children, and the affected extremity does not grow as rapidly as the other. Occasionally adults are attacked. Sensory disturbances are absent, the reflexes are abolished, and the electrical reactions are those of degeneration. In the very early stage pains, usually radiating from some point in the back, have been noted in a few instances. Ordinarily, the paralysis is more extensive at first than later — that is to say, many of the muscles involved recover completely.

Chronic Anterior Poliomyelitis. This is characterized by the slow development of paralysis in one or more groups of muscles or extremities of the body. The flexors are more likely to be attacked than the

extensors. The muscles show fibrillary twitchings and the reactions of degeneration, and the paralysis is usually flaccid. The process is usually self-limited, but bulbar symptoms may appear and cause death. The disease resembles closely progressive spinal muscular atrophy.

Periodic Paralysis. This is a disease characterized by the occurrence from time to time of paralysis of all four extremities. The paralysis is usually flaccid in type, occurs without pain, and is associated with extraordinary increase in the electrical resistance of the skin. The disease usually occurs in several members of the same family, the paroxysms lasting three or four days.

Diseases Characterized by Disturbance of Motion Occurring without Reference to any Definite Portion of the Cerebral Nervous System.

Chorea (Sydenham's chorea) is characterized by irregular twitching movements affecting various groups of muscles in the body that are usually functionally associated, so that the movements appear to be the result of voluntary innervation. These movements may be generally distributed, or more pronounced on one side than the other, or may even occur only in one part of the body. They may involve the muscles of the face, the arm, the leg, or the muscles of the trunk, particularly the diaphragm, giving rise to an irregular, jerking inspiration. They may vary in severity from slight, almost imperceptible contractions to severe, general convulsive movements in which the violence is so great that bruises or even fractures may occur. As a rule, the affected limbs are slightly weaker, and in some cases this paralysis is very pronounced (*paralytic chorea*). The mind is usually clear, but there may be some irritability of temper. In a few cases with violent movements there is pronounced insomnia and violent delirium (*chorea insaniens*). Speech may be affected either as a result of choreic movements of the lips or on account of psychic disturbance. Associated symptoms are the presence of a heart murmur, irregularity of cardiac action, rheumatic pains in the limbs, which usually disappear as the movements become more pronounced; and, occasionally in the violent form, fever.

Huntington's chorea is characterized by the development, between the ages of twenty and forty, of choreiform movements of moderate degree, associated with gradually progressive dementia. The disease is strictly hereditary, occurring only in the offspring of those who have suffered from it. The twitchings resemble those of chorea, but are rarely violent, and often associated with a slight rigidity. The first mental symptom is usually loss of memory. Later, the patient may have delusions of grandeur or severe melancholia. Usually life is prolonged to an advanced age, the mental symptoms gradually passing into the type of severe senile dementia. A curious condition is the tendency of the patient to avoid society.

Chorea Electrica. There are various varieties of this condition—one occurring in children, characterized by lightning-like contractions of groups of muscles, sometimes those of the trunk or those of the extremities; another, *Dubini's disease*, which appears to be an infectious

process, commences with violent pains in the head, neck, and back, slight fever, and general convulsions. Muscular contractions occur, usually involving all the muscles of the body that are characterized by their frequent recurrence and brief duration. Death is the usual termination.

Paramyoclonus Multiplex. This is a disease, probably hysterical in nature, characterized by lightning-like contractions in groups of muscles, which do not, however, produce movement that would in any way resemble co-ordinated action. Often the patient from time to time emits a peculiar sound resembling a grunt, probably the result of diaphragmatic involvement. The electrical reactions are normal, and the reflexes are sometimes slightly increased.

Habit spasm is characterized by the repetition of some peculiar, unnecessary movement, such as shrugging the shoulders, winking the eye, rubbing the elbow against the side, etc. Emotional disturbances or the presence of bystanders always increase the symptoms.

Saltatoric spasm (jumper's disease, latah) is a hysterical manifestation in which the patient, whenever he or she attempts to stand, is compelled to rise on the toes or even to spring from the ground. Often after such movements the patient falls. The spasm disappears if the patient lies down, but may be produced by pressure upon the soles of the feet.

General Tic (*Maladie de Gilles de la Tourette; maladie der tics convulsifs*). This is a psychical condition characterized by curious movements of the limbs and grimaces and the utterance of words, that have no relation to the evironment, that may be profane or obscene (*coprolalia*), or the imitations of sounds heard (*echolalia*). The patient becomes more or less melancholy, and may even be violently insane.

Paralysis Agitans. This is characterized by a peculiar, fine tremor of the extremities, rigidity of the muscles, disturbance of gait, and gradually progressive paresis. The first symptom noted is usually a slight impairment of agility. As the disease commences in advanced life, this is not regarded with suspicion; but later the immobility of the muscles of the face and the complete loss of facial expression suggests the nature of the case. It will now be found that the patient will have difficulty in rolling over, if lying down, and that there is difficulty in commencing to walk and afterward a tendency to take quick steps (*festination*). The patient, if studied, will be seen to have from time to time a slight movement forward or backward, which, if standing or walking, may cause him to fall in one direction or the other (*propulsion, retropulsion*). Speech is also involved, difficulty in articulation being characterized at first by slight halting and then the rapid utterance of the words. The tremor of the hands is spoken of as pill-roller's tremor (*q. v.*). Tremor of the head is a nodding movement to and fro. There may also be irregular movements of the toes or legs. The tremor is diminished or abolished temporarily by voluntary movement and disappears during sleep.

Tetany is probably an infectious disease characterized by cramp of the muscles of the arms and the persistence of peculiar nervous and mental alterations. The attack usually commences with paræsthesia or pain in the limbs; then the muscles controlling the fingers become stiff.

The flexors gradually contract and draw the fingers and thumb together, the so-called obstetrical hand. This cramp is tonic in character, and may last for several minutes or even for many hours. It is often associated with intense pain During the interval it may be reproduced by prolonged, severe pressure upon the nerve-trunks, particularly the median nerve (Trousseau's sign). The muscles show marked irritability to mechanical stimuli, particularly those of the face, and twitching may be caused by tapping upon the trunk of the facial nerve, upon the malar bone, or over the infraorbital foramen (Chvostek's sign). The muscles show extreme electrical irritability, contract to very weak currents, and in some cases AOTe and COTe have been obtained (Erb's sign). Finally, the patient is extremely sensitive to the induced current (Hoffmann's sign). During the attack, and even during the interval, there is sometimes slight œdema of the face, hands, and feet, and the latter have a tendency to assume a partial equino-varus position. Often there is slight fever.

Occupation Neuroses. These are characterized by the development of pain in the limb employed when the attempt is made to perform some habitual movement. It is most common as a result of writing. The patient notices at first that he becomes more readily fatigued than usual, and there may be dull pains in the joints or in the palm of the hand. The painful sensations may then extend up the arm, often as far as the shoulder. They are rarely severe, but by their persistent, dull character are extremely annoying. The motor symptoms are characterized by a tonic spasm of the muscles employed in grasping the pen, so that it is held too tightly, and often there is difficulty in holding it properly. From time to time the spasmodic condition may increase and cause inaccurate strokes. The writing is usually heavy and often quite illegible. The muscles apparently never degenerate. The electrical reactions are normal or only slightly altered. If the patient learns to write with the left hand, the symptoms of the disease usually develop in it after a short time. Similar symptoms occur in piano-players, violin-players, dairy-maids, telegraphists, and various other persons who are obliged to perform the same movement for long periods.

Thomsen's Disease. This is characterized by the occurrence of tonic spasm as the result of voluntary innervation of the muscles. The patient, upon attempting to make a movement, finds the part rigidly fixed for a longer or shorter interval of time. The spasm then relaxes, the movement can be performed, and does not recur while the muscles are kept active. There are occasionally cramp-like pains in the muscles and a peculiar alteration in the electrical reactions. (See Myotonic Reaction.) The disease is chronic, but subject to exacerbations, particularly as a result of exposure to cold, previous excessive exercise, or emotional disturbance.

Diseases of the Sensory Neuron, with Disturbances of Sensation.

These are generally included under the term *neuralgia*. Neuralgia is a condition characterized by pain of a dull, burning, or shooting

character that occurs in the distribution of some particular sensory nerve or nerves. The pain may be remittent or intermittent. It is exaggerated, as a rule, by external irritation or emotional disturbance. The nerve-trunk is often tender, not only during the attack, but also during the interval. Associated symptoms are often present. The most common are the vasomotor disturbances, the area of distribution of the affected side showing persistent or paroxysmal flushing or occasionally pallor. Secretion of sweat is sometimes increased, and there may be exaggeration of the activity of glands supplied by the nerve. Occasionally there is marked œdema of the skin, and sometimes a herpetic eruption. Very rarely in neuralgia there is local graying of the hair. Motor symptoms may also occur. These consist of spasmodic twitching that may be associated with exacerbations of the pain. Neuralgias due to various general conditions sometimes have a characteristic localization. Thus in diabetes, sciatica occurs; in malaria, supra-orbital neuralgia; in neurasthenia, occipital neuralgia.

Special Forms. *Neuralgia of the Trigeminal Nerve* (tic douloureux). This usually occurs in only one branch of the nerve, and is commonly unilateral. The pain is paroxysmal and very severe, and is often referred by the patient to some supposed source of peripheral irritation, as disease of the nose, carious teeth, etc. It is usually associated with increase in the secretion of various glands, such as the tear glands, the salivary glands, the nasal mucous membrane, etc. Trophic changes are not uncommon. These may vary from herpetic eruptions and graying of the hair to atrophy of the soft parts and even of the bones of the face. Occasionally trophic alterations of the cornea also appear.

Occipital Neuralgia. This involves the occipitalis major nerve, but occasionally the auricularis magnus and the nerves of the neck are also affected. The pain is distributed over the occipital region of the head, and is usually bilateral. The point of greatest tenderness is over the cervical vertebræ, usually slightly to one side of the spinous processes.

Brachial neuralgia is characterized by pain distributed in the arm of the affected side. This may be either persistent or paroxysmal. If the latter, paræsthesiæ in the hand or arm are frequent during the intervals. The points of tenderness are found where the nerves pass over the bones or just behind the clavicle. Occasionally trophic changes are observed.

Intercostal neuralgia is characterized by pain distributed along the course of the intercostal nerves. There are three characteristic tender points—one next to the spinal column, one in the axillary line, and one over the sternum or rectus abdominus. There are usually trophic disturbances in the skin over the affected nerve, characterized by reddening or especially by a herpetic eruption (*herpes zoster*).

Lumbar neuralgia is characterized by pain radiating from the lumbar to the gluteal region. Occasionally the anterior surfaces of the thighs are also involved. The sensitive points are found over the lumbar vertebræ along the edge of the crest of the ilium and over the linea alba.

Crural neuralgia is characterized by pains radiating from the front of the thigh into the feet. Paræsthesiæ are frequently present during the intervals of the attacks.

Sciatica is characterized by pain in the posterior surface of the thigh, often radiating to the feet. It is an exceedingly common form, usually paroxysmal in character, the attacks being preceded by paræsthesiæ. The pain is increased by any movement tending to stretch the nerve, and as a result the patient walks with a peculiar gait, the thigh of the affected side being held fixed and parallel to the body. This sometimes results in a slight curvature of the spine. The nerve is often sensitive through its entire length. The special points of tenderness are found near the posterior superior spine of the ilium, at the lower edge of the gluteus maximus, just outside the tuber ischii, and in the cavity of the knee-joint. The reflexes are usually slightly exaggerated. There is sometimes slight weakness of the muscles and occasionally fibrillary twitchings.

Other forms of neuralgia are *mastodynia*, or irritable breast; *neuralgia* of the phrenic nerve, characterized by deep pain in the thorax and slight dyspnœa; *coccygodynia;* and various neuralgia-like pains in the viscera.

Diseases somewhat similar to neuralgia are *meralgia paræsthetica*, characterized by tingling, burning, or tearing in the area of the distribution of the external cutaneous nerve of the thigh, usually unequally bilateral, and made worse by prolonged exercise, either walking or standing. Frequently there is a tender point just below the anterior superior spine of the ilium. Sensory disturbances in the form of hypæsthesia, hypalgesia, and diminished electro-cutaneous sensibility are very common.

Achroparæsthesiæ are characterized by tingling or pain in the extremities. The affected members are usually tender, and there is hyperæsthesia. Occasionally vasomotor disturbances are present. An allied condition is the symptom known as *tender toes* that occurs in the course of typhoid fever.

Diseases of the Sensory Neuron Characterized by Disturbance of Motion, Sensation, and Trophic Disorders.

Tabes Dorsalis. This is characterized by ataxia, particularly of the lower extremities, lancinating pains in the legs, loss of the knee-jerk, and the Argyll-Robertson pupil. It is divided into three stages: the preataetic, the atactic, and paralytic. The symptoms of the *preatactic* stage frequently commence with disturbance in the nerves affecting the eyeball. There may be paresis of the abducens, giving rise to diplopia; of the levator palpebræ, giving rise to ptosis; or sluggish or absent reaction to light on the part of the pupil, while the reaction of accommodation still persists (Argyll-Robertson pupil). The symptoms in the nerves of the lower extremities are particularly the lancinating pains that are felt in the posterior portion of the thigh. These come on from time to time, and the patient feels as if he has been stabbed. They are more frequent in damp weather, and are frequently confused with rheumatism. The knee-jerk is absent, and the patient may note that it is a little bit more difficult to walk in the dark. The station in the early stage is usually only slightly affected. There is a sense of constriction about the body (girdle pain), and sometimes hypæsthesia of the lower extremi-

ties that may be associated with a slight hyperalgesia in the zone just above it. The patients may also remark that they have slight difficulty in urination and some diminution of sexual potency. The second stage, or the stage of *ataxia*, is characterized by the symptoms of the preceding stage, all of which are now pronounced. In addition the patient exhibits inco-ordination of movement, especially in the lower limbs. Station is so impaired that it is usually impossible for him to stand alone with the eyes closed and the feet together. Walking in the dark is difficult and usually associated with frequent falls. In the daylight, with the aid of the eyes, the patient can usually walk quite well, but lifts the feet higher than usual from the ground, and separates them widely. (See Ataxic Gait.) The inco-ordination is manifested by the difficulty with which the patients perform certain movements, such as touching some object with the tip of the finger—as, for example, the nose, ear—or in bringing the heel of one foot against the knee of the other. There is diminished muscle-tone, and, of course, absolute loss of tendon reflexes, even when reinforced. There are paræsthesiæ, especially in the lower extremities; analgesia in the same situation, or sometimes delay in the conduction of pain. Micturition is sometimes difficult; at others there is incontinence, but insufficiency of the sphincter ani rarely occurs. Impotence is complete. The Argyll-Robertson pupil is present; there is usually myosis, nyctalopia, and occasionally atrophy of the optic nerve. In the latter condition it has been noted that when blindness has fully developed the ataxia becomes less pronounced or may disappear completely. The visceral crises are characterized by attacks of intense pain involving usually the stomach or sometimes affecting the larynx or heart or other viscera. The laryngeal crises are often accompanied by distressing cough and dyspnœa. Trophic changes occur, of which the most common are the arthropathies. These involve particularly the knee, hip, and shoulder-joints. In addition, the patient may have painless falling out of the teeth or rapid softening of them. In certain cases a chronic ulcer develops on the sole of the foot, which usually progresses until it has produced perforation (*mal perforante*). In the *paralytic* stage of ataxia the loss of muscle-tone has reached such an exreme degree that locomotion is impossible. The patients by this time have usually developed cystitis, and death occurs either as a result of exhaustion or of general septicæmia.

The Cervical Type of Tabes Dorsalis. This is characterized by the development of the symptoms chiefly in the arms. The lightning pains are found in the upper extremities, there is loss of the bicipital and tricipital reflexes, and the girdle sensation is usually felt in the upper part of the thorax. The ocular symptoms are the same. The visceral crises are likely to affect the larynx. In this form ataxia in the legs, Romberg's symptom, and the absence of the knee-jerk may not be present until late in the disease.

Friedreich's Ataxia. This is characterized by inco-ordination, loss of knee-jerk, weakness, irregular speech, and slight deformities. The disease commences in youth, and is usually hereditary in character. The first symptom is inco-ordination of the lower limbs. This gradu-

ally becomes more severe, the muscles grow weaker, the flexors more so than the extensors, often giving rise in time to pes equino-varus. The muscles of the back also grow weaker, giving rise to scoliosis; the knee-jerks are absent, the pupillary reflexes remain normal, and intelligence is unaffected. The speech is peculiar, some of the syllables being pronounced readily and others slowly, with a drawl. The gait becomes markedly ataxic, the patients keeping the legs widely separated. In time the paresis and inco-ordination become so severe that walking is impossible. The disease is progressive and usually affects several members of the same family.

The *cerebellar* type of hereditary ataxia differs from the foregoing by the fact that the knee-jerks are exaggerated, and there is occasionally absence of the pupillary reflex to light.

Diseases of the Peripheral Motor and the Sensory Neuron.

These are all characterized by disturbances of motion and sensation, usually associated with more or less severe trophic changes.

Neuritis. Inflammation of the nerves is characterized by pain localized in the nerve affected, tenderness, and perhaps paresis or paralysis of certain groups of muscles. The pain is made more severe if the limb is held in such a position that the nerve is stretched. As it is a true inflammatory condition, there are usually constitutional disturbances, such as fever, malaise, etc. Often the disease is progressive, extending from the peripheral to the more central nerve-trunks. This is spoken of as ascending neuritis. Along the course of the nerve there are often vasomotor and secretory disturbances, or the lesions may be more severe, such as atrophy of the skin, with glossiness, or trophic

Fig. 253.

Alcoholic neuritis. Foot-drop and wrist-drop.

changes in the nails. Multiple neuritis is characterized by the appearance of the symptoms of the disease in a number of nerves at the same time. The nerves of the limbs are far more frequently affected than those of the trunk. The symptoms are modified by the cause. In alcoholic polyneuritis there are usually slight paræsthesiæ of the limbs, with marked paresis of the muscles, particularly the extensors, giving rise to foot-drop and wrist-drop. (See Fig. 253.) The disease usually affects all four extremities. In lead-poisoning the disease is sometimes unilateral, is usually restricted to the arms, and the sensory disturb-

ances are very slight or absent. There is paralysis of the extensor muscles of the arm, which in severe cases goes on to muscular degeneration. Neuritis may also be produced by arsenic. Diphtheritic polyneuritis is usually characterized by paralysis of the muscles of the palate, but occasionally the muscles of the limbs are also involved. In certain of the chronic forms of polyneuritis, instead of loss of power, there is marked loss of co-ordination. This is spoken of as the ataxic variety.

Beri-beri, or *kakke*, is an infectious disease characterized by the symptoms of a peripheral multiple and symmetrical neuritis. The patients usually present general symptoms, as fever and chills, and then complain of a sense of weakness or heaviness in the legs, paræsthesiæ, and diminution of tactile sensation. The electrical examination of the muscles of the legs usually shows the reactions of degeneration. Later, the muscles undergo further degeneration and become paralyzed. There is œdema of the skin, and the anæsthesia becomes more pronounced. Occasionally pain-sense is preserved, giving rise to anæsthesia dolorosa. The paralysis becomes more extensive, and the patient may die as a result of the involvement of the respiratory muscles.

Multiple neuromata sometimes occur very extensively upon the nerves of the skin, at times producing symptoms of multiple pressure upon the nerves—that is, paræsthesiæ, paralyses, or loss of sensation. At other times they produce no symptoms whatever, and can only be recognized by inspection.

Diseases of the Spinal Cord Involving the Central Motor Neurons.

Primary spastic paraplegia is characterized by weakness of the legs without muscular degeneration and with increased reflexes. The disease was formerly supposed to be the result of the involvement of the lateral columns of the cord. The first symptoms are weakness or a feeling of heaviness in the legs; then spontaneous cramps appear. The reflexes are greatly exaggerated, and the muscle tone is so increased, particularly in the extensors of the thigh or knee and foot, that the patient walks with the leg partially extended, dragging the toe along the ground; the arms are rarely involved. The electrical reactions of the muscles are normal. The sphincters are very rarely involved, and sensation is usually unimpaired. If cramps are frequent, however, the muscles may be sore. In children the adductors become stronger than the abductors, and a peculiar, crossed-legged gait is thereby produced.

Amyotrophic Lateral Sclerosis. This is characterized by a spastic paraplegia, with exaggeration of the reflexes and degeneration of the muscles. The symptoms consist of weakness in the legs, which at the same time become stiff. The muscles rapidly atrophy; there are fibrillary twitchings and reactions of degeneration. The arms are usually involved first, the degeneration commencing in the muscles of the hands and giving rise ultimately to the production of various deformities, such

as the claw-hand. The tendon reflexes are greatly exaggerated; there are patellar clonus and ankle clonus. The muscles are greatly weakened, but remain rigid until late in the course of the disease. The sphincters are rarely involved, the pupillary reflexes are normal, and there are no sensory disturbances. Bulbar symptoms—that is, paralysis of the larynx, pharynx, and palate—occur, giving rise to dysphagia, alteration in speech, and frequently causing an inspiration pneumonia.

Multiple Sclerosis. This is a condition that involves the sensory and motor tracts in the spinal cord and occasionally in the brain. The characteristic symptoms are intention tremor, nystagmus, and scanning speech. The patient usually has weakness of the legs, with some tremor and exaggeration of the reflexes. In the arms the same conditions are present, and in the attempt to grasp any object a violent tremor is developed, which continues until the movement has been accomplished. Various areas of anæsthesia are also present, depending largely upon the localization of the lesions. There is usually persistent nystagmus, lateral in character; the speech is slow and drawling, and the patient has a tendency to laugh or weep without provocation. In a large proportion of the cases there is more or less complete atrophy of the optic nerve. Less frequent symptoms are vertigo, occurring in paroxysmal attacks, diminution of intelligence, and occasionally disturbances of the function of the bladder, and in a few cases atrophy and degeneration of the muscles. The disease is usually chronic, but from time to time there are exacerbations. It appears to be frequently associated with hysterical manifestations. In some cases bulbar symptoms appear early and rapidly lead to death.

Hypertrophic Cervical Pachymeningitis. This is characterized by pain in both arms, followed by muscular degeneration commencing in the hands. Later, there may be spastic paraplegia of the legs, with anæsthesia of the body below the affected segment. Occasionally this disease, which is usually due to tuberculous meningitis, may occur in other portions of the spinal cord, giving rise, therefore, to various symptoms.

Acute spinal meningitis is characterized by intense pain in the back, radiating into the legs; rigidity of the spinal column, with opisthotonos; intense hyperæsthesia of the skin of the body, and, if the disease lasts long enough, paralyses. Kernig's symptom—that is, the inability to extend the flexed leg as a result of flexor cramp—is said to occur only in this condition and in cerebral spinal meningitis. The *tâche spinale* occurs also in other conditions.

Syphilitic spinal meningitis produces a great variety of symptoms. There are, however, pains due to pressure upon the posterior roots, girdle pains of the body, and occasionally paralysis of the muscles of the extremities, with atrophy and degeneration. Often, also, the spinal cord is involved, giving rise to the symptoms of pressure or transverse myelitis (*q. v.*) or Brown-Séquard's syndrome (*q. v.*). The sensory symptoms, aside from the pains, consist of hyperæsthesia, hypæsthesia, or anæsthesia. The tendon reflexes of the lower extremities may be lost and reappear, and this by some is supposed to be pathognomonic of the disease.

Diseases Characterized by the Syndrome of Transverse Interruption of the Spinal Cord.

Pott's Disease (caries of the vertebræ). This is characterized by an angular deformity of the spine, spastic paraplegia, and various disturbances of sensation in the body below the level of the lesion. In the earlier stage the only symptoms may be pain in the back, usually radiating around toward the ventral surface. There may be no deformity, but sudden pressure upon the head, jarring of the spine by coming down heavily upon the heels, and pressure over the tender point in the back may elicit sharp pains. In this stage there are usually slight exaggeration of the reflexes and perhaps a slight weakness of the legs. Later, the angular deformity becomes apparent, usually in the form of a sharp projection in the dorsal portion of the spinal column, but it may appear also in the cervical and lumbar region. The weakness of the lower extremities becomes more pronounced, and may give rise to an actual paraplegia. The pains are usually severe, radiate around the trunk, and sometimes affect other portions of the body. Sensation may be slightly impaired. There may be distinct dissociation below the lesion—that is, loss of temperature and pain senses, with preservation of tactile sense—or there may be total anæsthesia. As in myelitis, bed-sores or other trophic changes of the skin are very likely to develop, and the patients suffer severely in general nutrition. In the earlier stages, and more particularly in the stage of recovery, after the deformity has become stationary, ataxia may exist. The reflexes are sometimes greatly exaggerated, and there is often ankle clonus. When the paraplegia has become complete all the reflexes are usually abolished. Girdle sensation is also very common. The course is very variable. At times the destruction of the body of the vertebra is rapid, and the symptoms develop acutely. At others it occurs very slowly, and the symptoms, even after years' duration, may be exceedingly slight. Caries of the upper cervical vertebræ produce pains that involve the neck and the occipital region of the head. The position of the head is peculiar; it is drawn slightly forward and carried very rigidly, and the chin is elevated. These patients may sometimes die suddenly as a result of pressure by the odontoid process on the medulla.

Tumors of the Membranes. The symptoms of this condition are extremely variable, according to the location, nature, and extent of the growth. Occasionally deformities occur as a result of pressure upon the arches of the vertebræ. Paraplegia usually develops, sometimes very suddenly, sometimes gradually. There is usually exaggeration of the reflexes and ankle clonus; but this in time may disappear, or may never occur if the tumor is situated in the lumbar region. When the posterior roots are pressed upon there are root pains and the girdle sensation. Sensory disturbances are more or less complete according to the degree of destruction that has occurred in the spinal cord. Dissociation of sensation rarely occurs, but anæsthesia is very common. After complete destruction of the spinal cord at any point trophic changes occur.

Chronic Internal Meningitis. This is usually characterized by pain that radiates into various portions of the body, particularly the limbs,

and by more or less hyperæsthesia. The motor symptoms consist of tremors, spasms, and occasionally, when the anterior roots are involved, paralyses, with muscular degeneration. In the milder forms the only motor symptoms may be inco-ordination of movement. Herpetic eruptions along the course of the nerves arising from the involved posterior roots are quite common.

Acute Myelitis. There are a number of varieties of this condition, the most common and typical being transverse myelitis. It is an acute inflammatory disease associated with constitutional disturbance—that is, chills, fever, and malaise, and is occasionally ushered in with a convulsion. The symptoms are those of transverse lesion of the spinal cord. Ordinarily the dorsal part is affected; and there are, therefore, in the earlier stages weakness and paræsthesiæ of the legs, and perhaps a girdle sensation and hyperæsthesia over the spine, the zone supplied by the involved segment. In the course of a few days or hours the weakness of the legs increases until there is complete paraplegia. The tone of the muscles is enormously exaggerated, the knee-jerks are increased, and there is both patellar and ankle clonus and often Sinkler's toe-jerk. The limbs are usually spastic and kept in a position of extension. From time to time the muscles give violent twitches. There is complete anæsthesia up to the horizontal line surrounding the trunk, at which point there is girdle sensation, and above it there is a zone of hyperæsthesia. The muscles supplied by the affected segment atrophy and give reactions of degenerations. Those in the region below maintain their nutrition for a considerable time. There is difficulty in micturition, usually paralysis of the bladder, and finally overflow from retention. The urine becomes alkaline, cystitis develops very rapidly, and is often followed by extensive sloughing of the surrounding parts. Bed-sores occur early and extend deeply. Trophic lesions also occur in the legs, the skin becomes thin and glazed, and the toe-nails are brittle. Even arthropathies have occasionally been observed. After the acute stage has passed more or less improvement may occur, characterized by gradual return of power in the legs and partial recovery of sensation.

Acute Focal Myelitis. This gives rise to only part of the symptoms described above, depending upon the tracts involved by the process and the various nuclei that have been destroyed. There is, therefore, usually a monoplegia, associated with exaggeration of the reflexes and irregular areas of anæsthesia, or, if the focus be in the arm or the leg centre, diminution or loss of the reflexes and degeneration of the muscles.

Disseminated myelitis gives rise to a complicated group of symptoms, according to the number, situation, and extent of the lesions. It resembles perhaps most closely transverse myelitis (*q. v.*).

Chronic myelitis is distinguished from the acute form by the more gradual development of the symptoms. The patient first notices weakness of the legs, perhaps characterized from time to time by complete transient loss of power (*giving way of the legs*). If the reflexes are examined at this time, they will be found slightly exaggerated; later they become very markedly increased, and ankle clonus develops. The patient also complains, in the early stages, of paræsthesiæ in the limbs

that may involve the arms as well as the legs, and sometimes the trunk. A girdle sensation is also frequently present. Finally, muscular atrophies occur, and even severe trophic disturbances; the picture ultimately resembling that of acute myelitis.

Pressure upon the spinal cord may be produced either by injury to the vertebral column or by growths in or hemorrhages into the membranes. The symptoms are those of transverse lesion. If due to tumor, they develop very slowly; if due to traumatism, as a rule, very rapidly. There is weakness or paralysis of the legs, with increase of the muscle-tone and exaggeration of the reflexes. Ankle clonus is almost invariably present. The pains are usually due to pressure upon the posterior roots, and are paroxysmal and lightning-like in character. Girdle sensation is also present. The muscles supplied by the segments of the cord involved undergo degenerative atrophy.

Landry's Paralysis. This is characterized by progressive paralysis of the legs, arms, and muscles of the throat, leading ultimately to death. The first symptoms noted are weakness of the legs, which may involve both, or at first only one. This gradually ascends, and at the same time the patient notices paræsthetic sensations. There are, however, few or no objective sensory disturbances excepting occasionally a slight hyperæsthesia. The reflexes are lost, the muscles are without tone, and the paralysis is, therefore, flaccid. Electrical changes do not occur, or only in very chronic cases. The paralysis gradually ascends, involving the muscles of the abdomen, the thorax, and arms. When the thorax is involved the patient usually has rapid respiration, and complains of dyspnœa. Later there are symptoms of bulbar involvement, difficulty in deglutition, and interference with speech. The diaphragm becomes paralyzed, and the patients die as a result of exhaustion. The intelligence remains normal throughout the disease; there is never loss of consciousness and there is no disturbance of the function of the bladder or rectum. Fever does not occur.

Hemorrhage into the Cord (spinal apoplexy). This is characterized by the sudden interruption of the functions of the cord at a certain level. There is usually, at the time the hemorrhage occurs, severe pain, then rapidly developing paralysis of the legs, which may be flaccid if the lumbar region is involved, or spastic if the lesion is higher up. Hæmatomyelia into the cervical region may cause paralysis of the arms, but death usually occurs suddenly. The sensory disturbances are irregular in character. At times there is dissociation of sensation, more frequently complete anæsthesia up to the level of the hemorrhage. The patient has no fever, consciousness is not disturbed, but there is interference with the functions of the bladder and rectum. Occasionally the hemorrhage involves particularly one side of the cord or only one-half of the gray matter, producing the syndrome of Brown-Séquard (*q. v.*). The diagnosis can frequently be made from the subsequent course of the case. If death does not occur, rapid improvement is usually the rule. The sphincters regain their functions, power returns in the limbs, and ultimately the patient may recover completely. In some cases, however, the recovery, although pronounced, is only partial.

Syringomyelia (cavity in the spinal cord). This is characterized by a group of symptoms whose occurrence together is almost pathognomonic. First, dissociation of sensation; pain and temperature senses are lost; tactile and muscle senses are retained. Second, degenerative atrophy of the muscles, associated with fibrillary twitchings and alteration of the electrical reactions. Third, trophic lesions which may involve the skin, particularly that of the fingers or the joints. The disease appears to develop with extreme slowness. The earliest symptoms may be the occurrence of painless whitlows—that is, inflammation around the finger-nail, with perhaps the ultimate destruction of the nail itself. These may recur in one finger after another for several years and without the presence of any other symptoms, excepting perhaps a slight disturbance of sensation in the fingers. Later, muscular atrophies appear. These involve particularly the muscles of the shoulder or the hand. In the latter situation they may give rise to the appearance that occurs in progressive spinal muscular atrophy. At the same time the sensory disturbances become more pronounced, gradually ascending the arm and perhaps involving the trunk. The upper border forms a horizontal line about the body—that is, the alterations are segmental in type. The trophic changes may then assume a more severe form, giving rise to deep, painless ulcerations in the fingers, and perhaps loss of the terminal phalanges. For a long time the symptoms may remain almost exclusively unilateral, and it is rare for the two sides to be equally affected. The motor symptoms, aside from the weakness resulting from the muscular atrophy, consist of weakness of the legs with exaggeration of the reflexes—that is, spastic paraparesis. At times the lower portion of the cord is particularly affected, and then the sensory and trophic changes are found in the legs. Station may be slightly altered in the latter stages of the disease, but this is by no means a characteristic symptom. Ultimately the patient develops scoliosis, trophic changes affect other parts than the hands, giving rise to arthropathies, or to a form of dry arthritis with absorption of the bone. There may be vasomotor disturbances, and in some cases inequality of the pupils. The intellect is undisturbed. The patients ordinarily die as a result of exhaustion or pulmonary involvement, but occasionally in the latter stages of the disease bulbar symptoms occur.

Morvan's Disease. This is characterized by the appearance of painless whitlows in the fingers, sometimes associated with deep ulcerations of the soft parts. There are usually sensory disturbances similar to those found in syringomyelia, with the addition of tactile anæsthesia, but muscular atrophy rarely exists. The disease is exceedingly chronic. It is possibly only a variety of syringomyelia.

Traumatism of the Spinal Cord. This may either produce destruction, partial or complete, of the tissue of the cord itself, giving rise to the syndrome of transverse interruption, or else give rise to a group of indefinite motor, sensory, and mental disturbances that have been grouped under the term traumatic neuroses. (See Hysteria.) The symptoms, the result of organic lesion, may come on gradually or immediately. They are similar to those produced by pressure upon the cord.

Diseases of the Brain Characterized by General Symptoms and Sensory and Motor Disturbances.

Diseases Characterized by Mental, Motor, Sensory, and Sometimes Trophic Disorders.

External pachymeningitis is a rare condition, usually secondary to traumatism or abscess, characterized by fever, headache, often sharply localized, and convulsions. Frequently the symptoms are masked. If there is much thickening of the membrane, evidence of focal disease in the form of paralyses or convulsions may be present. *Hæmatoma* of the dura mater is a condition usually occurring in cases of chronic disease. There may be slight fever and headache without other symptoms. In some cases, however, the onset is sudden and apoplectiform in type. The patients develop hemiplegia, unconsciousness, and occasionally unilateral convulsions.

Internal or Leptomeningitis. The symptoms vary according to the nature of the process, its localization, and extent. The patient may for a few days preceding an attack complain of malaise and headache, then there is often a chill followed by fever, convulsions, and delirium. The headache becomes more intense, and frequently there is vomiting, sometimes without associated nausea. The headache is usually severe, and often localized to the frontal or occipital regions; occasionally, however, it is more general. From time to time there are acute exacerbations, causing the patient to cry out—the hydrocephalic cry. The skin is hyperæsthetic; all the sensory nerves have their functions increased; there is photophobia and inability to tolerate noises. Frequently there is paresis of the vasomotors of the skin, so that localized cutaneous irritation, such as may be produced by drawing the end of a blunt object across the surface, gives rise to a persistent red mark (*tâche cérébrale*). The patient usually lies with the head drawn far back and the muscles of the neck tense and rigid. This, however, occurs only when the cervical portion of the spinal cord is also involved. It is an exceedingly important and an almost pathognomonic symptom. Any attempt to straighten the head causes intense pain. Examination of the eye-grounds usually shows intense congestion and more or less perineuritis. Sometimes there is very distinct choked disk. The pupils are often unequal, and strabismus and even nystagmus frequently occur. Paralysis of any of the cranial nerves indicates that the process is chiefly localized at the base, as in tuberculous meningitis. Paralysis of the oculomotor or some of its branches is exceedingly common. The facial nerve may also be paretic. The tendon reflexes are usually somewhat exaggerated, muscular tone is increased, and occasionally there is distinct monoplegia or hemiplegia. Fever, headache, and delirium usually persist throughout the course of the disease; and the former is often very high. The different forms of meningitis are often difficult to discriminate. By means of Quincke's lumbar puncture it is sometimes possible to make a bacteriological diagnosis from the fluid withdrawn. Meningitis due to certain pyogenic micro-organisms, such as the pneumococcus, staphylococcus, etc., may be suspected; when the

fever is high there is marked retraction of the head, indicating spinal involvement, and the course is steadily progressive to death. Some other disease may often be associated with the meningitic symptoms, or it may have occurred previously, as pneumonia, typhoid fever, etc. Epidemic cerebro-spinal meningitis may simulate the symptoms of purulent meningitis exactly. In some cases, however, the course is more prolonged, and even when the termination is fatal there is apt to be a remission of longer or shorter duration. Tuberculous meningitis is usually characterized by the presence of paralyses of some of the cranial nerves, particularly those of the eye muscles, and the absence of symptoms of spinal involvement. This disease may run an exceedingly slow course, and the diagnosis is often for a time impossible. *Kernig's sign* is said to be pathognomonic of meningitis. It consists of the inability of the patient to straighten the leg when the thigh has been flexed upon the abdomen and the leg upon the thigh.

Cerebral Hemorrhage (apoplexy). This is characterized by a great variety of symptoms, depending largely upon the location of the lesion. They may be divided into those of the attack and those that are permanent. The symptoms of the attack consist of prodromata—that is, headache, tendency to vertigo, a sense of fulness in the head, roaring in the ears, and perhaps some thickness of speech. These may pass off without an attack or may lead directly to it. The attack itself is usually characterized by the sudden occurrence of complete unconsciousness. The patient falls to the ground, and there is at first a temporary pallor. This is succeeded by flushing of the face, which may become almost purple. The pulse is full and bounding and with difficulty compressible. The breathing is stertorous, the eyes are partially opened; the pupils are usually contracted and often unequal. Often there may be vomiting, or involuntary micturition or defecation. The limbs remain completely paralyzed, or in some cases there are unilateral convulsions. If, as is commonly the case, the hemorrhage has involved the motor tract, there is complete flaccid paralysis of one side, with, however, increased reflexes. If death does not occur in the course of the first twenty-four hours, the patient usually begins to show signs of consciousness, and may be aroused from his comatose condition by sharp questioning. The patient then may go into a still more deeply comatose condition, with rise of temperature, followed by death, or there may be no further indications of hemorrhage, and recovery may set in. As a rule, in those cases in which the prognosis is favorable no rise of temperature occurs. It may now be found that the patient has hemianopsia, usually the visual fields on the same side of the lesion being blinded. Conjugate deviation may or may not have existed from the first, the patient ordinarily looking toward the sound side. If the speech centre has been involved, there is absolute aphasia; but even when it is not directly affected partial aphasia is very common. The hemiplegic limbs remain paralyzed; the others regain their power. It is now necessary to determine the extent of the damage and to locate as nearly as possible the situation of the lesion. Complete hemiplegia may involve the lower branch of the facial, the arm, and the leg. The upper branch of the facial and the muscles of the trunk commonly

escape, although the former may show slight paresis. Sensory disturbances may or may not be present. There is sometimes loss of all forms of sensation and sometimes disturbance of only the tactile or the muscular sense. Occasionally when tactile sense is preserved there may be loss of the stereognostic sense. Complete hemiplegia with disturbance of sensation almost invariably indicates destruction of the internal capsule upon the opposite side. Motor disturbances in the form of clonic convulsions may also occur in the paralyzed limbs, and occasionally, probably as the result of a double lesion, in the limbs of the sound side. They are commonly the result of cortical lesion, irritating in character, either infarction, or else some growth pressing upon and involving the cortex. As the case progresses there is usually more or less return of motor power and almost complete return of sensation. This may, however, be exceedingly gradual, several weeks elapsing before the sensory disturbances have entirely disappeared. The muscles that remain permanently paralyzed gradually atrophy, but nearly always give normal qualitative electrical reactions until the muscular substance disappears, leaving contracted fibrous tissue. The muscles themselves may show early contractions, the flexors ordinarily overcoming the extensors. Repeated attacks of apoplexy are by no means uncommon, and the double lesions thus produced may give rise to very complex symptom-groups. (See, also, Cerebral Localization and Aphasia.)

Cerebral Embolism and Thrombosis. This is a condition characterized by symptoms very similar to those of cerebral hemorrhage. Prodromal symptoms, in the form of headache, vertigo, weakness, and malaise, are often present. At times there also may be slight impairment of speech, or the patient may be dull and apathetic. The attack usually comes on more gradually than hemorrhage, although this is not invariably the case. In some instances consciousness is not entirely lost, and as a result the hemiplegia may develop before the coma. When unconsciousness does occur there is usually less congestion of the face and not such marked evidence of increased arterial tension as we find in hemorrhage. Among the other general symptoms may be mentioned convulsions, vomiting, and occasionally delirium. The permanent symptoms resemble exactly those produced by hemorrhage, but recovery is usually more rapid and more complete than in the former condition. Apoplexy occurring in children differs from that occurring in adults only by the fact that the initial symptoms are more severe, and the convulsions are frequent and may be repeated. The permanent symptoms differ slightly, inasmuch as aphasia rarely persists. The paralysis may be partial, and may in some instances be replaced by athetoid movements. Sensation is rarely impaired.

Bulbar paralysis is a disease of the peripheral motor neurons arising in the medulla. It is characterized by the degeneration of the muscles of the lips, tongue, and pharynx. The course is slowly progressive. The earliest symptom is dysarthria, then difficulty in swallowing, chewing, and phonation. The face becomes expressionless, the mouth remains open, saliva dribbles from it, and occasionally the eyelids are involved and the eye remains open (logophthalmus). The cardiac action and respiration may be rapid. Death usually occurs as a result

of inspiration pneumonia, or exhaustion. In the variety known as *asthenic bulbar paralysis* there may be long remissions or even permanent recovery.

Encephalitis. This is a condition that rarely can be diagnosed during life. It may be suspected, however, if, in the course of some other acute infectious disease, the patient develops intense headache, severe delirium, and perhaps local palsies. There may be general exaggeration of all the reflexes, with ankle clonus, and usually hyperæsthesia of the skin, and exaltation of the special senses. Examination of the eye-grounds usually fails to reveal optic neuritis.

Abscess of the Brain. This is a local disease, giving rise to local and general symptoms. General disturbances are chiefly fever, chills, leukocytosis, headache, and delirium. The symptoms of focal disease depend, of course, upon the location of the abscess. The commonest seat is in the temporo-sphenoidal lobe, as a result of infection following ear disease. This often gives rise to mind-blindness or amnesia. Sometimes there are no general symptoms if the abscess is located in the blind regions of the brain. The focal symptoms may not be manifest until rupture has occurred. This often gives rise to an epileptiform attack.

Tumors of the Brain. Like the preceding lesion, these give rise to two groups of symptoms: general, which are merely those of increased intracranial pressure; or local, due to the involvement of centre and tracts. The general symptoms of brain tumor are (1) headache. This is usually very severe, of a boring character, and subject to exacerbations; (2) vomiting. This is paroxysmal, and often occurs without nausea; (3) papillitis. It usually occurs early, is intense, and often leads rapidly to blindness. The local symptoms are, of course, numerous. Tumors in the frontal lobe give rise to none, or at most to some disturbance of intelligence and perhaps a tendency to make puns. Tumors in the motor region may cause irritative or destructive changes in the tissue. Irritation is manifested by local spasms, which may or may not be succeeded by general convulsions (Jacksonian epilepsy). Paralytic lesions are those of monoplegia or hemiplegia. Tumors in the parietal lobes may cause interference with the muscle sense or some disturbance of vision or speech centres, according to their situation. Tumors in the occipital lobes usually cause mind-blindness—that is, inability to recognize objects, and preservation of the pupillary reflexes. Tumors in the different fossa of the skull often give rise to symptoms dependent upon pressure upon the cranial nerves. In the anterior fossa there may be loss of the power to smell upon one side. In the middle fossa the nerves chiefly affected are the optic, giving rise to unilateral blindness, or, if the tumor involve the chiasm, to bitemporal hemianopsia; the oculomotor nerves, the abducens and the pathetic, giving rise to more or less complete ophthalmoplegia. Tumors in the posterior fossa commonly involve the facial and auditory nerve, and it is said that facial paralysis with nerve-deafness on the same side is characteristic of tumor in this situation. The hypoglossal nerve may also be involved. Tumors may, of course, grow slowly, rapidly, or cease to increase in size, and the symptoms

show a corresponding rate of development. In rapidly growing tumors apoplectiform attacks are frequent, but a certain amount of compensation occurs, and remissions are not uncommon. In slowly growing tumors the symptoms may remain apparently stationary for long periods. Tumors are sometimes entirely latent, and are simply discovered accidentally at the autopsy.

Sclerosis of the Brain. This is usually a diffuse or a multiple lesion that gives rise to a great variety of symptoms, more or less indefinite in character. Ordinarily the lesion is congenital, or develops shortly after birth. The patient remains an imbecile or an idiot, and soon develops epileptic convulsions. If the sclerosis is more pronounced on one side than the other there is usually a tendency to fall toward the opposite side. There may be arrest in development in these limbs, and more or less muscular paralysis. Occasionally, apparently as a result of fœtal thrombosis or embolism, the sclerosis may be limited to one portion of the brain or even to one hemisphere. In this case there is always arrest in the growth of the opposite side of the body.

Hydrocephalus (chronic infantile form). This is characterized by an extraordinary alteration in the contour of the head, which becomes greatly enlarged and globular in shape, while the face remains small and infantile in appearance. The symptoms are sometimes exceedingly pronounced; at other times entirely absent. Persons with a moderate degree of hydrocephalus have displayed through life a normal intelligence. In other cases the head is heavy and the muscles of the neck unable to support it. The child is an imbecile or an idiot, and epileptic convulsions are very common. Occasionally ocular symptoms may be present. These consist of ptosis, strabismus, or nystagmus, and sometimes of atrophy of the optic nerve, and blindness.

Acute Delirium. This is a disease characterized by prodromata and a stage of excitation, and usually terminates in death. The prodromata consist of disturbances of the general health, loss of appetite, and insomnia. The patient is restless, anxious, and may show diminution of intelligence, and become more or less violent. He then rapidly passes into the stage of excitation, is restless, noisy, and frequently homicidal, shouting disconnected words or sentences, singing or shrieking. Sometimes there are delusions of persecution, and he attempts to escape. In addition, there are the symptoms of the so-called typhoid state, high fever, profound prostration, dry tongue, and rapid and weak pulse. The patient refuses all food, is continually active, and emaciates very rapidly. Among the objective symptoms are increase of the reflexes, narrowing of the pupils, and hyperæsthesia, with more or less hypalgesia. From this stage the patient passes into a state of collapse, lies in a condition of muttering delirium, with carphology, and usually dies from exhaustion.

General paralysis of the insane is a form of progressive dementia characterized by delusions of grandeur or states of depression associated with exacerbations of maniacal character. There are, in addition, weakness and tremors of the muscles of the face, paresis of the extremities, the Argyll-Robertson pupil, and peculiar disturbances of speech. It is usual to recognize three stages. The prodromal stage, character-

ized by irritability or sometimes by depression; diminution or loss of the moral sense; impaired judgment, particularly in business affairs; and a tendency to extravagance and dissipation. Frequently symptoms associated with degeneration, such as intolerance for alcohol, intense egotism, etc., appear. The sexual function in this stage is often greatly increased. Memory fails and the intellectual capacity is considerably diminished. There are often slight disturbances of speech, and sometimes paralytic pupils. Frequently there is insomnia and occasional attacks of migraine. In the second stage, which usually develops gradually, the attacks of migraine are replaced by apoplectic or epileptic attacks or by distinct maniacal conditions; memory is greatly impaired, the intellect is considerably disturbed, the patient becoming unable to do easy mathematical problems, to comprehend his environment, or to sustain a simple conversation. Usually there are delusions of grandeur, the patient believing himself rich, beautiful, successful, intelligent, and reiterating constantly his advantages, although from time to time there will be states of depression and partial recognition of the failure of power. In other cases, however, particularly chronic alcoholics, there is distinct melancholia; the patient is hypochondriacal, or may have delusions of persecution, or a sense of misfortune. The disturbances of speech are characteristic; the most common is the omission of syllables. This may best be tested by asking the patient to repeat certain words, particularly those containing a number of r's and l's, as "third riding artillery brigade," "truly rural," etc. There is marked tremor of the lips and of the tongue, producing a sort of ataxia in the speech, with the disturbance of the formation of nearly all the sounds. The pupillary changes are similar to those described in the prodromal stage, but usually are more pronounced. The extremities are weak, and often exhibit distinct tremors. Finally, the patient becomes completely demented, usually lies quietly and placidly in bed, or occasionally mutters unintelligible sounds. Sensation, either as a result of impaired perception or because of degenerative changes in the peripheral nervous system or the spinal cord, becomes greatly impaired, particularly the pain-sense. The patient is unable to stand, and has involuntary or rather unperceived micturition and defecation, and frequently develops bed-sores or cystitis. A curious and quite common symptom is the gnashing of the teeth, which in some cases is almost persistent. Death usually occurs from exhaustion. Among the less frequent symptoms are a curious unsteadiness of gait, exaggeration of the reflexes, rapid diminution in weight, particularly in the last two stages.

Epilepsy. This is a condition characterized by attacks of clonic convulsions, associated with loss of consciousness and usually some impairment of intelligence. In the characteristic epileptic fit we can usually distinguish three stages: the prodroma, the attack, and the postepileptic stage. In the prodromal stage aurae are frequently present. These may be of the most varying character. A patient may either have a curious sensation in the epigastrium, paraesthesiæ in a limb, and the subjective sensation of movement, or disturbance of the special senses, particularly an unpleasant odor or a whirring sound. Sometimes the sensations are painful or distressing, as a sense of con-

striction about the throat. At other times there is giddiness, vertigo, or nausea, or the recurrence of some particular idea. Occasionally the auræ consist of some imperative movement, such as whirling about, running, or jumping. At the commencement of the attack there is usually a cry—the epileptic cry. Ordinarily this is a curious sort of gasping, due to the forcible contraction of the thorax and partial closure of the glottis. In some cases, however, it may be a loud shriek. The patient then falls to the ground, and the convulsive movements commence. These are rarely of equal vigor on both sides; the head and eyes show conjugate deviation; the face is bluish and pallid; the mouth is filled with frothy fluid, which is often blood-stained, on account of biting the tongue; the limbs may be extended or flexed in tonic contraction. This is soon replaced by a violent to-and-fro tremor. The patient is completely unconscious, and may, in falling, cause himself serious injury. There is no conjunctival reflex, the pupils are widely dilated; frequently the urine is passed during the attack, and there is occasionally profuse sweating. Toward the end the convulsions become less frequent. Respiration is re-established; at first irregular, then gradually it becomes more and more steady. The cyanosis disappears, and the patient usually passes into a profound sleep. This may last several hours, and he then awakes, feeling dull and fatigued, but otherwise normal. At other times, immediately after the attack, there is vomiting or nausea, and sometimes a feeling of excessive hunger. He may become maniacal, usually with homicidal mania, or the postepileptic stage may be manifested by nothing more serious than some imperative movement, such as running or shouting. The convulsive stage may be replaced by purely sensory phenomena, without complete loss of consciousness, or there may be merely a fine tremor, or the patient may simply run or be otherwise violent, while wholly unconscious.

Petit Mal. In this condition the loss of consciousness is so transitory and the motor symptoms are so slight that its nature often escapes detection. The patient, if talking, will suddenly stop for a moment; there is a peculiar rigidity of the expression and perhaps slight swaying. This will disappear almost immediately, and the patient will resume the conversation. Sometimes after these attacks there will be a feeling of drowsiness for a short period. Auræ may be present in the form of giddiness or twitching of the limb. The attack may also occasionally be ushered in with a scream or a peculiar gasping expiration. Immediately after the attack automatic movements may be performed. Attacks of *petit mal* often occur during sleep, and the only symptoms then that point to the existence of the disease are a feeling of heaviness in the morning, perhaps a sore and bitten tongue, and nocturnal enuresis.

Focal epilepsy (Jacksonian epilepsy). This form resembles general epilepsy, with the difference that the motor or the sensory disturbances always commence in the same part of the body, and from this part gradually extend until they become general. Thus, the thumb may first be affected, showing a tonic and then a clonic spasm; then the hand, the arm, the whole of that side, or both sides; or the disturbance may commence in the foot. The disease almost invariably indicates the existence of a focal lesion in the brain.

General Symptoms in Epilepsy. Epileptics are usually dull, apathetic, having a tendency to excess in eating. An excess of indican is often present in the urine. Often there is distinct mental impairment, or, when the disease occurs early in life, there may be congenital imbecility or idiocy. The temper of epileptics is usually irritable, and they are likely to commit acts of violence.

Migraine (hemicrania). This is a disease characterized by paroxysmal attacks of headache associated with nausea and vomiting, and frequently with disturbances of the special senses. The attacks are usually followed by prolonged sleep. The headache is peculiar, in that it commences slowly as a dull but severe pain that gradually increases in intensity, with occasional exacerbations or throbbing, and is limited to one side of the head. Occasionally, however, it is bilateral, but is then usually unequal. At the same time the patient experiences a sensation of intense nausea that may be followed by vomiting. The special senses are affected in various ways. There may be photophobia, hyperacusis, and occasionally the appearance of peculiar scotomata, which commence as a bright spot that spreads, the outer edge being of an irregular, jagged character, and finally disappears at the periphery of the field of vision. New lines constantly form at the centre, and follow those first appearing. Sometimes the patient complains of dimness of vision, and this may affect only part of the visual field. Occasionally there is temporary aphasia, particularly if the pain occurs in the left side of the head. In addition, the patients may observe vasomotor symptoms, paræsthesia, or occasionally stiffness or spasms in the limb. The paroxysm usually terminates in sleep, which may be prolonged, and when the patient awakens all symptoms have disappeared. Sometimes there is a severe attack of polyuria.

Menière's Disease. This is characterized by attacks of vertigo, associated with nausea. The attack usually begins with tinnitus, then intense vertigo, which may come on so suddenly that the patient falls to the ground, or else he is obliged to lie down, and remain in this position until the attack is over.

Hysteria is a disease due to disturbance of the self-control, producing a curious complex of symptoms that appear to be the result of imitation or of a desire to attract attention or sympathy, associated with certain disturbances of the special senses and of sensation. The psychical symptoms are a certain tendency to self-consciousness, so that the patient is anxious to describe his or her sufferings to surrounding persons; is in the habit of performing ludicrous or startling acts for the purpose of attracting attention; is emotional, weeping or laughing readily, and is often irritable and suspicious. Among the *sensory symptoms* are areas of tactile anæsthesia or analgesia. These may involve exactly one-half of the body, including the accessible mucous membranes, or they may be symmetrical in distribution on both sides of the median line, and often form geometrical figures. These are not the result apparently of simulation on the part of the patient, because they remain unchanged for a number of days. Tenderness—that is, hyperalgesia—may be present over the ovaries and the spine. The areas of anæsthesia may be transferred from one part of the body to the other,

either spontaneously or as a result of suggestion. The latter is most effectual when the transfer is made by means of a magnet or metals.

The special senses may have their function exalted, so that the patients have an extraordinary acuteness of smell or hearing, or find it difficult to endure strong lights.

Depression of the function of the special senses is perhaps more common, particularly loss of the sense of smell and taste. Hysterical deafness is exceedingly rare. Hysterical blindness not infrequently occurs, is characterized by widely dilated pupils, that usually react to light, and, of course, by normal eye-grounds. The hysterical stigmata associated with the eye are of great importance, partly on account of their peculiarities, partly on account of their persistence. The most frequent is simple contraction of the formed field. This, however, occurs in other conditions, and is, therefore, not as characteristic as contraction of the formed field with inversion of the color field—that is to say, a red object will be seen further from the central visual point than a blue one. Monocular diplopia, in the absence of structural defect in the eyeball, is pathognomonic of hysteria. In rare cases three images may be perceived.

The *motor symptoms* are paresis, or occasionally complete paralysis. The commonest form of this is *hysterical aphonia*, in which the patients are unable to contract the vocal cords for the purpose of producing sound, but may be perfectly able to cough or perform any other function with them. In these cases speech usually returns suddenly under the influence of a strong emotion or suggestion. The paralysis in other parts of the body occurs in imitation of some form of organic disease. Thus there may be paraplegia, hemiplegia, or monoplegia. Loss of power is rarely complete, and occasionally patients move the limbs when they believe themselves unobserved. The electrical reactions remain normal, although the degree of resistance in the skin may be greatly increased. The reflexes are exaggerated, especially those due to cutaneous irritations, such as the plantar reflex, but ankle clonus does not occur. The gait may be staggering, imitating cerebellar ataxia or the ataxia due to intoxication; sometimes there are tremors, coarse and irregular, and rarely constant. In some cases of hysteria actual contractures of the muscles occur, indicating the existence of trophic disorders. Spasmodic contractions sometimes occur in the muscles of the abdomen, giving rise to an apparent or *hysterical abdominal tumor*. Actual trophic changes may also occur in hysterical patients, but these are rare in this country. There may be hemorrhages into or from the skin, particularly from the forehead, palms of the hands, and the soles of the feet (*stigmata of the passion*), or there may be localized areas of gangrene in the skin.

The *attack* (*crise hystérique*) may be divided into the prodromal period and the convulsive. The auræ consist of a variety of sensory disturbances, of which the most common is the sensation of a ball rising in the throat (*globus hystericus*). The patient may also have a sensation of heat or cold, or moisture of the skin, or various painful impressions. Occasionally the tenderness over the ovary is greatly increased (*ovaria*), and the attack may be precipitated by pressure in this region. It is impossible to describe all the movements that occur in the *grande crise*.

The convulsion may be tonic or clonic. The patient may assume the most extraordinary positions. Among the most characteristic is opisthotonos, in which the heels and the back of the head rest upon the floor or bed, while the body forms an arch; or the patient may assume attitudes that suggest or are characteristic of mirth, sorrow, fear, passion, etc. Consciousness is rarely entirely lost, although there may be subsequently total amnesia for the period of the attack, and, no matter how violent the movements of the patient, injury to any part never occurs. Gradually the movements become less violent, the patient becomes quiet, and consciousness returns. During the attack the pupils are usually dilated, the reflexes may be increased, and respirations are commonly extremely rapid, in one case that I observed they reached 100 per minute. Occasionally the attack may be cut short by pressure upon one of the hysterogenic zones. After the attack the patient may be perfectly normal. At times there may be persistent, curious, perverse tendencies, such as unwillingness to eat, or, at least, a simulation of fasting.

Neurasthenia is a disease characterized by an exceedingly complex symptomatology. The symptoms may be divided into the general and special groups: the former including those common to all forms of neurasthenia, the latter those associated particularly with subjective and objective functional disturbance of the various organs. The mental symptoms are various. The patients are usually querulous, depressed, and hypochondriacal. They are very irritable, but incapable of prolonged emotional exaltation. They find difficulty in concentrating their attention, particularly upon those subjects with which they have previously been familiar. Memory is impaired and the intellectual capacities apparently diminished. It must be remembered, however, that careful testing of the memory or judgment rarely shows that it is seriously affected. An important symptom is the insomnia. This may be of all varieties, but ordinarily the patient, after sleeping in the early part of the night, will awaken and be unable to sleep again for some hours. The statements by the patients in regard to this symptom are very unreliable. Frequently they complain of unpleasant or frightful dreams when they actually have slept. Among the sensory symptoms the most important is headache. This is of a peculiar but almost typical form. The patient complains of a heavy, dull feeling, as if wearing some heavy object, the usual simile being a lead helmet. Occasionally the pain is localized; sometimes to the occipital region and sometimes to a circumscribed area, the latter usually the result of suggestion. Another symptom that is very common is pain in the back. This is usually felt in the neck or the lumbar and sacral region; it is of a dull, persistent character, and may be associated with points of tenderness over the spine. Occasionally there are disturbances of the special senses. The patient may complain of inability to see sharply, or there may be muscæ volitantes. At other times he will fail to hear distinctly or may complain of roaring or tinnitus. Actual diminution of the visual power or of the sense of hearing does not occur. The patients may complain, however, of paræsthesiæ in the limbs and of various symptoms usually the result of suggestion. Sensation is other-

wise normal. There is usually a general decrease in muscular power. Sometimes this may be preserved for short periods of activity, but fatigue, as a rule, comes on very rapidly. At other times it is impossible for the patient to exert the amount of force that would be normal for his muscular development. Occasionally this weakness is localized to one limb or side of the body. When the patient is directed to hold a limb rigid or to extend the fingers forcibly a fine tremor of the extremities occurs. This may be persistent or readily exhausted; in addition, fibrillary twitchings of the muscles not infrequently occur. The tendon reflexes are generally exaggerated. Ankle clonus, however, excepting the form spoken of as pseudoclonus, is exceedingly uncommon. Absence of the knee-jerk does not occur in neurasthenia. The cutaneous reflexes are sometimes greatly exaggerated, sometimes depressed. Vasomotor symptoms are very common. The patient flushes easily, and there is often dermographia; he complains of palpitation and occasionally of irregularity of the heart's action. Often perspiration is produced by slight exertion.

In addition to these symptoms, the neurasthenic may complain of various local disorders of the nervous system; he usually suspects that he has locomotor ataxia, and he will probably have learned the symptoms of this condition sufficiently well to imitate them more or less accurately, or he may believe himself suffering from general paresis or brain tumor, or any other condition with which he may be familiar. From general paresis the diagnosis is sometimes quite difficult unless the Argyll-Robertson pupil, which never occurs in neurasthenia, is present. Another common form is gastro-intestinal neurasthenia. The patient may complain of excessive acidity, and, in fact, vomit from time to time masses of acid material, or there may be difficulty with digestion and hypochlorhydria or anacidity. Constipation is an exceedingly frequent symptom. From time to time the patient may also evacuate large quantities of mucus, and sometimes there may be persistent mucous diarrhœa. This is one of the most intractable forms of the disease. Finally, the patient may be a sexual neurasthenic and believe himself to be suffering from organic or functional disease of the genital organs. To this variety is usually, but I believe incorrectly, reckoned the various types of sexual perversion. The degree of neurasthenia is spoken of as mild or severe, according as the symptoms are slight or pronounced.

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